Transcranial Cerebral Oximetry: A Non-invasive Tool For Estimating Cerebral Oxygen Metabolism
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Abstract

PRINCIPLES

Transcranial cerebral oximetry is a non-invasive technique for monitoring changes in cerebral oxygen metabolism, which presents additive informations when the conventional key variables as peripheral oxygenation and/or systemic hemodynamics would not be predictive.

Figure 1

Fig 1: Decrease of regional cerebral oxygen saturation (rSO2) during hyperventilation maneuvre whereas peripheral oxygen saturation (SaO2) remains unchanged. (1=normoventilation, 2= episode of hyperventilation, 3= restart of normoventilation)

The method relies on the measurement of absorption of light at multiple wave lengths in the 690 – 1100 nm spectral range and this allows the transmission of photons through skin, bone, brain and liquor.

Figure 2

Fig 2: Schematic presentation of the range of light. Possible changes of the oxygenation status of intracerebral chromophores are detected transcranially.

A further biophysical premission is the change in the absorption of hemoglobin depending on its oxygenation status [1] and the change of absorption in the cytochrome aa3 depending on its redox state. To determine differences in concentration of the dominant chromophores of brain the Beer Lambert law has been modified:

\[ \text{Attenuation} = \alpha \cdot c \cdot d \cdot B + G \]

\( \alpha \) = absorption coefficient of the chromophore
\( c \) = concentration of the chromophore
\( d \) = distance between probes
\( B \) = differential pathlength factor (the mean path of photons in multiscattering tissue is longer than the distance between light collection probes [2]).
\( G \) = light loss depending on geometry of the probe and the optical properties of the volume sampled.
The values obtained with near infrared spectroscopy (NIRS) represent primarily the oxygenation status of the chromophores of the venous compartment (75%) of cerebral vascular bed (arterial 20%, capillary 5%) [3].

**Figure 3**
Fig 3: Schematic diagram of cerebral vascular bed (modified from [4]).

Changes in the parameters of transcranial cerebral oxymetry result from changes in the balance between cerebral oxygen supply and oxygen consumption. The setting of NIRS – systems are schematically based on a near infrared light emitter and a receiver. These are placed usually at the lateral forehead and should have an interoptode distance of at least 4 cm. The NIR - light to and from the optodes is carried by flexible optical fibres.

**MAIN NIRS – PARAMETERS**

**Figure 4**

<table>
<thead>
<tr>
<th>Oxymetred hemoglobin</th>
<th>[Cty36]</th>
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</thead>
<tbody>
<tr>
<td>Deoxygenned hemoglobin</td>
<td>[Decy36]</td>
</tr>
<tr>
<td>Total hemoglobin</td>
<td>[Tb-tct]</td>
</tr>
<tr>
<td>Oxized cytochrome aa3</td>
<td>[ctO2]</td>
</tr>
<tr>
<td>Regional O2 saturation index</td>
<td>[RStO2]</td>
</tr>
<tr>
<td>Tissue oxygen index</td>
<td>[TNI]</td>
</tr>
<tr>
<td>Tissue hemoglobin index</td>
<td>[THI]</td>
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</tbody>
</table>

**INDICATIONS**

Publishing on the use of NIRS in various clinical applications continues to increase. NIRS provides information which could guide users on interpretation of situations in which cerebral oxygenation potentially may be impaired.

- Intraoperative monitoring
- Carotid endarterectomy [5]
- Neuroendovascular procedures (aneurysm embolization, thrombolysis) [6,7]
- Cardiac surgery [8]
- Aortic arch surgery [9]
- Intensive care medicine and emergency medicine
- Early identification of intracranial hematoma [10,11,12]
- Detection of desaturation events in severely head injured patients [13,14,15]
- Monitoring of intracranial hemodynamic changes [16]
- Management of aneurysmal subarachnoidal hemorrhage [17,18]
- Detection of cerebral hypoxia during low cardiac output, pulmonary and vascular diseases, sepsis, anaemia [19]
- Estimation of cerebral blood flow [20]
- Evaluation of cerebrovascular CO2 reactivity [21]
- Fetal, neonatal and pediatric medicine [22,23]
- Sleep apnoea [24]
- Epilepsy [25,26]

**INTERPRETATION OF NIRS-DATA**

For interpretation of the clinical significance of readings indicating cerebral dysoxygenation the underlying pathophysiology must be considered. Therefore the synchronous monitoring of relevant parameters and/or the knowledge of some specific preconditions are essential:

- Systemic arterial pressure
- Systemic arterial oxygenation
- Oxygen carrying capacity (hemoglobin)
- Body temperature
- Carbon dioxide
- Depth of anesthesia
- Cerebral arterial or venous obstruction
- Cerebral seizures

**EXAMPLES OF NIRS-RECORDINGS**

NIRS - devices promise high sensitivity to major and even minimal physiologic, pathophysiologic and therapeutic events.

