Changes in regional cerebral tissue oxygen saturation during anesthesia induction in female patients undergoing breast cancer surgery

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Research article

Keywords: propofol, regional cerebral tissue oxygen saturation, anesthesia induction, PaO₂

Posted Date: December 20th, 2019

DOI: <https://doi.org/10.21203/rs.2.19383/v1>

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Abstract

Background Although the monitoring of regional cerebral oxygen saturation (rScO₂) is widely used for cardiac and non-cardiac surgeries, conflicting reports regarding changes in rScO₂ during anesthesia induction remain. We designed this cohort clinical study to assess precise alterations in rScO₂ and the possible mechanism.

Methods This cohort study was designed to examine changes in rScO₂ with anesthesia induced by a target control infusion of propofol from the beginning of anesthesia to 30 minutes after induction in patients undergoing breast cancer surgery. rScO₂ values from the right and left sides of patients' foreheads were averaged to directly determine cerebral oxygenation from FORE-SIGHT data. Mean arterial pressure (MAP), heart rate (HR), partial pressure of oxygen in arterial blood (PaO₂), partial pressure of carbon dioxide in arterial blood (PaCO₂), hemoglobin concentration (Hb), and cardiac output (CO) were measured every minute until 30 minutes after anesthesia induction.

Results A total of 30 female patients treated between January 2016 and April 2016 were included in this study. The average rScO₂ at 7 minutes was 81.7%, which was higher than the average rScO₂ at baseline (67.3%) and at 15 minutes (68.3%). Average rScO₂ correlated significantly with PaO₂ during the first 7 minutes of anesthesia induction.

Conclusion During anesthesia induction, changes in rScO₂, which increased to a peak value at 7 minutes, may be correlated with increases in PaO₂, and the return of rScO₂ to baseline at 15 minutes may have occurred due to flow-metabolism coupling and balancing between white matter and gray matter.

1. Background

Near-infrared spectroscopy (NIRS) was introduced as a technique for the noninvasive monitoring of regional cerebral oxygen saturation (rScO₂) in 1977.[1] NIRS measures the relative concentrations of oxyhemoglobin and deoxyhemoglobin within the field of view. Under most circumstances, the contribution from cerebral venous saturation predominates; therefore, rScO₂ does not indicate oxygen delivery but instead provides information regarding the balance between the regional oxygen supply and demand.[2] Some cardiac surgery centers have obtained evidence that rScO₂ monitoring might lead to better perioperative outcomes.[3, 4] rScO₂ monitoring is also used in many non-cardiac surgeries and provides various types of useful information for these surgeries.[5, 6]

However, recent articles have reported conflicting findings regarding changes in rScO₂ during the anesthesia induction period. In 2007, Paisansathan et al. reported that rScO₂ increased from 58% to 68% following fentanyl and thiopental induction and that rScO₂ returned to baseline 20 minutes after desflurane anesthesia maintained using an Oxiplex TS oximeter.[7] In 2009, Nissen et al. used an INVOS Cerebral Oximeter to describe an rScO₂ increase from 67% to 74% following propofol and fentanyl

induction, with little change thereafter in rScO₂ until the end of the surgery.[8] In 2013, Meng et al., who utilized an Oxiplex TS oximeter, found that rScO₂ remained stable at 67% following propofol and fentanyl induction.[9]

We suspected that changes in rScO₂ were correlated with changes in the partial pressure of oxygen in arterial blood (PaO₂), and we designed this cohort clinical study to test this hypothesis and to determine the possible mechanism.

2. Materials And Methods

2.1 Study design

This cohort study was designed to examine changes in rScO₂ during anesthesia induction among patients undergoing breast cancer surgery. Major assessments were conducted from the onset of anesthesia induction to 30 minutes after induction. We followed recommendations in the **Strengthening the Reporting of Observational Studies in Epidemiology** (STROBE) Statement in designing our study and reporting study findings. This study was approved by the Ethics Committee of the First Hospital of China Medical University (protocol no. 2015110301, Chairman Prof. Xinghua Gao, December 4, 2015) and was registered with the Clinical Trials Registry (NCT02687334). All participants provided written informed consent in accordance with the Declaration of Helsinki.

