reduction of the number of valuable organ donors, a critical problem currently.4 Thirdly, brain death defined as the irreversible cessation of all functions of the entire brain is a continuous process where discrete critical events such as disappearance of brainstem reflexes, flat EEG, and cerebral circulatory arrest can be reliably documented, but may happen in different combinations and different times in different brain-injured patients.5 We cannot pinpoint it.6

Conflict of interest
None declared.

F. A. Rasulo
F. Volonté
R. Bertuetti
N. Latronico*
Brescia, Italy
*E-mail: nicola.latronico@med.unibs.it

2 Wijdicks EF. Brain death worldwide: accepted fact but no global consensus in diagnostic criteria. Neurology 2002; 58: 20–5

doi:10.1093/bja/aeq328

Headache and nuchal rigidity and photophobia after an epidural blood patch: diagnosis by exclusion of persistent post-dural puncture headache mimicking meningitis

Editor—Epidural blood patch (EBP) has been shown to be effective in either complete resolution of symptoms or in marked reduction in the severity of post-dural puncture headache (PDPH).1 EBP is safe but not free of side-effects.

A healthy 28-yr-old parturient gravida 1, para 0 presented for induction of labour at 39 weeks of gestation and requested epidural analgesia. An epidural catheter was successfully inserted at L2–3 level at the first attempt, using a 16 G Tuohy and loss of resistance to sterile saline. The space was located at 4.5 cm and the catheter was threaded for an extra 5 cm into the epidural space. Neither blood nor cerebrospinal fluid return was noted in the catheter. A bilateral sensory block was achieved at T6. Caesarean delivery was uncomplicated. A single dose of cefazolin 2 g was given i.v. as soon as the baby was delivered. The epidural catheter was removed in the post-anaesthesia care unit. Eight hours after delivery, the patient complained of a
thorbing fronto-occipital headache with interscapular pain and nausea that intensified through postoperative day 1 (POD1). The patient was started on i.v. hydration, i.v. paracetamol 1 g every 6 h, ibuprofen 100 mg, and ranitidine 50 mg every 8 h, and bed rest. Symptoms did not improve and pain intensity worsened by the end of POD2. On POD3, blood studies and body temperature (36.8 °C) were normal with no symptoms of local or systemic infections. Consequently, the patient was offered an EBP. While continuously monitoring the patient in the sitting position, the L2–3 epidural space was located at the first attempt. Twenty millilitres of autologous blood were withdrawn from the antecubital vein in a sterile way and slowly injected into the epidural space. The patient remained in the supine position for 2 h after which she assumed an upright position and reported marked improvement in her PDPH and other symptoms. Analgesics were stopped and the patient was monitored.

Four hours after the EBP, the patient started complaining of severe dull headache accompanied by neck stiffness. The patient reported a postural occipital headache, nuchal rigidity, and photophobia with no nausea. Both Kernig’s and Brudzinski’s signs were absent. Neither fever nor chills were noted while monitoring body temperature every 1 h. Blood and urine samples were sent for analysis and culture. On physical examination, there was no evidence of either focal neurological deficit or increased intracranial pressure. The symptoms did not improve on POD3 and POD4. Both the blood and urine cultures were negative and the white cell count was normal. Another EBP was repeated on POD5 and the headache and nuchal rigidity resolved within the first hour of performing the procedure. Symptom-free, the patient was discharged home on POD7.

On the basis of the symptoms on POD1, our patient fitted the criteria for PDPH. After failure of the first EBP to resolve the patient’s symptoms, a second EBP performed 2 days later resolved the symptoms of PDPH.

The role of EBP in managing PDPH has been previously described. An audit reported that 88% of all dural puncture cases developed PDPH and that 70% of these cases were successfully treated with EBP. In parturients with accidental dural puncture, the diagnosis of dural puncture was delayed until presentation of headache in 27% of cases.

In our patient, the relapse of headache on POD3 and development of new symptoms of nuchal rigidity and photophobia could have resulted from septic meningitis, aseptic meningitis, drug-induced aseptic meningitis, cerebral venous sinus thrombosis, subdural hematoma, or ineffective initial epidural blood patching.

Meningitis after EBP has been reported in parturients who were afebrile at the time of EBP. Our patient was afebrile between Caesarean delivery and the administration of the EBP and the absence of fever through the first five postoperative days undermines the possibility of septic meningitis. Aseptic meningitis, characterized by a triad of fever, nuchal rigidity, and photophobia, usually presents within 24 h of spinal anesthesia and could be secondary to the irritative effect of blood in the epidural space. Despite the development of nuchal rigidity and headache in our patient, the absence of fever makes aseptic meningitis an unlikely diagnosis. Non-steroidal anti-inflammatory drugs, cephalosporins and ranitidine, that our patient received can induce aseptic meningitis.

