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**Research Communication**

# CHANGES IN CEREBRAL OXYGENATION DURING CORONARY ARTERY BYPASS GRAFTING AND ITS DEPENDENCE ON HAEMATOCRIT, MEAN ARTERIAL PRESSURE AND PARTIAL PRESSURE OF OXYGEN IN ARTERIAL BLOOD

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**Abstract:**
**Background**: *Cerebral protection has always been an important concern during cardiac surgery. Near infrared spectroscopy (NIRS) can continuously monitor cerebral oxygenation and is increasingly being used as a surrogate measure to ensure the wellbeing of brain. This prospective observational study was designed to observe the changes in cerebral oxygenation in patients undergoing coronary artery bypass surgery (CABG) with the aid of cardiopulmonary bypass (CPB) during hypothermia and normothermia, and to determine if there was any correlation between the cerebral oximetry values and variables like hematocrit (Hct), mean arterial pressure (MAP), partial pressure of oxygen in blood (PaO2), CPB flows and temperature.*
**Methods**: *Forty patients scheduled to undergo elective CABG with the aid of CPB were enrolled in this study. The regional cerebral oxygen saturation (rso2), haematocrit, (MAP), PaO2, temperature and pump flows during CPB were measured at following time points during the surgery -T1:Baseline before induction of anaesthesia (on room air), T2:After induction of anaesthesia with a FiO2 of 100% , T3:After induction of anaesthesia with a FiO2 of 50% , T4:At the initiation of CPB (the lowest value of rso2 at the time of initiation of CPB), T5:On CPB at 35°C , T6:On CPB at 32°C, T7:On CPB after rewarming at 36°C , T8:After weaning from CPB with a FiO2 of 100% (after protamine administration) and T9:After weaning from CPB with a FiO2 of 50% (just before sternal closure). During CPB, pump flows were also recorded to find any deviation from the standard protocol.*
**Results**: *The mean baseline rso2 values were 64.35 and 64.97 for right and left frontal lobes, respectively and there was a relative increase in rso2 values with increase in PaO2 levels in the preCPB period. There was a maximum relative decrease of 12% in rso2 values with the initiation of CPB and the values remained below baseline throughout the hypothermic CPB. An insignificant decrease in rso2 values occurred with hypothermia which reversed at rewarming. The rso2 values reached baseline values in the post-CPB period. Based on post hoc analysis we observed that rso2 values could be predicted as 0.329 X per unit change in haematocrit; 0.133 X per unit change in MAP and 0.005 X per unit change in PaO2.*
**Conclusion:** *In patients undergoing cardiac surgery with CPB cerebral oximetry values were well maintained with maximal decrease of 12% at the time of initiation of CPB. Mild decrease in rso2 occurred with institution of CPB which reversed by the end of rewarming. The rso2 values differ insignificantly during hypothermic CPB. Cerebral oxygenation appears to be influenced by haematocrit, mean arterial pressure and partial pressure of oxygen in blood in pre, during and post CPB period.*

**Keywords**: *Cerebral; Oxygenation; Coronary; Artery; Grafting; Cardiopulmonary; Bypass*

## Introduction

In healthy adults normal cerebral blood flow (CBF) is approximately 50 ml/100g/min.[1] CBF depends on the cerebral perfusion pressure (CPP) which is defined as the difference of mean arterial pressure and intracranial pressure. Normal CPP in a healthy brain varies between 70-100 mm Hg. [2] If the CPP falls below 50 mm Hg there is slowing of brain potentials as measured by electroencephalography (EEG). At a CPP of 25-40 mm Hg the EEG becomes flat and at less than 25 mm Hg irreversible ischemic changes may occur in the brain.[3] Early investigators have suggested that cerebral blood flow (CBF) remains relatively constant at MAPs between 50–150 mm Hg.[4] Below the lower limit of autoregulation – a linear relationship exists between MAP and CBF. However, a recent study in awake, normotensive adults has shown that the mean lower limit of cerebral autoregulation is 73–88 mm Hg.[5,6] The lower limit of cerebral autoregulation may be as low as 20–30 mm Hg in anesthetized patients during hypothermic CPB, with moderate

