Purpose: To assess the cerebral oximeter, which measures regional oxygen saturation \( (rSO_2) \) continuously and non-invasively, as a cerebral monitor during carotid endarterectomy (CEA). The \( rSO_2 \) was compared with Somatosensory Evoked Potentials (SSEPs) as an indicator for shunting and as a predictor of postoperative neurological deficits.

Methods: Seventy-two consenting patients undergoing CEA with general anaesthesia were studied. Normocarbia, normothermia and normotension were maintained. Cerebral monitoring consisted of bilateral median nerve SSEPs and the INVOS 3100 cerebral oximeter with the sensor pad placed on the ipsilateral forehead. Decreases in SSEP amplitude of 50% and in \( rSO_2 \) of 10% were considered clinically significant. Neurological assessment was performed at emergence from anaesthesia, 24 hr postoperatively and at discharge. The \( rSO_2 \) changes were compared with SSEP changes and with neurological deficits. Statistical analysis was with chi square and analysis of variance. \( P < 0.05 \) was considered significant.

Results: During carotid artery clamping, \( rSO_2 \) decreased from 72 ± 8% to 68 ± 9% and mean arterial blood pressure increased from 92 ± 14 mmHg to 98 ± 14 mmHg. In four patients, the carotid artery was shunted because of SSEP changes after cross-clamping. Five patients had ≥10% decreases in \( rSO_2 \) following clamp application. Changes in both SSEP and \( rSO_2 \) occurred in two patients. Three of the four shunted patients had transient postoperative neurological deficits. One patient had a transient deficit without changes in either monitor. There were no persistent postoperative deficits. Compared with SSEPs, \( rSO_2 \) had a sensitivity of 50% and a specificity of 96%.

Conclusion: Clinical experience with this evolving technology is ongoing. Its role in neurovascular procedures has yet to be established.

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CAROTID endarterectomy (CEA) has been demonstrated to be beneficial in patients with high grade carotid stenosis.\textsuperscript{1,2} However, this surgery is associated with a 2–7.5% risk of perioperative stroke which may be haemodynamic or embolic in origin.\textsuperscript{1–3} To minimize this risk, some centres choose to use intraoperative cerebral monitors to warn of impaired cerebral perfusion. A change in monitoring during carotid artery cross-clamping may be an indication for intervening with physiological manipulations, such as induced hypertension, or mechanical manoeuvres, such as insertion of a shunt. Cerebral monitoring of the patient’s response may also act as a predictor of postoperative neurological status.

At our institution, median nerve somatosensory evoked potential (SSEP) monitoring is the standard cerebral function monitor during CEA. A 50% reduction in amplitude of N20 following application of the cross-clamp is used as the indicator for shunting. The Somanetics INVOS 3100 Cerebral Oximeter is a new cerebral monitor that uses near-infrared spectroscopy (NIRS) to measure regional cerebral oxygen saturation. It has the advantages of being continuous, non-invasive, portable, easy to use and is not affected by anaesthetic agents. The purpose of this study was to compare NIRS with SSEP monitoring during CEA as an indicator for shunting and as a predictor of postoperative neurological deficit.

Methods
With Ethics Committee approval, we studied 72 consenting patients undergoing CEA with general anaesthesia. Prior to induction of anaesthesia, an intravenous infusion of normal saline was commenced and a 20 gauge radial intraarterial line was inserted. Routine monitoring included: five lead electrocardiography, invasive and non-invasive blood pressure, pulse oximetry, end-tidal capnography and vapour analysis, peripheral nerve stimulation, oesophageal stethoscope and oesophageal temperature probe. The cerebral monitors were the Somanetics Invos 3100 Cerebral Oximeter and SSEPs.

Prior to induction, the self-adhesive probe of the cerebral oximeter was applied to the forehead ipsilateral to the site of surgery using tape to exclude ambient light. The screen displays trends using values recorded at 30- or 60-sec intervals and absolute values of regional oxygen saturation (rSO\textsubscript{2}) which change every four seconds. When an adequate tracing was obtained, a baseline rSO\textsubscript{2} was recorded. Any changes, especially at the time of cross-clamping, were noted by the investigator. On the basis of previous studies,\textsuperscript{5–6} a decrease in saturation of $\geq$10% was considered clinically significant. The surgeon was not informed of changes in cerebral oxygen saturation.

An electrophysiology technician was present throughout the surgery to perform monitoring of SSEPs as is the routine practice at our institution. Following induction of anaesthesia, stimulating needle electrodes were placed over both median nerves and recording needle electrodes were inserted in the scalp over the somatosensory cortex (C3' and C4') and over the C2 spine. The reference electrode was placed on the forehead. Bilateral baseline SSEPs were recorded using standard parameters. The SSEPs were monitored continuously during carotid cross clamping. Clinically significant SSEP changes were defined as a >50% reduction in the amplitude of the cortical peak N20. The surgeon was informed of SSEP changes and a shunt inserted at his discretion.

