Transcranial Cerebral Oximetry During Surgical Procedures: A New Matrix Model For Minimization Of Problems Of Interpretation And Intervention In Desaturation Events

G Schwarz, G Litscher

Citation

G Schwarz, G Litscher. *Transcranial Cerebral Oximetry During Surgical Procedures: A New Matrix Model For Minimization Of Problems Of Interpretation And Intervention In Desaturation Events*. The Internet Journal of Neuromonitoring. 2003 Volume 3 Number 2.

Abstract

Transcranial cerebral oximetry is a method to detect cerebral hypoxia and to avoid cerebral dysfunctions. Regional cerebral oxygen saturation was measured in a 35-year-old female during cerebral aneurysm surgery. For the effective clinical use of near infrared spectroscopy (NIRS) during surgical or endovascular procedures the intrinsic and extrinsic factors which can influence the NIRS data have to be considered. Therefore a NIRS matrix as a short information for the specific management to correct changes of cerebral oxygen saturation (COS) was developed.

The aim of development of the NIRS matrix is to support the patient management specifically adapted to the surgical, neurointerventional and anesthesiological context.

INTRODUCTION

Transcranial near infrared spectroscopy (NIRS) can detect changes in cerebral oxygen metabolism even following minimal physiologic, pathophysiologic, and therapeutic events [1,2,3]. This method has potential applications for monitoring patients at risk of cerebral oxygen desaturation during certain types of surgery [4] such as cardiac surgery, major vascular surgery and carotid endarterectomy, for patients with surgical positioning issues, for patients older than 60 years [5] and neuroendovascular procedures [6]. There are prerequisites and limitations associated with NIRS which have to be appreciated for the use of this technique [7]. However, reports about the beneficial aspects of intraoperative NIRS monitoring for early identification of vulnerable episodes [8] and the improved postoperative neurological outcome [9] are encouraging.

Using NIRS effectively requires accurate interpretation of the measurement values and this in turn requires correcting for intrinsic and extrinsic factors 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 procedural care.

METHODS

Our NIRS matrix for intraoperative applications 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.

NIRS MATRIX DESCRIPTORS

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 (Tab. 1): mean arterial blood pressure (MAP), hemoglobin (Hb), peripheral oxygen saturation (SaO2), partial carbon dioxide pressure

(pCO2), 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 to the "associated parameters" which within the groups of basic data described above showed no marked changes (Fig. 1). Each of the basic data can also assume the position of key variable that cause 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 (Fig. 2).

5. Finally the matrix deduces the "intervention" most likely to normalize the COS or return it to its baseline level (Fig. 3).

NIRS MATRIX - GROUPS OF LOGISTICS CHAINS

The vertical grouping (Fig. 3) 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 via key variables contain information on cerebral oxygen supply (MAP, pO2, hemoglobin, pCO2) and oxygen demand (core temperature).

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 the head in patients with bilateral carotid stenosis or insufficiency of the arterial circle

of Willis.

Continuous supply of oxygen to the brain can also be compromised by obstructed venous return due to malpositioned catheters and cannulas or cerebral edema.

A very important unique pattern during endovascular procedures can be observed in patients with arterial vasospasm after subarachnoid hemorrhage. Periodic fluctuations of increases and decreases of COS ( ) may appear from time to time with a relative stable baseline or they can be observed as a 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 beneficial effect of central inhibitory medications such as anesthetic agents on the equilibrization of COS becomes apparent especially with the sometimes necessary suppression of neuronal activity. Cerebral seizures, with their dramatic increase in cerebral neuronal activity (verifiable with EEG), can increase cerebral oxygen consumption and thus lower COS.

NIRS MATRIX - EXPLANATION OF TERMS

Figure 1
Table 1: "Basic data" are defined as the summation of the intraoperatively monitored parameters (= vital data) that mainly can influence the COS.

Basic data
mean arterial pressure (MAP)
hemoglobin (Hb)
peripheral oxygen saturation (SaO_2)
partial carbon dioxid pressure (pCO_2)
core temperature (t)

If one parameter of the "basic data" is significantly altered it becomes the "key variable". The other parameters of the "basic data" which are unchanged or minimally changed are summarized as "associated parameters".

The complete horizontal linkage of the main determinants of the matrix from COS to "intervention" is called "logistics chain".

The vertical combinations of corresponding "logistics chains" are summarized to specific "logistics chain groups".

