An NIRS matrix for detecting and correcting cerebral oxygen desaturation events during surgery and neuroendovascular procedures

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Background: Transcranial cerebral oximetry was developed for early detection of cerebral hypoxia to avoid cerebral dysfunctions. However, near infrared spectroscopy (NIRS) data obtained during surgery are subject to intrinsic and extrinsic influences that have to be accounted for when interpreting the recordings.

Methods: We developed an NIRS matrix to provide brief information for a specific intervention to correct changes of cerebral oxygen saturation (COS). Selected vital data and the descriptors of cerebrovascular and neurofunctional status were linked to logistic chains.

Results: The matrix is horizontally and vertically grouped and contains five descriptors: 1. change of COS; 2. key variable (parameter related to the change of COS); 3. associated parameters (vital data that do not cause COS alterations); 4. interpretation of values or preconditions most probably due to COS changes; and 5. the intervention most likely to normalize the COS or return it to baseline. The descriptors are grouped horizontally to a logistics chain.

Conclusion: The modular expandable NIRS matrix we describe has promise for clinical use in surgical, neurointerventional and anaesthesiological contexts. [Neurol Res 2005; 27: 423–428]

Keywords: Near infrared spectroscopy (NIRS); NIRS matrix; cerebral oxygen saturation (COS); neuromonitoring

INTRODUCTION

Transcranial near infrared spectroscopy (NIRS) can even detect small changes in cerebral oxygen metabolism following minimal physiologic, pathophysiologic and therapeutic events1–3. NIRS has potential applications for monitoring patients at risk of cerebral oxygen desaturation during certain types of surgery4 such as cardiac surgery, major vascular surgery and carotid endarterectomy, for patients with surgical positioning issues, for patients older than 60 years5, and patients undergoing neuroendovascular procedures6. Intraoperative NIRS monitoring has potential for early identification of vulnerable episodes7 and consequent improved neurological outcome8 but the technique has prerequisites and limitations that have to be appreciated9. Using NIRS effectively requires accurate interpretation of measurements and this in turn requires correcting for intrinsic and extrinsic influences that can affect the results. We developed a matrix, i.e. the shortest unit of information, for interpreting NIRS data with the aim of integrating NIRS results into clinical care.

METHODS

The NIRS matrix contains five descriptors which are causally related to one another. The horizontal left–right link at one level contains the changes of cerebral oxygen saturation (COS) at one end. At the other end, as the result of conclusions, there are specific anesthesiologic interventions to consolidate the altered COS-values.

(a) Descriptors of the NIRS matrix

1. The major determinant is the ‘change of COS’ as an indicator of changes in cerebral oxygen balance. COS changes are described as decrease (in special cases fluctuations of couplets of COS decreases and increases) from baseline.

2. The next determinant is the ‘key variable’, which as the value that undergoes marked changes is related to the changes in COS. This determinant results from a group of values obtained with conventional intraoperative monitoring named the ‘basic data’: mean arterial blood pressure (MAP), hemoglobin (Hb), peripheral oxygen saturation (SaO2), partial carbon dioxide pressure (pCO2) and core temperature (t). In the logistics
chain group A (explained below) one parameter of the basic data (=vital data) becomes the key variable once it shows a marked change (shift into an abnormal range or change from baseline). It is thus necessary to obtain a series of values to detect changes. For the logistics chain groups B and C (explained below) the key variables are not vital parameters obtained from the patient but rather physical (anatomical) (B) or (neuro-)pharmacologic (C) variables that have an effect on the COS.

(3) The key variable is compared with the ‘associated parameters’ which within the groups of basic data described above showed no marked changes. Each of the basic data can also assume the position of key variable that causes a decrease of the COS. For the sake of didactic order the matrix does not use complex representations of synergistic constellations or antagonistic combinations.

(4) The ‘clinical interpretation’ of the most probable underlying event is deduced from the data constellation COS – key variable – associated parameters.