2.2 Patients

A total of 33 patients undergoing elective radical operations for mastocarcinoma between January 2016 and April 2016 at the First Hospital of China Medical University were enrolled in this study. Patients were included in the study if they were ethnic Chinese, between 18 and 65 years old, American Society of Anesthesiologists (ASA) physical status I or II, and undergoing a radical operation for carcinoma of the breast. Patients were excluded if they had a body mass index exceeding 30 kg/m², had a history of hypertension or diabetes mellitus, or were allergic to anesthesia drugs used in the study.

2.3 General anesthesia induction procedure

All of the anesthesia inductions in the study were conducted by the same anesthesiologist. Electrocardiography and pulse oximetry were continued throughout the surgery; end-tidal carbon dioxide (EtCO₂), invasive arterial pressure (Vigileo-FloTrac; Edwards Lifesciences, Irvine, CA, USA), cardiac output (CO, measured using the Vigileo-FloTrac system) and rScO₂ (FORE-SIGHT, CAS Medical Systems, Branford, CT, USA) were monitored throughout the surgery. The nasopharynx temperature was monitored and maintained at 36.0-36.5°C using a warm blanket.

With the administration of an inspired oxygen fraction (FiO₂) of 1.0, general anesthesia was induced with 0.04 mg/kg midazolam, 0.4 µg/kg sufentanyl, 2-2.5 mg/kg propofol (Fresenius Kabi, Austria GmbH), and 0.2 mg/kg cisatracurium. After 4 minutes of assisted ventilation with 100% oxygen, all patients were

intubated within 30 seconds. Patients' lungs were ventilated with intermittent positive pressure. Tidal volume was adjusted to 6-8 ml/kg, and the ventilator rate was adjusted to maintain EtCO₂ at 35-45 mmHg.

2.4 Intervention

The FiO₂ was maintained at 1.0 during the entire 30-minute observation period. To maintain propofol anesthesia, total intravenous anesthesia was achieved by administering propofol at a target plasma concentration of 2.5-4 µg/ml immediately after induction. Remifentanyl (0.2-0.5 µg/kg/minute) was also administered to all patients during the operation.

During anesthesia induction, bradycardia (heart rate (HR)<45 bpm) and hypotension (mean arterial pressure (MAP)<20% below baseline) were treated with supplemental doses of atropine (0.5–1.0 mg) and ephedrine (5–10 mg), respectively. Tachycardia (HR>110 bpm) and hypertension (MAP>20% above baseline) were treated with esmolol (5-10 mg) and urapidil (10-25 mg), respectively. Patients who required these vasoactive agent treatments were removed from the study.

2.5 Measures

Sensors to detect rScO₂ were placed on the right and left forehead and covered with opaque tape to prevent light interference. RScO₂ values from the right and left sides were averaged to directly determine cerebral oxygenation from the FORE-SIGHT data. MAP, CO, HR, rScO₂ and peripheral oxygen saturation (SpO₂) were measured prior to the induction of anesthesia, and the instantaneous values of MAP, HR, rScO₂, CO and arterial blood gas pressures were measured every minute for 30 minutes, starting from anesthesia induction.

2.6 Study outcomes

The primary outcome was changes in rScO₂ with an FiO₂ of 100% during the first 30 minutes after anesthesia induction. The secondary outcome was correlations between rScO₂ and MAP, HR, CO, partial pressure of carbon dioxide in arterial blood (PaCO₂), PaO₂, and hemoglobin (Hb) concentrations.