RESULTS

The results (NIRS matrix) are summarized in Table 2:

Figure 2

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

MAP = mean arterial pressure
SaO₂ = peripheral oxygen saturation
Hb = hemoglobin
pCO₂ = partial carbon dioxide pressure
t = core temperature
(≡) = within normal ranges
↓ = decrease
↑ = increase
↓↑ = fluctuation of COS decrease and increase
v_m = mean blood flow velocity (transcranial Doppler sonography)
art diam = cerebral artery diameter (cerebral angiography)

CASE REPORT

We report about simultaneous transcranial cerebral oximetry and laser Doppler flowmetry monitoring during a cerebral aneurysm surgery (a. cerebelli sup.) in a 35-year-old female.

Figure 1 shows an intraoperative recording session of more than 3 hours. LDF-Flux, rSO2 and skin temperature at the recording position of the laser Doppler probe were sampled and documented simultaneously. At "A" (time event marker in Fig. 1; middle panel) the skin incision was performed.

The Flux and the rSO2 values increased. At "B", "C" and "F" (Fig. 1) anesthesia was deepened and burst suppression activity in the electroencephalogram (EEG) appeared.

Only scant changes were seen in the Flux values, which were not reproducible. The rSO2 showed no significant alterations during this period. The marker "D" (Fig. 1) indicates the rupture of the aneurysm. It is clearly demonstrated that the rSO2 values decreased. The Flux also showed a reduction, though with a significant time delay in comparison to the intracerebral oxygenation. Systolic blood pressure decreased from 120 mmHg to 80 mmHg.

Further, there was a period of time in which an extreme reduction of temperature was observed. This was caused by the application of cold red cells transfusion following the rupture of the aneurysm ("E"). The external microcirculation did not follow this trend. Finally, the end of the operation is marked by "G". Corresponding to the NIRS matrix, in the presented patient the COS (rSO2) was reduced significantly at "E". The key variables were MAP and Hb . The associated parameters SaO2, pCO2 and t were unchanged. The interpretation showed a combination of a marked hypotension and a decrease of O2 transport capacity by blood loss.

The intervention resulted consecutively in blood pressure correction by blood product replacement.

The effect of the intervention was documented by an immediate increase of COS (rSO2). The temporary overswing may be caused by the decrease of t and changes in the distribution of cerebral blood volume.

Figure 3

Figure 1: Regional cerebral oxygenation (rSO2) changes, Flux in arbitrary units (AU) from the laser Doppler flowmetry (LDF) and temperature (Temp.) measured on the recording position (forehead) of the LDF probe during aneurysm surgery in a 35-year-old female prior, during and after rupture (Â"DÂ") of the aneurysm (modified from [24]).
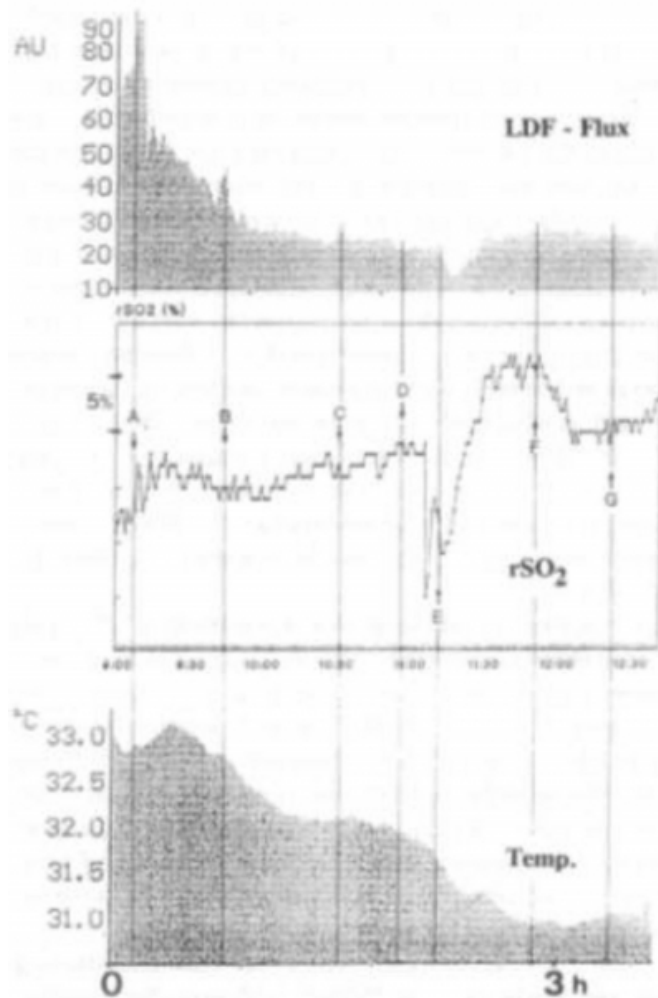
DISCUSSION

Accurate interpretation of intraoperative NIRS data requires integrating of extracerebral influences. The NIRS matrix (Tab. 3) is based on this fact. The matrix corresponds to a configuration of logistics chains in the sense of forward chaining.

