Extradural analgesia has served obstetric patients for many years, but not without complications, as Dr Harding's list of references shows. Deliberate puncture of the dura mater represents a further significant breach of the patient's defences. In our enthusiasm to improve patient comfort and well being it is as well to remember the dictum "First of all do no harm".

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**Sir,—** We thank Dr Sinton for his detailed comments on our case report. The combined spinal-extradural was used to provide analgesia for vaginal examination and rupture of membranes in this woman with a known low lying placenta. In our experience, this technique provides excellent sacral analgesia without motor block, which is obviously undesirable in early labour.

We apologize that full details were not given; the extradural catheter was resident only after attempts to produce bilateral analgesia had failed. The second extradural catheter placement also afforded unsatisfactory analgesia and another subarachnoid block was the only alternative to a general anaesthetic for the emergency Caesarean section. The patient had expressed a desire to remain awake during delivery.

It is our practice to offer extradural blood patch early in the treatment of post-dural puncture headache if simple analgesics and non-steroidals have failed and the patient desires further treatment. Extradural blood patches have been used since 1960 [1] with a very low incidence of serious complications [2].

We do not feel that comparison of the combined spinal-extradural technique with micro-spinal catheters is appropriate. Micro-spinal catheters of 27-gauge or less were withdrawn in the USA in 1992 (not the early 1980s) after 11 cases of cauda equina syndrome. Micro-spinal catheters of 27-gauge or less were withdrawn in the USA in 1992 (not the early 1980s) after 11 cases of cauda equina syndrome were reported [3, 4].

All patients have the right to fully informed consent before agreeing to any medical procedure. Our firm intention, as with all other doctors, is to do no harm to our patients.

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**Near-infrared spectroscopy in adults**

**Sir,—** We refer to the article by Germon and colleagues [1], which described the effects of extracerebral ischaemia on measurement of regional cerebral oxygenation (rSO$_2$) by near-infrared (NIR) spectroscopy (Invos 3100 cerebral oximeter).

On application of a tourniquet around the scalp, the authors showed an initial rapid reduction in rSO$_2$ to 13%, plateauing by 3 min, which returned to no more than initial values on release of the tourniquet. As arterial collaterals from the skull may take 2–3 min to fully engorge the scalp after inflation of a tourniquet around the skull, their results may be explained fully by a large increase in extracranial chromophore attenuation the light signal both into and out of the skull. To determine how long it takes for hypoxia to develop in skeletal muscle during tourniquet ischaemia, we applied tourniquets to the upper arm in six healthy male subjects and applied the oximeter to the forearm muscles. The reduction in oxygenation was greater than 50% and did not plateau until 5–6 min. Furthermore, hyperaemia occurred on release of the tourniquet with oxygenation increasing to 25% above initial levels (fig. 1). This contrasts markedly with the results of Germon and colleagues over the frontalis muscle. We believe the authors have misinterpreted their results by attributing them to changes in extracranial oxygenation rather than a highly unusual change in chromophore content caused by tourniquet engorgement.

In the second study the frontalis muscle was exercised for 1 min. Exercise induced hyperaemia and increased chromophore contents may again explain their findings. Finally, the authors did show a good correlation between rSO$_2$ and induced hypoxia measured by pulse oximetry, despite the application of a scalp tourniquet. This at least demonstrates that even in these highly artificial circumstances this instrument can still detect cerebral hypoxia.

Carotid endarterectomy is an ideal model to study the accuracy of cerebral oximetry. Selective injection of indocyanine green into the internal and external carotid arteries demonstrated that sensors with a 30–40-mm separation, between light source and the superficial and deep detectors, respectively, exclude extracranial attenuation while allowing maximum intracerebral penetration [2]. We have reported excellent correlations between rSO$_2$ and jugular bulb venous oxygen saturation, middle cerebral artery blood flow and general hypoxia [3, 4]. The extracranial circulation is extensively collateralized and on unilateral occlusion of the external carotid artery there is normally little change in rSO$_2$ values. However, with severe internal carotid disease, the extracranial circulation forms important collaterals to the brain.

![Figure 1](image-url)  
**Figure 1** Mean (SEM) changes in oxygenation in forearm skeletal muscle ischaemia using a Somanetics Invos 3100 oximeter (n = 6). T = Tourniquet.
changes, all of which affect curaneous blood flow. In several such cases whom we have monitored, these changes have rendered the measurement of \( rSo \), otherwise impossible.

It should come as no surprise that complete forearm ischaemia results in a much greater reduction in \( rSo \) than scalp ischaemia. The volume and thickness of tissue rendered ischaemic by the scalp tourniquet is clearly much smaller. As intracranial oxy-
generation remains unaltered and contributes in part to the \( rSo \) signal, the reduction in \( rSo \) would be less than that seen with an arm tourniquet when all tissue in the field of interrogation is rendered ischaemic. Differential changes in the desaturation of skin, subcutaneous tissue and muscle explain the different time course and reperfusion seen with forearm ischaemia. The time course of \( rSo \) reduction which occurs in response to scalp ischaemia is remarkably similar to that observed when an earlier version of the Invos 3100 with less depth resolution was placed on the scalp during hypothermic circulatory arrest [1]. Using this earlier monitor Harris and Bailey [2] failed to show any increase in \( rSo \) in response to hypercapnia causing 100% increase in middle cerebral artery (MCA) flow velocity, and Brown, Wright and Royston [3] showed a poor correlation with jugular venous oxygen saturation in patients undergoing cardiac surgery.

We agree with Picton and colleagues that carotid endar-
terectomy is an interesting model in which to study cerebral oxygenation. In one of the first studies performed using the Invos 3100, Williams and colleagues [4] showed a weak correlation with jugular bulb venous oxygen saturation, middle cerebral artery blood flow and general hypoxia. Perhaps more im-
portantly, we are not aware of any work which relates changes in \( rSo \) to absolute measures of regional cerebral blood flow, and the same group of authors has shown a poor correlation with jugular venous oxygen saturation in three patients undergoing carotid endarterectomy using the updated optode configuration (with light receivers 3 cm and 4 cm from the light source) [5]. In one patient, following clamping of the internal carotid artery, there was a reduction in \( rSo \) on the operated side with a reduction in MCA flow velocity on that side. This is anecdotal support for our finding that the new config-
uration is sensitive to oxygenation changes in cerebral tissue. However, all the other events described affect extra- in addition to intracranial oxygenation and therefore this study provided no evidence that changes in intracranial oxygenation can be separated. In neither study was there statistical evidence to support the claim that there are, "excellent correlations between \( rSo \) and jugular bulb venous oxygen saturation, middle cerebral artery blood flow and general hypoxia". Perhaps more im-
portantly, we are not aware of any work which relates changes in \( rSo \) to absolute measures of regional cerebral blood flow, and the updated monitor we were unable to reliably detect the changes scalp and reperfusion would expect from hypercapnia causing a 15% increase in cerebral blood flow [6].

We believe that NIR spectroscopy has huge potential as a clinical monitoring technique but agree with a recent editorial that the introduction of new technology into clinical practice should be based on solid evidence of efficacy rather than anecdotal belief [7].


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