2.7 Statistical analysis

The sample size was calculated based on the difference of (mean ± standard deviation [SD]=1) rScO₂ between 7 minutes(60%) and 15 minutes (80%) in the pilot study with $\alpha = 0.05$ and power = 0.8 using PASS11 software (NCSS LLC, Utah, USA). The study was adequately powered with $n = 30$. Thirty-three patients were originally enrolled to compensate for an estimated 10% dropout rate. Data are expressed as the means±SD, absolute values, or medians. Before statistical testing was performed, the normality of the distribution of each continuous variable was analyzed using the Kolmogorov-Smirnov test. Paired Student's t-tests were used for statistical analysis of rScO₂ values at different time points. The statistical analysis was performed using the SPSS for Windows software package, version 18 (SPSS, Inc., Chicago,

IL). Linear mixed-effects models were used to test whether there was a significant correlation between a measured variable and its explanatory variables. The R package (<http://cran.r-project.org/>) was used for statistical analysis and figure creation. The threshold for significance was $p < 0.05$.

3. Results

Among the 33 potential patients assessed for eligibility, one patient was excluded; thus, 32 patients were initially enrolled in the study. Ultimately, 2 patients were excluded from the analysis due to missing $rScO_2$ data (Figure 1). A total of 30 female patients (mean age: 49 years; range: 25–65 years) were included in the final sample. Basic information for all variables is presented in Table I.

The analysis incorporated a total of 900 observation points, which consisted of measurements of the examined variables obtained every minute for the 30 patients. Graphical data exploration suggests that average $rScO_2$ increased from the beginning of the induction to reach its peak value at 7 minutes and then returned to baseline at 15 minutes after induction (Figure 2G). The average $rScO_2$ at 7 minutes was 81.7%, which was higher than the average $rScO_2$ at baseline (67.3%) and at 15 minutes (68.3%) (Table I). Changes in MAP, HR, and CO were prospective as a result of normal effects of anesthesia induction (Figure 2A, B, and C). PaO_2 increased with the administration of 100% oxygen. There were no significant changes in $PaCO_2$ and Hb during the 30-minute period (Figure 2D and F).

Average $rScO_2$ did not correlate with MAP, HR, CO, Hb, $PaCO_2$, or PaO_2 during the 30-minute induction period (Figure 3A–F, $p > 0.05$). Average $rScO_2$ correlated significantly with PaO_2 during the first 7 minutes of anesthesia induction (Figure 3G, $p < 0.01$).

4. Discussion

Among the cohort of 30 females who underwent elective radical operations for mastocarcinoma, we found that the average $rScO_2$ at 7 minutes after anesthesia induction was higher than the average $rScO_2$ at baseline but that the average $rScO_2$ returned to baseline at 15 minutes after anesthesia induction. Changes in $rScO_2$ did not correlate with MAP, HR, CO, Hb, $PaCO_2$, and PaO_2 during the 30-minute induction period but correlated significantly with PaO_2 during the first 7 minutes after anesthesia induction.

Several animal experiments with nearly the same results as our study may support our findings, although they do not directly mention the correlation of PaO_2 and $rScO_2$. [10–12] Brain oxygen tension (PtiO₂) sensing provides a continuous measure of oxygen partial pressure for real-time monitoring of temporal oxygen changes in the cerebral tissue. The PtiO₂ increased after the induction of arterial hyperoxia, and the extent of this increase depended on the initial cerebral blood flow (CBF) value. The response of PtiO₂ demonstrated an early phase of rapid increase followed by an increase in PaO_2 . [12] The highest brain tissue oxygen levels were reached in all cases at the end of the increased FiO_2 phase. [11] PaO_2

oscillations were transmitted to the cerebral microcirculation in a porcine model,[13] which may explain our finding that rScO₂ increases with PaO₂ during the first 7 minutes after anesthesia induction.

Two studies have examined the relationship between rScO₂ and PaO₂ in awake patients and general anesthesia patients. When the carotid artery was cross-clamped in awake patients who were undergoing carotid endarterectomy (CEA), ipsilateral rScO₂ was increased by the administration of 100% O₂ compared with 28% O₂. The underlying mechanism of this increase may relate to the associated increase in the O₂ content of the blood or to improvement in cerebral blood flow.[14] In patients undergoing CEA with general anesthesia, rScO₂ was reliably improved by increasing the FiO₂. [15] However, these two studies only described the phenomenon of correlated increases in rScO₂ and PaO₂; neither details regarding nor the exact mechanism underlying this phenomenon have been elucidated.

