via orbital and other anastomoses so that occlusion of these vessels results in a reduction in intracerebral oxygenation.

In our experience based on over 200 carotid studies, the Invos 3100 cerebral oximeter reliably detects changes in intracerebral oxygen saturation. The gross changes in extracerebral blood volume precipitated in Dr Germon's studies are unlikely to occur in practice and are probably irrelevant clinically. This piece of equipment is valuable in carotid surgery and monitoring of intracerebral oxygenation may have wide application in stroke medicine, following head injury and throughout complex anaesthesia.

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Sir,—We are grateful to Mr Picton and colleagues for stimulating further debate on the principles and applications of near-infrared (NIR) cerebral spectroscopy. They have questioned the methodology of our study and have drawn attention to some of our own experimental and clinical work.

We have no doubt that the application of the scalp tourniquet as we have described results in rapid, prolonged and stable ischaemia of the extracranial tissues. Pulsatile red cell flux measured by laser Doppler ceases within a few seconds. Cyanosis of the scalp develops quickly and persists until the tourniquet is released. We are unaware of any arterial collateral which pass through the skull vault to supply the scalp and the senior neurosurgical author has encountered none in over 2000 craniotomies. Finally, in a separate study of 10 subjects, as yet unpublished, total scalp haemoglobin measured by NIR spectroscopy did not change in response to tourniquet inflation.

Although no significant change in total chromophore content of scalp occurs in these studies, we appreciate that the scalp ischaemia induced is profound. Nevertheless, it is claimed of this oximeter that the effects of extracranial changes in tissue oxygenation and blood flow on the final computation of cerebral rSO$_2$ are made negligible by the use of a double detector system. Unless this spatial resolution is achieved, NIR cerebral spectroscopy has few advantages over conventional monitoring of arterial oxygen saturation and cerebral perfusion pressure.

Having demonstrated that profound extracranial ischaemia had a predictable effect on the measurement of rSO$_2$, we wished to challenge the system with a more modest change in extracranial blood flow and oxygenation. We agree with Picton and colleagues that the frontalis muscle exercise test may produce a biphasic response of hyperaemia, followed by ischaemia in a muscle which is not accustomed to rapid repetitive contraction. Again, this test should not consistently affect the computation of rSO$_2$ if the double sensor system excludes the influence of moderate changes in extracranial blood flow and oxygenation. We agree with Picton and colleagues that carotid endarterectomy 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 correlation (r$_{14}$ = −0.04, P < 0.05) between rSO$_2$ and MCA flow velocity. However, their data were obtained following clamping of the common carotid artery and their observations may have been affected by changes in extracerebral blood flow. A second study described the response of rSO$_2$ to intraoperative events occurring 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$_2$ on the operated side with a reduction in MCA flow velocity on that side. This is anecdotal support for our finding that the new configuration is sensitive to oxygenation changes in cerebral tissue. However, all the other events described affect extra— in addition to extracranial—oxygenation and therefore this study provided no evidence that changes in intra- and extracerebral oxygenation can be separated. In neither study was there statistical evidence to support the claim that there are, "excellent correlations between rSO$_2$ and jugular bulb venous oxygen saturation, middle cerebral artery blood flow and general hypoxia". Perhaps more importantly, we are not aware of any work which relates changes in rSO$_2$ to absolute changes in regional cerebral blood flow, and the updated monitor we were unable to reliably detect the changes in scalp and jugular venous oxygen saturation caused by hypercapnia causing a 115% 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 expert belief [7].