(5) Finally the matrix deduces the ‘intervention’ most likely to normalize the COS or return it to its baseline level. The linkage of the main descriptors as described above results in a ‘logistics chain’ (Figure 1).

(b) Groups of logistics chains of the NIRS matrix

The vertical grouping (Table 1) of the horizontal logistic chains was done with causal differentiation:

(A) biologic measurement values (vital data),
(B) cerebrovascular components,
(C) neurofunctional components.

The logistics chains in group A contain information on cerebral oxygen supply and oxygen demand. The pathophysiologic chain of the configurations in group B is its common endpoint, the compromised oxygen supply to the brain. Primary causes are anatomical obstructions of blood vessels and patient positioning, most commonly decreased perfusion with rotation of

![Diagram](image)

**Figure 1:** Complete linkage of the main determinants from ‘COS’ to ‘intervention’ called ‘logistics chains’

<table>
<thead>
<tr>
<th>COS</th>
<th>Key variable</th>
<th>Associated parameters</th>
<th>Interpretation</th>
<th>Intervention</th>
<th>Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>↓ Hb ↓</td>
<td>MAP, SaO₂, pCO₂, t: (=)</td>
<td>O₂-transport capacity ↓ blood loss, hemodilution</td>
<td>Blood product replacement</td>
<td>A</td>
<td></td>
</tr>
<tr>
<td>↓ MAP ↓</td>
<td>Hb, SaO₂, pCO₂, t: (=)</td>
<td>Excessive hypotension, impaired autoregulation</td>
<td>Blood pressure correction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>↓ SaO₂ ↓</td>
<td>MAP, Hb, pCO₂, t: (=)</td>
<td>Systemic arterial hypoxeration</td>
<td>FiO₂ ↑, optimization of ventilation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>↓ pCO₂ ↓</td>
<td>MAP, SaO₂, Hb, t: (=)</td>
<td>CBF ↓, reduced cerebral perfusion</td>
<td>Correction of ventilation (normoventilation)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>↓ t ↑</td>
<td>MAP, Hb, SaO₂, pCO₂; (=)</td>
<td>CMRO₂ ↑</td>
<td>Adaptation as required (normothermia/mild hypothermia)</td>
<td>B</td>
<td></td>
</tr>
<tr>
<td>↓ Head rotation</td>
<td>carotid stenosis/incomplete arterial circle of Willis</td>
<td>Arterial obstruction</td>
<td>Orthograde head position</td>
<td></td>
<td></td>
</tr>
<tr>
<td>↓ * Carotid clamping*</td>
<td></td>
<td>Carotid occlusion*</td>
<td>Shunt, declamping*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>↓ Malpositioned venous catheters/cannulas</td>
<td>MAP, Hb, SaO₂, pCO₂, t: (=)</td>
<td>Venous obstruction</td>
<td>Correct position of catheters</td>
<td></td>
<td></td>
</tr>
<tr>
<td>↓ v_m ↑, art diam ↓</td>
<td>MAP, Hb, SaO₂, pCO₂, t: (=)</td>
<td>Vasospasm</td>
<td>Vasodilatation (nimodipine; papaverine intraarterial; balloon angioplasty)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>↓ Inadequate anesthesia</td>
<td>MAP, (=)v ↑, SaO₂, Hb, pCO₂; t: (=)</td>
<td>CMRO₂ ↑</td>
<td>Deepening of anesthesia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>↓ Cerebral seizures</td>
<td>MAP, SaO₂, Hb, pCO₂, t: (=)</td>
<td>CMRO₂ ↑</td>
<td>Anticonvulsive management</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

MAP, mean arterial pressure; SaO₂, peripheral oxygen saturation; Hb, haemoglobin; pCO₂, partial carbon dioxide pressure; t, core temperature; (=), within normal ranges/not pathognomonically changed; ↓, decrease; ↑, increase; ↓↑, fluctuation of COS decrease and increase; v_m, mean blood flow velocity (transcranial Doppler sonography); art diam, cerebral artery diameter (cerebral angiography).