A study that utilized a regional real-time technique for measuring human cerebral microcirculation and rScO₂ by combined laser-Doppler flowmetry and spectroscopy also supports and explains our results.[16] An increase in the propofol dosage resulted in increased rScO₂ at 2 mm cerebral depth (gray matter) without coupled reductions in capillary venous blood flow. At 8 mm cerebral depth (white matter), the altered propofol dosage produced no observed effects on measured and calculated parameters. These findings suggest that cerebral metabolic demand in cortical regions may be reduced by propofol administration; however, the cerebral blood flow/cerebral metabolic rate of oxygen of white matter remains unaltered.[16] Propofol only affects the coupling of flow and metabolism in the cerebral microcirculation, resulting in increased capillary venous blood flow and rScO₂. Regardless of whether regional or cortical CBF is the factor that alters the rScO₂, one study determined that rScO₂ does not respond to a 100% increase in blood flow in the middle cerebral artery.[17] We also tested the correlated factors including HR, MAP and CO.

Flow-metabolism coupling remains intact during a stepwise increase in propofol after traumatic brain injury.[18] After 7 minutes of anesthesia induction in a normal brain, the effect-site propofol concentration[19] and PaO₂ become stable, and flow-metabolism coupling may function to balance the cerebral metabolism between gray matter and white matter. All of these responses are aspects of cerebral autoregulation, which we believe is the reason that rScO₂ returned to baseline at 15 minutes in our study.

Using the reasons described above, we can clearly determine exact explanations for changes in rScO₂ during anesthesia induction. The following reasons explain increases in rScO₂. 1) At the beginning of anesthesia induction, with increasing PaO₂, gray matter had regionally greater oxygen supply than demand; as a result, rScO₂ increased along with PaO₂. 2) An increase in propofol dosage resulted in an increased rScO₂ at 2 mm cerebral depth (gray matter) without coupled reductions in capillary venous blood flow. The following reason explains the return of rScO₂ to baseline. 1) At 15 minutes after induction, flow-metabolism coupling of white matter was functional, and cerebral autoregulation created a balance between white matter and NIRS-detected gray matter; as a result, rScO₂ returned to baseline (Figure 4A and B).

The following explanations for differences in the changes in $rScO_2$ reported by Nissen et al.[8] and Meng et al.[9] were provided by Dr. Meng. First, Meng et al. used frequency-domain NIRS; second, their patients were all intubated for ventilation; and third, they offered a unique interpretation of the mechanism underlying their observations. In our research, we provide additional details about the changes in $rScO_2$ each minute for 30 minutes for patients, all of whom were intubated, and we used the FORE-SIGHT oximeter to achieve more accurate monitoring.[13, 20] We also offered greater detail regarding the mechanism underlying our observations. The present study had several limitations. The patients in our study were all female, the sample size was small, and this investigation was a single-center study. Because there was insufficient space to attach both FORE-SIGHT sensors and bispectral index sensors, we did not monitor the bispectral index.

5. Conclusion

We concluded that during anesthesia induction, changes in $rScO_2$, which increased to a peak value at 7 minutes, may be correlated with PaO_2 ; the return to baseline at 15 minutes may have occurred due to flow-metabolism coupling and balancing between white matter and gray matter.