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Inhaled nitric oxide in acute respiratory failure

Sir,—I found the recent paper by Young and colleagues [1] interesting, although most of their findings are in agreement with previously published human studies. However, I believe there was a significant error in their calculation of venous admixture (QVa/QT). The values at first inspection appeared very low, particularly in the latter part of table 1. My own estimations of QVa/QT (assuming haemoglobin = 130 g litre\(^{-1}\), PaO\(_2\) = 3.6 kPa and PaCO\(_2\) = 5.0 kPa) from the appropriate equations produced values of 0.46, 0.57, 0.73, 0.51, 0.52, 0.35, 0.33, 0.47, 0.38, 0.42, 0.30, 0.23, 0.21 and 0.28 quoted values of 0.39, 0.35, 0.36, 0.25, 0.05, 0.10, 0.26, 0.18, 0.17, 0.07, 0.11, 0.03 and 0.04, respectively, for the 14 patients. In the calculation of oxygen content, if physically dissolved oxygen is inadvertently calculated as 0.003 ml/100 ml/mm Hg, instead of 0.003 ml/100 ml/m Hg, then one obtains shunt figures similar to those obtained in the article.

Alteration of haemoglobin concentration and PaO\(_2\) values within clinically feasible limits does not markedly alter the values obtained. Calculated shunt is sensitive to SvO\(_2\), and therefore PaO\(_2\), but manipulation of this within clinically likely limits did not account for the discrepancy.

As improvement of QVa/QT derangement is a major putative clinical use of nitric oxide, it is important to get the sums correct. It would be useful to know how the results in table 2 change with correct calculation of QVa/QT.

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Postoperative extradural analgesia

Sir,—We read with interest the recent article by Leith and colleagues [1] on the results of a 4-yr audit of 770 patients receiving postoperative extradural analgesia at Vancouver General Hospital, using a mixture of 0.15% bupivacaine with 0.005% diamorphine.

Over the past 8 yr we have used different opioids at various strengths mixed with 0.125% bupivacaine in order to determine the most effective analgesia with the lowest incidence of major or minor complications. We have found that the best opioid to add to bupivacaine is fentanyl at a concentration of 8.3 \(\mu\)g ml\(^{-1}\) (i.e. fentanyl 10 ml in a 60-ml syringe).

Table 1 No. of patients (%) experiencing pain on movement

<table>
<thead>
<tr>
<th>Pain on movement</th>
<th>No. of patients</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>No. of patients (%) with complications. f = Ventilatory frequency, SAP = systolic arterial pressure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>f &lt; 10 (b.p.m.)</td>
<td>Nausea</td>
<td>Pruritis &lt; 100 mm Hg</td>
<td>Motor block</td>
<td></td>
</tr>
<tr>
<td>%</td>
<td>3.0</td>
<td>12.4</td>
<td>3.3</td>
<td>1.4</td>
</tr>
</tbody>
</table>

The audit conducted in York showed that from 1990 to 1993, on average, 47% of patients experienced moderate or severe pain on movement while our own audit of 515 patients over 2 yr had an incidence of 22.3% experiencing the same degree of pain on movement. The incidence of complications with our extradural mixture was also comparable; an incidence of respiratory depression (frequency less than 10 b.p.m.) of 3.0% compared with 2.6%. We found an incidence of vomiting of 12.4% compared with 16.8%, and an incidence of itching of 3.3% compared with 12%, in the audit from York, and an incidence of hypotension of 1.4% compared with 34% with the diamorphine mixture.

There has been much discussion in the past on which opioid should be added to bupivacaine in order to provide the best quality analgesia, and the lowest complication rate. One such study by Enevery and colleagues [3] demonstrated that diamorphine was the more appropriate agent. This study used relatively small numbers (61 patients divided into three groups) and we suggest that the different results from these two extensive audits should stimulate further work in this area.

The interesting finding of our own audit was that patients in the over 80-yr age group had an incidence of respiratory depression of 8.6% compared with 2.2% in the under 80-yr age group. This has led us to halve the quantity of fentanyl in the analgesic mixture in all patients over 80 yr of age. The audit will be repeated after 12 months.

We feel that the results clearly support our choice of opioid, namely fentanyl, in our analgesic mixture. While we have demonstrated some differences between our two services, we would wholeheartedly support the conclusion, still reflected in some quarters, that the ward-based extradural service provides a high quality postoperative recovery.

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