Anaesthesia was induced with 2–3 $\mu$g.kg\textsuperscript{-1} fentanyl, a sleep dose of propofol or thiopentone. An appropriate muscle relaxant was given to facilitate tracheal intubation. Anaesthesia was maintained with oxygen-nitrous oxide (FiO\textsubscript{2} 0.5), isoflurane, boluses of fentanyl and non-depolarizing muscle relaxants. Normothermia was maintained. Arterial blood gases were analysed and mechanical ventilation controlled to keep PaCO\textsubscript{2} between 38 and 42 mmHg. Blood pressure was maintained at preoperative levels using appropriate titration of anaesthetic agents, fluid administration and phenylephrine.

Neurological assessment was performed by the neurosurgeon immediately postoperatively, 24 hr after surgery and on discharge from hospital. New neurological deficits were classified as transient if they lasted < 24 hr and persistent if > 24 hr.

Statistical analysis was with chi square and analysis of variance. $P < 0.05$ was considered significant. Changes in cerebral oximetry were compared with SSEP changes and with postoperative neurological deficits. The sensitivity and specificity of cerebral oximetry compared with SSEPs were calculated.

Results
Fifty-three male and 19 female patients with a mean (± SD) age of 67 (± 9) yr and weight of 78 ± 21 kg were studied. Duration of cross-clamp time was 51 ± 22 min. Core body temperature was 35.5 ± 0.7°C. During cross-clamping, there was an increase in mean arterial blood pressure (MAP). Heart rate and arterial oxygen saturation, as measured by pulse oximetry, remained stable. Cross-clamping was associated with a decrease in rSO\textsubscript{2} which increased towards baseline values following release of the clamp. (Table I)

Four of the 72 patients had > 50% reduction in SSEP amplitude following cross-clamping. (Table II)
TABLE I Measured variables (mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th>Preclamp</th>
<th>Clamp on</th>
<th>Clamp off</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAP (mmHg)</td>
<td>92 ± 14</td>
<td>98 ± 14*</td>
<td>90 ± 13</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>65 ± 13</td>
<td>66 ± 13</td>
<td>66 ± 13</td>
</tr>
<tr>
<td>SaO₂ (%)</td>
<td>98 ± 1</td>
<td>98 ± 1</td>
<td>98 ± 1</td>
</tr>
<tr>
<td>rSO₂ (%)</td>
<td>72 ± 8</td>
<td>68 ± 9*</td>
<td>70 ± 8</td>
</tr>
</tbody>
</table>

* P < 0.05 from preclamp & clamp off

TABLE II Relationship of changes in rSO₂ to changes in SSEP

<table>
<thead>
<tr>
<th>SSEP Change</th>
<th>rSO₂</th>
<th>No rSO₂ change</th>
</tr>
</thead>
<tbody>
<tr>
<td>No SSEP Change</td>
<td>3</td>
<td>65</td>
</tr>
</tbody>
</table>

rSO₂ change = decrease ≥10%
SSEP change = decrease in amplitude N20 ≥50%

In all cases, the SSEP returned to baseline after shunt insertion. In five patients, application of the clamp was associated with ≥10% decrease in rSO₂ which lasted the duration of clamp time. Changes in rSO₂ coincided with SSEP changes in two patients.

Three of the four shunted patients sustained transient postoperative neurological deficits. (Table III) Another patient had a transient deficit without changes in either SSEP or rSO₂. In all four patients the deficit was contralateral weakness noted on emergence from anaesthesia and lasting less than six hours. There were no persistent postoperative neurological deficits. One patient in this series died within 24 hr of surgery. This patient suffered a fatal intracerebral haemorrhage secondary to uncontrolled postoperative hypertension.

Reliable SSEPs, there were two true positive rSO₂ changes, three false positives, two false negatives and 65 true negatives. (Table II) Compared with SSEP changes, a 10% reduction in rSO₂ has a sensitivity of 50% and a specificity of 96%.

Discussion

Central nervous system monitors fall into two main groups, those that monitor functional endpoints and those that monitor oxygen supply to the brain. The functional monitors which have been used in CEA are the awake patient, EEG and SSEPs. Stump pressure measurement, xenon measurements of cerebral blood flow, Doppler assessment of flow velocity, intraoperative angiography, jugular venous oxygen content and now NIRS monitor oxygen supply indirectly.

The application of NIRS to the noninvasive study of human cerebral metabolism was described by Jobsis in 1977.² It is based on the Beer-Lambert law which states that the absorption of light is proportional to the concentration of chromophores, their absorption coefficients and the optical path length. Near infrared light, in the wavelength range of 650–1100 nm, penetrates human scalp, skull and cranial contents for several centimetres. It is absorbed by oxyhaemoglobin, deoxyhaemoglobin and oxidized cytochrome aa₃ at characteristic wavelengths (900 nm, 760 nm and 780–870 nm respectively). The absorption of light by oxyhaemoglobin relative to total haemoglobin, with a small contribution by the redox state of cytochrome aa₃ is used to deduce cerebral oxygen saturation.⁸

Intracranial attenuation of light is detected by two sensors. Attenuation due to superficial tissues, measured by the sensor closer to the light source, is subtracted from the total attenuation, measured by the more distant sensor, to derive attenuation of light due to intracranial contents.⁹ The cerebral oximeter used in our study has a source-detector distance of 30 and 40 mm. Because previous source-detector distances of 10 and 27 mm enhanced interference from external circulation,¹⁰,¹¹ distances were increased to 30 and 40 mm in newer models. However a recent study has shown application of a scalp tourniquet induced a decrease in rSO₂, suggesting external circulation interference even at distances of 30 and 40 mm.¹² There may be inter-individual variation in the distribution of external and internal cerebral circulation. Change in rSO₂ following application of the external carotid artery clamp in our study may have provided a measure of external circulation interference.