Like most of matrices the NIRS matrix for intraoperative use can provide only a rough guideline and the risk of oversimplification is imminent. However, this matrix can support decision making in clinical practice.

For the configuration of the matrix, the individual changes in

COS are assigned only 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.

According to the experience of some authors [10,11,12] the number of therapeutic interventions to correct COS is not wide spread and this is reflected in the configuration of the matrix.

The most evident problem with NIRS use is the lack of recognized limits for alarm or intervention at the moment. A number of reports indicate that COS decreases of > 25 % signify imminent cerebral ischemia/hypoxia and require immediate intervention. Roberts et al. [12] reported that awake patients undergoing carotid endarterectomy who had a rSO2 decrease of > 27 % were at risk of developing neurodeficits. Studies on conscious individuals involved in + Gz acceleration [13], implantable cardioverter–defibrillator testing [14], and tilt–table syncope testing [15] consistently found severely impaired cerebral function with a relative rSO2 decline of 25 % or more. Moehle [16] defined a cerebral oxygen desaturation event as a COS decrease > 20 % from baseline for greater than 3 min in the cardiopulmonary bypass surgical patient.

Although abnormally low absolute rSO2 values have been associated with evidence of cerebral dysfunction [17], in our opinion NIRS monitoring is mainly a trend monitoring [18,19]. Accordingly we chose for the NIRS matrix the relative changes of COS (uni- or bilateral decrease or fluctuation of alterning patterns of dercreases and increases) as described above related to baseline for indicating significant changes of cerebral oxygen metabolism.

The ambivalence of NIRS data should be kept in mind. Marked drops in COS are very sensitive indicators of potentially threatening changes in cerebral oxygen metabolism. But case studies of dynamic pathologic processes such as intracranial pressure crises or transtentorial herniation [20] show that the converse does not apply - i.e., that largely stable COS values do not guarantee cerebral integrity.

Despite these limitations NIRS monitoring has considerable potential to improve cerebral monitoring during surgical [21] and neurointerventional [22,23] procedures. We aim to include

our NIRS matrix in a computer-assisted system with automated linkage of different data combinations to provide an immediate monitoring support adapted to the surgical, neurointerventional and anesthesiological context.

References

1. Litscher G, Schwarz G. Near infrared spectroscopy and multivariable monitoring. In: Litscher G, Schwarz G, eds. Transcranial cerebral oximetry, Lengerich Berlin Düsseldorf Riga Scottsdale Wien Zagreb: Pabst Science Publishers, 1997: pp. 10-61

2. Urlesberger B, Trip K, Ruchti JJ, Kerbl R, Reiterer F, Müller W. Quantification of cyclical fluctuations in cerebral blood volume in healthy infants. Neuropediatrics 1998; 29: 208-211

3. Colier WN, Quaresima V, Oeseburg B, Ferrari M. Human motor-cortex oxygenation changes induced by cyclic coupled movements of hand and foot. Exp Brain Res 1999; 129: 457-461

4. Widman RA, Gonopolskiy O. Near-infrared spectroscopy - future aspects. In: Litscher G, Schwarz G, eds. Transcranial cerebral oximetry, Lengerich Berlin Düsseldorf Riga Scottsdale Wien Zagreb: Pabst Science Publishers, 1997: pp. 232-251

5. Moller JT, Cluitmans P, Rasmussen LS, Houx P, Rasmussen H, Canet J, Rabbitt P, Jolles J, Larsen K, Hanning CD, Langeron O, Johnson T, Lauven PM, Kristensen PA, Biedler A, van Beem H, Fraidakis O, Silverstein JH, Beneken JEW, Gravenstein JS, for the ISPOCD investigators. Long-term postoperative cognitive dysfunction in the elderly: ISPOCD1 study. Lancet 1998: 857-861

6. Misra M, Dujovny M, Alp MS, Aletich V, Ausman JI. Transcranial cerebral oximetry in endovascular procedures. In: Litscher G, Schwarz G, eds. Transcranial cerebral oximetry, Lengerich Berlin Düsseldorf Riga Scottsdale Wien Zagreb: Pabst Science Publishers, 1997: pp. 98-109

7. Schwarz G, Litscher G, Voit-Augustin H. Transcranial cerebral oxymetry: A non-invasive tool for estimating cerebral oxygen metabolism. Internet J Neuromon 2001; http://www.ispub.com/ostia/index.php?xmlFilePath=journals/ijnm/vol2n2/tco.xml