Abbreviations

$rScO_2$: regional cerebral oxygen saturation

MAP: Mean arterial pressure

HR: heart rate

PaO_2 : partial pressure of oxygen in arterial blood

$PaCO_2$: partial pressure of carbon dioxide in arterial blood

Hb: hemoglobin concentration

CO: cardiac output

NIRS: Near-infrared spectroscopy

ASA: American Society of Anesthesiologists

$EtCO_2$: end-tidal carbon dioxide

FiO_2 : inspired oxygen fraction

$PtIO_2$: Brain oxygen tension

CBF: cerebral blood flow

Declarations

Ethics approval and consent to participate

This cohort study was designed to examine changes in rScO₂ during anesthesia induction among patients undergoing breast cancer surgery. Major assessments were conducted from the onset of anesthesia induction to 30 minutes after induction. We followed recommendations in the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement in designing our study and reporting study findings. This study was approved by the Ethics Committee of the First Hospital of China Medical University (protocol no. 2015110301, Chairman Prof. Xinghua Gao, December 4, 2015) and was registered with the Clinical Trials Registry (NCT02687334). Website: <https://clinicaltrials.gov/ct2/results?term=NCT02687334&Search=Search>. Written informed consent was obtained from the parents or legal guardians of all participants in the trial.

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Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on request.

Conflicts of Interest

The authors have no conflicts of interest.

Funding

This work was funded a grant from the Natural Science Foundation of Liaoning Province (20180551176) to Wen-fei Tan and a grant from the Fund for Scientific Research of The First Hospital Of China Medical University (FHCMU-FSR) to Wen-fei Tan.

Contributions

TWF designed the study, interpretation of results and wrote the manuscript. SF collected data and wrote the manuscript. JF collected data and wrote the manuscript. MH statistical analysis and review of the manuscript. All authors read and approved the final manuscript.

Acknowledgement

None.

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Table

Table 1.

Patient characteristics (n=30).

Age (years)	49.4±9.6
BMI (kg/m ²)	23.2±2.5
MAP (mmHg)	81.1±11.7
HR (beats/minute)	77.6±12.3
Body temperature (°C)	36.3±0.1
PaO ₂ (mmHg)	86.9±4.9
PaCO ₂ (mmHg)	42.7±3.3
Hb (g/l)	131.1±12.4
CO (l/minute)	6.0±0.6
rScO ₂ at baseline (%)	67.3±3.9
rScO ₂ at 7 minutes (%)	81.7±1.6*
rScO ₂ at 15 minutes (%)	68.3±2.9

BMI, body mass index; MAP, mean arterial pressure; HR, heart rate; PaO₂, partial pressure of oxygen in arterial blood; PaCO₂, partial pressure of carbon dioxide in arterial blood; Hb, hemoglobin; CO, cardiac output; rScO₂, regional cerebral tissue oxygen saturation. *p<0.05 for Student's t-tests of rScO₂ at 7 minutes vs rScO₂ at baseline and rScO₂ at 7 minutes vs rScO₂ at 15 minutes.

Figures

Figure 1

Patient flowchart indicating the patients included in the enrollment, allocation, follow-up, and analysis phases of the study.

Figure 2

Time course of analysis for all variables. Thick red lines indicate median values. Thick black lines indicate upper and lower boundaries of 95% confidence intervals. 'Time 0' is the beginning of anesthesia induction. Data were collected every minute from induction to 30 minutes. MAP, mean arterial pressure (A); HR, heart rate (B); CO, cardiac output (C); Hb, hemoglobin concentration (D); PaO₂, partial pressure of oxygen in arterial blood (E); PaCO₂, partial pressure of carbon dioxide in arterial blood (F); rScO₂, regional cerebral tissue oxygen saturation (G). The interval between data points for each patient is 1 minute, with time points extending from anesthesia induction to 30 minutes later.

Figure 3

Correlations between regional cerebral tissue oxygen saturation (rScO₂) and MAP, mean arterial pressure (A); HR, heart rate (B); CO, cardiac output (C); Hb, hemoglobin concentration (D); PaO₂, partial pressure of oxygen in arterial blood (E); PaCO₂, partial pressure of carbon dioxide in arterial blood (F) (A-F, $p > 0.05$); and PaO₂ for the first 7 minutes (G) ($p < 0.01$). Lightly colored lines represent individual patients, and dark bold lines indicate averages. The interval between data points for each patient is 1 minute.

Figure 4

Theoretical analysis of the mechanism underlying changes in regional cerebral tissue oxygen saturation (rScO₂) following anesthesia induction (A). One example of real-time changes in rScO₂ following anesthesia induction (B).

Supplementary Files

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