hemodilution.[7,8] Advantages of lower MAPs during CPB include less trauma to blood elements and a reduction in noncoronary collateral flow to the heart. This reduction in collateral flow improves myocardial protection and maintains a clear surgical field. However, patients with advanced atherosclerotic disease of the aorta, advanced age, hypertension or diabetes may require higher CPP on CPB. There is a good amount of evidence to suggest that cerebral oxygenation is maintained even at lower than conventional flows on CPB with maintenance of CPP (1.2 vs 2.3 l/min/m2). [9] Therefore a monitor which would continuously reflect the cerebral oxygen saturation was long sought for. With the continuous monitoring of cerebral oxygen saturation it would be much safer to trade-off between the CPP and CPB flows.

Near Infrared Spectroscopy (NIRS) is used to measure the intravascular regional hemoglobin oxygen saturation (rSO2) of brain. NIRS optode contains an infrared light source which is transmitted through the skull to the outer layer of the cerebral cortex. The same optode has different sensors which distinguish between the photons reflected from skin, muscle, skull, dura and brain. Cerebral oximetric values measured by NIRS are thought to reflect 75% venous contribution. [10] Blood vessels larger than about 0.2 mm typically contain sufficient haemoglobin to trap all incident photons. [11] Thus, reflected photons represent partial photon absorption in the microcirculation which contains approximately 75% venous blood. In the absence of many noninvasive modalities to monitor perfusion of brain, during cardiac surgery on CPB, NIRS is increasingly routinely used. Apart from being noninvasive, it has a response time of 10.9 S[12] in respect to changes in CBF. It was found that if hemoglobin is less than 8.5g/dl and rSO2 is less than 60% blood transfusion consistently increased both variables, whereas, there was no effect on rSO2 if rSO2 was greater than 65%.[13] However, during CPB, evidence based guidelines suggest to maintain a hematocrit of 20. Many questions remain to be answered including do the rSO2 values fall significantly with hemodilution on CPB and is blood transfusion required to maintain these values? Does moderate hypothermia affect NIRS values? Will increasing the partial pressure of oxygen in arterial blood prevent cerebral desaturation? Therefore, we designed this study –

1. To observe the changes in cerebral oxygenation in patients undergoing CABG with the aid of CPB during hypothermia and normothermia, and
2. To find any correlation between the cerebral oximetry values and variables like hematocrit, mean arterial pressure, partial pressure of oxygen in blood, CPB flows and temperature.

## Patients and Methods

This observational study was conducted at our tertiary care centre after approval from the institutional ethics committee. After obtaining an informed consent, adult patients proposed to undergo coronary artery bypass grafting (CABG) with the aid of CPB by a single surgeon, over a period of 6 months, were included in this study. The exclusion criteria were emergency surgery, off pump CABG, evidence of carotid disease, history of cerebrovascular accident or syncope, evidence of any liver or kidney disease, acute coronary syndrome and severe uncontrolled hypertension (MAP>150 mmHg). All the patients underwent carotid and renal doppler evaluation prior to surgery.

Preoperatively, as per institutional protocol, all the patients received their cardiac medications in the morning on the day of surgery except for angiotensin converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs). Morphine sulfate (0.1 mg/kg) and promethazine (0.5 mg/kg) were administered, intramuscularly, as premedication 1 hour prior to surgery. In the operating room (OR), electrocardiogram (ECG), pulse oximetry, invasive arterial pressure monitoring, and central venous pressure monitoring were obtained. Two NIRS sensors (Equanox Advance sensor, model 8004CA) were applied on the forehead, one on each side and rSO2 was continuously monitored using Nonin EQUANOX cerebral oximeter (Nonin Medical Inc., Minnesota, USA). General anaesthesia was induced with intravenous midazolam 2 mg, fentanyl 3–5 micg/kg and thiopentone 3–5 mg/kg. Endotracheal intubation was facilitated with intravenous rocuronium bromide in a dose of 1 mg/kg. End tidal gases and nasopharyngeal temperature monitoring was instituted once the patient's trachea was intubated. The lungs were