8. Daubeney PEF, Smith DC, Pilkington SN, Lamb RK, Monro JL, Tsang VT, Livesey SA, Webber SA. Cerebral oxygenation during pediatric cardiac surgery: identification of vulnerable periods using near-infrared spectroscopy. Eur J Cardiothorac Surg 1998; 13: 370-377

9. Yao FF, Ho CA, Huang SW, Tseng C. Maintaining adequate cerebral oxygen saturation during cardiac surgery shortened intensive care unit stay in female gender. Anesth Analg 2002; 94: S83*

10. Austin EH3rd, Edmonds HLJr, Auden SM, Seremet V, Niznik G, Sehic A, Sowell MK, Cheppo CD, Corlett KM. Benefit of neurophysiologic monitoring for pediatric cardiac surgery. J Thorac Cardiovasc Surg 1997; 114: 707-717

11. Edmonds HLJr, Rodriguez RA, Audenaert SM, Austin EH3rd, Pollock SBJr, Ganzel BL. The role of neuromonitoring in cardiovascular surgery. J Cardiothorac Vasc Anesth 1996; 10: 15-23

12. Roberts KW, Crnkowic AP, Linneman LJ. Near - infrared spectroscopy detects critical cerebral hypoxia during carotid endarterectomy in awake patients. Anesthesiol 1998; 89(3A): A934

13. Chelette TL, Albery WB, Esken RL, Tripp LD. Female exposure to high G: performance of simulated flight after 24 hours of sleep deprivation. Aviat Space Environ Med 1998; 69(9): 862-868

14. Singer I, Edmonds HLJr. Changes in cerebral perfusion during third-generation implantable cardioverter-defibrillator testing. Am Heart J 1994; 127: 1052-1057

15. Singer I, Edmonds HLJr. Head-up tilt testing predicts syncope during ventricular tachycardia in implantable cardioverter-defibrillator patients. J Interven Cardiol 1998; 11(3): 205-211

16. Moehle AD. Neuromonitoring in the cardiopulmonary bypass surgical patient: clinical applications. J Extra Corpor Technol 2001; 33: 126-134

17. Bhasker Rao B, Van Himbergen D, Edmonds HLJr, Jaber S, Ali AT, Pagni S, Koenig S, Spence PA. Evidence for improved cerebral function after minimally invasive bypass surgery. J Card Surg 1998; 13: 27-31

18. Schwarz G, Litscher G, Kleinert R, Jobstmann R. Cerebral oximetry in dead subjects. J Neurosurg Anesthesiol 1996; 8: 189-193

19. Schwarz G, Litscher G. Transcranial cerebral oximetry, transcranial Doppler sonography, and heart rate variability: useful neuromonitoring tools in anaesthesia and intensive care? Eur J Anaesthesiol 2002; 19: 543-549

20. Unterberg A, Rosenthal A, Schneider GH, Kiening K, Lanksch WR. Validation of monitoring of cerebral oxygenation by near infrared spectroscopy in comatose patients. In: Tsubokawa T, Marmarou A, Robertson C, Teasdale A, eds. Neurochemical monitoring in the intensive care unit, Tokyo: Springer, 1995: pp. 204-210

21. Prabhune A, Sehic A, Spence PA, Church T, Edmonds HLJr. Cerebral oximetry provides early warning of oxygen delivery failure during cardiopulmonary bypass. J Cardiothorac Vasc Anesth 2002; 16(2): 204-206

22. Kamingo M, Ochi M, Onizuka M, Takahata H, Shibata S. An additional monitoring of regional cerebral oxygen saturation to HMPAO SPECT study during balloon test occlusion. Stroke 1999; 30: 407-413

23. Hernandez-Avila G, Dujovny M, Slavin KV, Luer MS, Nijensohn E, Geremia G, Ausman JI. Use of transcranial cerebral oximetry to monitor regional cerebral oxygen saturation during neuroendovascular procedures. AJNR Am J Neuroradiol 1995; 16: 1618-1625

24. Litscher G, Schwarz G. Transcranial cerebral oximetry - Is it clinically useless at this moment to interpret absolute values obtained by the INVOS 3100 cerebral oximeter? Biomed Technik 1997: 42: 74-77

Author Information

Gerhard Schwarz

Department of Anesthesiology for Neurosurgical and Craniofacial Surgery and Intensive Care, Medical University of Graz

Gerhard Litscher

Department of Biomedical Engineering and Research in Anesthesia and Critical Care, Medical University of Graz