*Subjects of interdisciplinary information.
the head in patients with bilateral carotid stenosis or insufficiency of the arterial circle of Willis. Additionally, the effects of clamping and declamping of major arteries (e.g., carotid artery) necessary for oxygen carriage to cerebral structures on COS may provide helpful interdisciplinary information for the anesthesiologist and surgeon. Oxygen supply to the brain can also be compromised by obstructed venous return due to malpositioned catheters and cannulas or cerebral edema. A specific pattern during endovascular procedures is observed frequently in patients with arterial vasospasm after subarachnoidal hemorrhage over the affected area of the brain. Periodic spontaneous fluctuations of increases and decreases of COS (\( \uparrow \downarrow \)) can appear from time to time with a relatively stable baseline or they can be seen as a continuous regular pattern\(^6\). In this situation the characteristic increase of mean flow velocity of transcranial Doppler sonography or the angiographic aspect of diminished diameters of cerebral arteries take the role of the key variable. Additionally, functional factors (especially the key variables of oxygen supply) can accentuate the changes in COS in patients with predisposing anatomical factors. Group C reflects the influences of neuronal activity on cerebral oxygen consumption. Inadequate anesthesia may lead to increased oxygen consumption by the waking brain and thus to decreased COS. The benefit of central inhibitory medications such as anesthetic agents on the equilibration of COS becomes apparent especially when neuronal activity has to be suppressed. Cerebral seizures, with their increase in cerebral neuronal activity, can increase cerebral oxygen consumption and thus lower COS.

RESULTS

NIRS matrix

The horizontal linkage from the individual descriptors of the matrix — beginning with the ‘alterations of COS’ and ending at the ‘intervention’— forms the logistics chain. The vertical composition of different logistics chains builds the block configuration of the NIRS matrix (Table 1).

Case reports

Case 1

A 35-year-old woman underwent a craniotomy after acute subarachnoidal bleeding (Hunt & Hess, grade 2) from an aneurysm of the superior cerebellar artery. Total intravenous anesthesia (TIVA) was obtained with propofol (6–12 mg/hour) and remifentanil (0.025 \( \mu \)g/kg/hour). Paralysis was obtained with cisatracurium (0.9 mg/kg/hour i.v.). Ventilation was set to end expiratory \( \text{CO}_2 \) (et\( \text{CO}_2 \)) values of 32–34 mmHg. Monitoring included invasive blood pressure (BP), electrocardiogram (ECG), peripheral oxygen saturation (Sa\( \text{O}_2 \)), central venous pressure (CVP), body temperature (Siemens Sirecust1281, SiemensAG, Erlangen, Germany). Although craniotomies are not recommended indications for NIRS monitoring, we used the NIRS method and the NIRS matrix in this patient. NIRS monitoring for registration of regional cerebral oxygen saturation (r\( \text{SO}_2 \)) was done with the cerebral oximeter INVOS 3000 (Somanetics, Troy, USA). After 2.5 hours of smooth surgery the aneurysm ruptured, causing severe bleeding with a precipitous drop of BP, et\( \text{CO}_2 \) and hemoglobin and increased heart rate (Figure 2). Sa\( \text{O}_2 \) remained stable (Figures 2 and 3). Packed red cells were administered rapidly. The hemodynamic status stabilized after blood was replaced and the aneurysm clipped. Before rupture of the aneurysm NIRS values (r\( \text{SO}_2 \)) showed a stable trend at 47–50 (Figure 3, a). Corresponding to the NIRS matrix (Group A) the COS dropped sharply (24%) immediately after rupture (Figure 3, b). The key variables BP and Hb were reduced. The associated parameters, especially Sa\( \text{O}_2 \) were unchanged; the decrease of et\( \text{CO}_2 \) followed the hemodynamic alterations and was not caused by hyperventilation. The interpretation of the cause for the decrease of COS resulted in marked systemic hypotension and a decrease of oxygen transport capacity by blood loss. The anesthesiologic intervention was the rapid correction of blood pressure by replacing blood products. The effect of the therapeutic interventions was reflected by the immediate increase of COS. The temporary “overswing” of COS (Figure 3, c) may be caused by changes in the regional distribution of cerebral blood volume caused by rapid replacement of blood and/or the effects of the clipping. After this episode the COS returned to values (Figure 3, d) slightly above baseline. The patient’s post-operative course was uneventful with no neurological deficits.