Intraoperative monitoring during neurosurgical procedure [27]

**Figure 5**

Fig 4: NIRS-monitoring during neurosurgical procedure. Note the marked decrease of rSO2 after rupture of a cerebri posterior artery aneurysm (D) and the increase of rSO2 after application of red blood cells (E). Compare the decreased peripheral microcirculation parameter (Flux) and the body temperature measured by laser Doppler flowmetry (LDF) and the temperature effects in addition to the increase of blood pressure and hemoglobin on rSO2-values increase (modified from [26]).

Increased intracranial pressure: NIRS can document pharmacodynamic effects after minimal therapeutic interventions [28].

**Figure 6**

Fig 5: Continuous NIRS registration during posterior fossa surgery in a neuropediatric patient. A: Stable trace of NIRS recordings (TOI); B: Immediate decrease of Hb tot, Oxy Hb and TOI during short lasting cardiac arrest during neurosurgical manipulation at lower brainstem structures; note the significant transient increase of NIRS-values at the recovery period; C: Restored TOI trace.

**Figure 7**

Fig 6: Reproducible increase of Oxy-Hb after application of 10 ml mannitol 20 % or 20 ml hydroxy ethyl starch 10 % in a patient with impaired cerebral compliance after severe head trauma [27].

Monitoring during hyperbaric oxygenation [29]
Figure 8
Fig 7: NIRS-monitoring in a healthy volunteer under hyperbaric conditions. Note the maximal decrease of rSO2 during compression and air breathing at 2.5 ata and the maximal increase of rSO2 values during hyperbaric oxygenation (HBO).

LIMITATIONS
- Focal limitation: The saturation values are representative only of the region beneath the sensor and may not be sensitive to changes on other locations.
- Presence of cerebral extravascular blood collections within the frontal subarachnoid, subdural or intraparenchymal tissue compartments may interfere with the recordings; admixture of signals with that obtained from a stagnant pool of poorly or un oxygenated blood can result in values of no clinical significance.
- Sensor placement: recordings from regions of known infarct, otherwise damaged or absent brain tissue may result in spurious readings. A post-craniotomy metal plate implant obviously makes monitoring impossible. Conversely, the absence of frontal bone can result in overscale reflected signals.
- Extracerebral contaminations: methodological problem [30,31,32], which is discussed controversially [33].

NIRS – INTERFERENCES
- Electrocautery
- Mechanical irritation, motion
- Strong ambient light (infrared sources)
- Dyes
- Dyshemoglobinemia
- Bilirubin-biliverdin

USER MISTAKES
- Malpositioning of optodes: correct placement should be as high as possible on the forehead to avoid readings from frontal sinuses, lateral sensor placement prevents data obtained from sagittal sinus.
- Insufficient light shielding
- Inadequate probe fixation

CLINICAL PROBLEMS
Although there are some very encouraging reports using NIRS-monitoring in severe brain trauma as presented above, it must be taken into consideration that scalp hematomas, bilateral hematomas or deep intracranial hematomas can lead to false negative readings [12]. Some authors rise the question whether NIRS is able to detect ischemic events in head traumatized patient [34]. Dramatic intracranial volume shifts as during transtentorial herniation [35] and in the complete loss of cerebral function in brain death [36,37] and global circulation arrest [38] are not reflected adequately by NIRS in each patient.

Figure 9
Fig 8: Nearly stable rSO2 values during episodes with increased intracranial pressure (ICP) and reduced jugular venous saturation (SjvO2) in severe brain injury (modified from [35])
ACTUAL PROGRESS OF NIRS DEVICES

- 10x brighter light intensity - signal noise ratio x10: increased signal stability, less light interference
- Signal quality index (SQI)/signal strength index
- Synchronous bilateral measurements
- Different modes for NIRS-measurements in adults and pediatrics
- Additional algorithms for rSO2, and TOI calculation
- Higher accuracy for absolute values and trend analysis due to SQI
- Higher measuring frequency – shorter measurement cycles

FUTURE ASPECTS

- NIRS mapping [39]/dynamic NIRS-imaging
- Multiple NIRS data integration into CCT/MR
- Actual on-line measurement of photon pathlength/pathlength resolved spectroscopy [19]:
  - time of flight (TOF)
  - phase-shift measurement by phase-modulation spectroscopy

CONCLUSION

The use of NIRS to provide continuous, real time imaging of tissue oxygenation at the patient’s bedside is conceptually appealing. Although declines of cerebral oxygen saturation of 25-30 % seem to be associated with correctable neurological dysfunction [37], at the moment we do not have clinically useful intervention thresholds. Furthermore, at the present point of technical development it is more reasonable to look at the continuous dynamics of NIRS-parameters than at the absolute values of single readings. The ability to detect and observe the progress of cerebral events as they occur will require new NIRS devices that can accurately measure photon path length and process data from multiple detectors into tomographic images. Research is active and the future will see an increasing number of technological improvements in NIRS. After solving the actual technical problems an increasing benefit in clinical applications is expected. NIRS devices should then be valuable tools to monitor trends for situations in which intracranial hemoglobin saturation may change dangerously.

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