Subdural hematoma after dural puncture is a rare complication of epidural analgesia, but has been reported. Although no imaging was done in our patient, neither the symptoms nor the progression of these symptoms support the possibility of subdural hematoma.

Ineffective blood patching is common and multiple EBPs are sometimes needed to resolve PDPH in up to 40% of cases. In our patient, the symptoms of postural headache and nausea occurring on POD1 were treated as PDPH.

In summary, we report a PDPH occurring in a 28-yr-old parturient after an epidural for labour analgesia in which the headache persisted after a first EBP and was further compounded by the onset of new neurological symptoms including nuchal rigidity and photophobia. The case resembled meningitis, but the absence of fever pointed to a persistent PDPH resolved with a second EBP.

Conflict of interest
None declared.

G. Kanazi
F. Abdallah
A. Dabbous
S. Atweh
M. El-Khatib*
Beirut, Lebanon
*E-mail: mk05@aub.edu.lb

5 Sprigge JS. Epidural blood patch. Anaesthesia 1999; 54: 300–1
6 Oh J, Camann W. Severe acute meningeval irritative reaction after epidural blood patch. Anesth Analg 1998; 87: 1139–40
Influence of ambient light on cerebral oximeters

Editor—Cerebral oximetry has become a standard monitoring in cardiac surgery and some studies have shown its usefulness in non-cardiac surgery. In the future, it may become widely used for routine monitoring. There are two different technologies used, absolute and relative oximetry. Absolute oximetry uses fibreoptic laser light of four different wavelengths (690, 780, 805, and 850 nm; ForeSight, Casmed medical, CT, USA), and relative oximeters emit 2 (730, 810 nm; Invos cerebral oximeter, Somanetics Corporation, MI, USA) or three (730, 810, and 880 nm; Regional Oximetry system 7600, Nonin medical Inc., MN, USA) different wavelengths, using LED. The proponents of absolute oximeters advocate that no baseline calibration is needed, hence any change during surgery does not have to be related to the baseline values, and that their monitoring cannot be influenced by ambient light.

In order to test the hypothesis that absolute oximeters are less affected by ambient light, we tested three different cerebral oximeters (Foresight, Invos cerebral oximeter, and Nonin regional oximetry system) in the laboratory, simulating OR conditions with ambient light of different direction and intensity. We placed all three devices on a large table, with the only light source being natural light on an overcast day entering a rectangular room from only one side. We then placed all three monitors next to each other, making sure that all sensors would receive light of the same intensity and direction. All monitors were switched on and the sensors exposed to ambient light; by shifting the sensors around, we tried to create different ambient light angles and shades.

The Foresight monitoring did not appear to be influenced by ambient light, irrespective of the angles, shades, light intensity or direction, and showed no value and the message ‘ambient light, no reading’. The Nonin monitor appeared to be influenced by ambient light, especially with partial covering of the sensor when increasingly low saturation values were displayed without any warning signal to indicate ambient light or artifact. When the sensors were placed in direct light, very high saturation numbers could be achieved (Fig. 1A). The Invos showed a bit more independence from ambient light than the Nonin; however, even without attaching the sensors to anything, saturation numbers in the ‘normal’ range could be achieved (Fig. 1A).

The results of the laboratory testing could be reproduced in a simulated OR setup with dimmed light—similar to the ambient lighting used for endoscopic surgery. The clinical importance of the influence of ambient light on cerebral oximeter devices should not be underestimated: one can easily imagine a clinical scenario of a patient undergoing surgery in dimmed light conditions (e.g. endoscopic surgery), where the adhesive sensors partially lose contact with the patient’s skin and are thus exposed to ambient light. Relative cerebral oximeter values such as created in the laboratory setting could be easily occurring in this scenario. At present, absolute oximetry is not influenced by ambient light, relative oximetry is. Further refinement is required to make sure that relative oximeters reliably detect artifact values created by ambient light.

Fig 1 (A) Direct artificial light on the sensor of the Regional oximetry system 7600 (Nonin Inc., USA) creates very high values. (B) Both relative oximeters, the Regional oximetry system 7600 (Nonin Inc.) and the Invos cerebral oximeter (Somanetics), show aberrant values when sensors are exposed to ambient light.