Case 2

A 36-year-old woman was scheduled for endovascular embolization and stent implantation to correct an
monitoring was done with an INVOS 4000 cerebral oximeter (Somanetics). Ventilation settings were adjusted so that baseline etCO$_2$ was $\sim 30$ mmHg. The bilateral COS curves were in the low range (rSO$_2$, 40–45) (Figure 4). The only notable laboratory value was an arterial pCO$_2$ of 28 mmHg. At this point imaging studies showed a vasospasm (Figure 4, time point A, and Figure 5a). In the NIRS matrix (Group A), in the group of basic parameters, the low initial COS values were assigned to the low pCO$_2$ as the key variable. The interpretation yielded reduced cerebral perfusion due to hypocapnea. The intervention consisted of correction of ventilation. As the respiratory minute volume was reduced incrementally, etCO$_2$ and COS rose continuously. With etCO$_2$ values stable $\sim 40$ mmHg, the bilateral COS values were also stable (rSO$_2$, $\sim 60$) and thus 20% higher than before the respiration settings were changed. Angiography of the basilaris artery showed no evidence of vasospasm (Figure 4, time point B, and Figure 5b). A temporary blood pressure drop (ΔMAP $>18\%$) (Figure 4) showed no effect on COS values. After the intervention the patient awoke adequately from anesthesia and showed no neurologic deficits.

**Case 3**

A 47-year-old woman presented with acute subarachnoidal bleeding (Hunt & Hess, 3). Imaging showed a ruptured aneurysm of the basilar artery and an intact aneurysm of the left internal carotid artery. Transcranial
Doppler sonography (TCD) showed blood flow velocities of 185 and 154 cm/s in the right and left middle cerebral arteries, respectively. NIRS monitoring was performed with an INVOS 4000 (Somanetics) cerebral oximeter. NIRS showed a unilateral (right) alternating pattern (COS $\downarrow \uparrow$, matrix group B) (Figure 6a). The key variables were the very high bilateral values of mean blood flow velocity by TCD. The interpretation yielded a vasospasm of the cerebral resistance vessels. The suggested intervention was intraarterial application of papaverine (20 mg bolus, 125 mg continuously over 30 min) in addition to the running triple H therapy. The NIRS recording during administration of papaverine showed disappearance of the alternating COS pattern on the right side (Figure 6a). On the left side, NIRS values increased by $\sim 20\%$ during administration of papaverine. The effect of papaverine was transient and the same procedure was repeated the following day. Ten weeks later the patient’s speech was slow but there were no further neurologic deficits.

**DISCUSSION**

For the configuration of the matrix, the individual changes in COS are assigned one univariate descriptor change according to the key variable from the pool of the basic data. Bivariate and polyvariate links of the key variables and/or links of parameters in the pool of basic data to key variables have to be made individually. Interpretations and correctional interventions should be made on this basis in surgical, neurointerventional and anesthesiologic context. This management is reflected by our case reports.

In the experience of some authors $^{10-12}$ the number of specific therapeutic interventions to correct COS...
is limited. In this sense the interventions of the NIRS matrix include the most important standard therapeutic aspects. In: Litscher G, Schwarz G, eds. Transcranial Cerebral Oximetry, Lengerich: Pabst Science Publishers, 1997; pp. 232–251

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