mechanically ventilated with a tidal volume of 8 mL/kg and a mixture of air and oxygen in the ratio of 50:50 to attain a PaCO2 of 35–40 mmHg. In the pre CPB period anaesthesia was maintained with isoflurane 1 MAC, additional dose of fentanyl (2-5 micg/kg) and midazolam (0.05mg/kg). Maintenance of anaesthesia, during and after CPB, was done as per institutional protocol using additional boluses of fentanyl, midazolam and vecuronium bromide. Blood sugar levels were maintained below 200 mg/dl throughout the surgery. All patients underwent the same CPB protocol of hypothermic CPB (32 °C) using a hollow fibre membrane oxygenator and an arterial line filter of 40 micron. The CPB circuit was primed with a volume of 1500ml. During CPB, FiO2 and hematocrit were maintained at 50% and 20%, respectively. CPB flows were maintained at 2.4l/min/$m^2$ for temperature ≥36°C and at 2l/min/ $m^2$ during hypothermia. PCo2 was maintained between 35-40 mm Hg during the alpha stat management on CPB. At the time of rewarming, intravenous nitroglycerine and dopamine were started in the doses of 0.5 and 5µg/kg/min, respectively, to enable weaning from CPB. Haemoglobin was maintained between 8-9g percent after weaning from CPB. All the patients were rewarmed gradually to a nasopharyngeal temperature of 36 °C and pacing was instituted if the heart rate was less than 70 beats per minute. After surgery, all the patients were transferred to the intensive care unit (ICU).

Cerebral oximetry values (rSO2), of both right and left sides; mean arterial pressure, hematocrit, partial pressure of oxygen in blood (PaO2) and CPB flows were recorded at the following time intervals:

T1: Baseline before induction of anaesthesia (on room air)
T2: After induction of anaesthesia with a FiO2 of 100%
T3: After induction of anaesthesia with a FiO2 of 50%
T4: At the initiation of CPB (the lowest value of rso2 at the time of initiation of CPB)
T5: On CPB at 35°C
T6: On CPB at 32°C
T7: On CPB after rewarming at 36°C
T8: After weaning from CPB with a FiO2 of 100% (after protamine administration)
T9: After weaning from CPB with a FiO2 of 50% (just before sternal closure)

During CPB, pump flows were also recorded to find any deviation from the standard protocol.

**Statistical analysis**

Statistical Analysis was performed using SPSS 20 software. The demographic characteristics, ejection fraction, CPB time, aortic cross clamp (AoXC) time, rSO2, mean arterial pressure, hematocrit, PaO2, CPB flows, mediastinal chest tube drainage (MCTD), amount of PRBC and blood products transfused, duration of ICU and hospital stay were expressed as mean±SD. Incidence of diabetes, hypertension, unstable angina, postoperative atrial fibrillation, mediastinal exploration and use of IABP was expressed as proportion.

Repeated measures ANOVA was used to compare (within subject effects) the changes in values of right and left regional cerebral oximetric values, MAP, hematocrit and CPB flows at different time points.

The correlation between rso2 values and the other parameters (MAP, hematocrit and PaO2) was predicted by applying generalized estimating equation (GEE). With GEE, the correlation between rso2 (both right and left) and any one parameter was predicted by adjusting other variables and keeping them constant.