Since 70–80% of the cerebral cortical vascular bed is venous, the oxygen saturation that is measured is predominantly venous. Values are, therefore, affected by oxygen extraction, arterial oxygen content and blood flow. Because the proportion of venous blood volume remains constant under controlled physiolog-

TABLE III Relationship of changes in rSO₂ and SSEP to postoperative neurological deficits

<table>
<thead>
<tr>
<th>Neurological Deficit</th>
<th>SSEP</th>
<th>rSO₂</th>
<th>rSO₂ &amp; SSEP change</th>
<th>No change</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Neurological Deficit</td>
<td>1*</td>
<td>—</td>
<td>2*</td>
<td>1</td>
</tr>
<tr>
<td>No Neurological Deficit</td>
<td>1*</td>
<td>3</td>
<td>—</td>
<td>64</td>
</tr>
</tbody>
</table>

* = shunted
All postoperative neurological deficits were transient.
ical conditions and cross clamping defines the onset of a potential cerebral insult, CEA lends itself to cerebral oximetry monitoring.

To date, regional cerebral oxygen saturation during CEA has been found to correlate with jugular venous oxygen saturation,\textsuperscript{10,13} transcranial Doppler measure of cerebral artery blood flow velocity\textsuperscript{10,13,14} and subjective assessment of internal carotid artery (ICA) backflow.\textsuperscript{6} In our study, we found that changes in rSO\textsubscript{2} did not reliably relate to clinically important changes in SSEPs. This is the first published study examining the relationship between SSEPs and cerebral oximetry.

Since there is considerable inter-individual variation in baseline measures of rSO\textsubscript{2},\textsuperscript{4,6,13,17} it is appropriate to use relative changes in rSO\textsubscript{2} rather than absolute values. Clinical experience with this technology is evolving. At this stage, a change in rSO\textsubscript{2} of 10% appears to be a reasonable arbitrary threshold.\textsuperscript{4,5,6}

In our study, blood pressure increased following application of the cross-clamp. This reflects the practice of maintaining or increasing blood pressure during cross-clamping. In previous studies, rSO\textsubscript{2} has been sensitive to blood pressure changes.\textsuperscript{15,16} It is possible that a more marked decrease in rSO\textsubscript{2} during carotid clamping could be expected if hypertension had not been maintained.

Disparity between cerebral oximetry and SSEPs may be attributable to their monitoring of different regions of cerebral circulation. Placement of the oximeter self-adhesive probe on the forehead detects changes in circulation to the frontal lobe whereas SSEPs detect changes in the distribution of the middle cerebral artery. Unfortunately, placement of the probe over the parietal lobe would require shaving the patient's head. Williams \textit{et al.}\textsuperscript{10} found improved correlation between rSO\textsubscript{2} and jugular venous oxygen saturation when the probe was placed over the parietal lobe and source-detector distance was increased. From their study, it is difficult to determine how much of the improvement was due to a change in probe placement and how much to a change in source-detector distance.

Evaluation of a cerebral monitor for CEA surgery is problematic. The prevalence of postoperative neurological deficit is so low\textsuperscript{4-3} that a very large number of patients would need to be studied to calculate negative and positive predictive values of a new monitor. Sensitivity and specificity require comparison with an accepted standard. At our institution, the standard for monitoring during CEA is SSEPs. Compared with SSEPs, we found a change in rSO\textsubscript{2} of $\pm$10% had a low sensitivity and high specificity. The ideal monitor for detection of impending cerebral ischaemia during CEA should have high sensitivity.

Another standard for CEA monitoring is an awake patient. The few studies of cerebral oximetry in the awake patient have found that changes in rSO\textsubscript{2} were not associated with changes in the patient's neurological condition.\textsuperscript{17,18} Like us, both studies of awake patients report a decrease in rSO\textsubscript{2} during carotid artery clamping. In the study by Samra \textit{et al.},\textsuperscript{17} patients were monitored bilaterally to differentiate systemic causes of changes in rSO\textsubscript{2}, such as hypoxaemia and hypotension, from causes related to surgery. This improves the yield of information and may be a useful application of the monitor.

Comparison of the cerebral oximeter with established cerebral monitors has produced varying results. While it has the advantages of being non-invasive and easy to use, its limitations are that it is a regional, superficial monitor of cerebral oxygenation. As such, the probe should be placed over the area of circulation at risk. The issue of external circulation remains unresolved, even with increased source-detector distances. Further refinement of the technology is ongoing. With more clinical experience, the ultimate role of the cerebral oximeter in neurovascular procedures will be established.

Acknowledgement

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