**Table 1:**
Demographic profile, comorbidities, number of coronary vessels involved, Ejection fraction, medications, CPB and AOXC time.

| S .No | Variable | Values |
|---|---|---|
| 1 | Age(y) | 57.42 ± 9.28 |
| 2 | Weight(kg) | 65.58 ± 10.13 |
| 3 | Height(cm) | 164.55 ± 7.25 |
| 4 | Body surface area(BSA)(m2) | 1.72 ± 0.15 |
| 5 | Gender (Males/Females) | 38/40 |
| 6 | Diabetics | 15/40 |
| 7 | Hypertensives | 25/40 |
| 8 | Smokers | 15/40 |
| 9 | History of unstable angina | 3/40 |
| 10 | History of myocardial infarction | 5/40 |
| 11 | History of both unstable angina and myocardial infarction | 1/40 |
| 12 | Number of coronary vessels involved {median (range)} | 3 (2-3) |
| 13 | Ejection fraction(%) | 50.65 ± 11.52 |
| 14 | Patients on nitrates | 37/40 |
| 15 | Patients on b blockers | 38/40 |
| 16 | Patients on ACEI/ARII antagonists | 22/40 |
| 17 | CPB time (minutes) | 83.58 ± 22.51 |
| 18 | AOXC time (minutes) | 39.10 ± 9.26 |

*CPB – cardiopulmonary bypass; AOXC – aortic cross clamp

rso2R: regional oxygen saturation of right side of frontal lobe; rso2L: regional oxygen saturation of left side of frontal lobe; MAP: mean arterial pressure; paO2: partial pressure of oxygen in arterial; blood; CPB: cardiopulmonary bypass.

**Table 2:**
Changes in cerebral oximetric values (right and left), haematocrit, mean arterial pressure, PaO2 and CPB flows during surgery.

| | rso2R (%) | rso2L (%) | Hematocrit (%) | MAP (mm Hg) | PaO2 (mm Hg) | CPB Flows (L/min) |
|---|---|---|---|---|---|---|
| T1 | 64.35±7.08 | 64.97±6.12 | 41.68±4.00 | 105.94±14.20 | 87.65±11.09 | - |
| T2 | 67.68±6.45 | 68.29±6.63 | 37.90±3.87 | 95.23±13.93 | 437.90±69.45 | - |
| T3 | 63.77±5.44 | 64.74±6.19 | 35.71±3.33 | 92.26±13.53 | 201.84±52.29 | - |
| T4 | 56.58±6.38 | 57.13±5.89 | 19.71±4.52 | 43.55±12.00 | 272.32±56.65 | 1.95±0.47 |
| T5 | 59.00±4.17 | 59.61±5.37 | 18.74±2.86 | 55.77±9.15 | 275.87±33.95 | 2.50±0.24 |
| T6 | 56.94±4.95 | 58.03±5.19 | 19.77±2.19 | 63.58±10.68 | 246.52±50.44 | 2.17±0.23 |
| T7 | 58.65±6.17 | 59.71±5.60 | 21.45±2.34 | 62.84±11.04 | 242.10±50.04 | 2.48±0.20 |
| T8 | 64.94±6.23 | 64.77±7.37 | 23.74±2.37 | 74.26±11.04 | 267.87±85.22 | - |
| T9 | 62.39±5.54 | 62.39±6.46 | 26.94±2.95 | 81.06±15.43 | 126.06±56.60 | - |

T1:Baseline before induction OF anaesthesia(FiO2 21%) ; T2:After induction of anaesthesia with a FiO2 of 100% ; T3:After induction of anaesthesia with a FiO2 of 50% ; T4: At the initiation of CPB; T5:On CPB during cooling at 35°C ; T6:On CPB during hypothermia at 32°C; T7:On CPB during rewarming at 36°C; T8:After weaning from CPB with a FiO2 of 100%; T9:After weaning from CPB with a FiO2 of 50%.

## Results

The demographic profile of the 40 patients is presented in table 1. The average ejection fraction, CPB time and AOXC time was 50.65±11.52%, 83.58 ± 22.51 and 39.10 ± 9.26, respectively.

The baseline mean rso2 values in our patients were found to be 64.35±7.08 (range, 50-85) and 64.97±6.12 (range, 51-82) for right and left side, respectively. There was a significant relative increase (5.1%) in both rso2R and rso2L at T2 (after intubation and on 100% FiO2) in comparison to baseline values. However both values decreased at T3 (on 50% FiO2) in comparison to T2. There was an average relative decrease of 12.0% in the values of rso2R and rso2L at T4, that is, at the time of initiation of CPB. Once full CPB flows were achieved (T5) the rso2R and rso2 L significantly increased. With the institution of hypothermia (32°C) on CPB (T6) there was a significant decrease in oximetry values which improved after the patient was rewarmed to a nasopharyngeal temperature of 36°C(T7). There was no significant difference between rso2 values at T5 and T7. After weaning off CPB, at T8 and T9, the oximetric values tend to approach the baseline values and were not significantly different from the rso2R and rso2L, at T1 and T3. In our patients both rso2R and rso2L followed the same trends during surgery with only minor differences. Although the rso2 values over the duration of surgery significantly differed on pairwise comparisons; at no point of time did the NIRS values fall below 50%.

The baseline (T1) hematocrit value was 41.68±4.00 which significantly decreased at T3. At time T4 there was a steep decline in hematocrit from 35.71±3.33 to 19.71±4.52. Thereafter there was no significant change in the hematocrit values at T5 and T6 during CPB. After full rewarming, at T7 there was a significant increase in hematocrit, in comparison to T6, which persisted through T8 and T9.

There was a significant decrease in MAP at T2 (95.23±13.93 mm Hg), compared to baseline value at T1 (105.94±14.20 mm Hg). No significant change in MAP occurred until institution of CPB, at which (T4) the MAP was lowest (43.55±12.00mm Hg) during the whole surgical period. At T5 and T6 the MAP significantly increased and at T7 there was an insignificant fall in MAP. The increase in MAP at T8 and T9 was statistically significant.

The baseline PO2 at T1 was 87.65±11.09 mm Hg. Immediately after intubation at T2 the PO2 values significantly increased to 437.90±69.45 mm Hg and at T3 declined to 201.84±52.29mm Hg. At T4 the PO2 increased significantly with no significant change between T4 –T5, T6-T7 and T7-T8 comparisons. At T6 the PO2 decreaesd siginificantly in comparison to T5 as it did at T9 in comparison to T8.

At initiation of CPB (T4) the flows were lowest with a mean value of 1.95±0.47 l/min/m2. Then there was a significant increase in flows to 2.50±0.24 l/min/m2 at T5. During hypothermia, at T6, the flows were significantly less with a mean of 2.17±0.23 l/min/m2. At T7 (nasopharyngeal temperature of 36°C) there was an increase in the CPB flows which were not significantly different from flows at T5.

It was found that a change in the hematocrit of the patient by one unit lead to a change in rso2R by a factor of 0.329 and in rso2L by 0.343, both of which were statistically significant (table3). Similarly, an increase or decrease of MAP by 1mm Hg caused a statistically significant change in the values of rso2R and ros2L by a factor of 0.133 and 0.130, respectively. However when regional cerebral oxygen saturation was correlated with PO2 in arterial blood there was a very weak correlation between them which was not statistically significant ($p>0.05$). For every 1 mm Hg change in PaO2 both rso2R and rso2L changed by a factor of 0.005 only. Thus, it may be possible to predict the change in cerebral oximetry values per unit change of other variables like hematocrit, MAP and PaO2 on which it depends.

**Table 3:**
Coefficients of correlation between rso2 and different variables when generalized estimating equation is applied on a population-averaged model

| Variables | Coefficient of correlation | P value |
|---|---|---|
| rso2R with haematocrit | 0.329 | <0.001 |
| rso2R with MAP | 0.133 | <0.001 |
| rso2R with PO2 | 0.005 | 0.07 |
| rso2L with haematocrit | 0.343 | <0.001 |
| rso2L with MAP | 0.130 | <0.001 |
| rso2L with PO2 | 0.005 | 0.09 |

rso2R: regional oxygen saturation of right side of frontal lobe; rso2L: regional oxygen saturation of left side of frontal lobe; MAP: mean arterial pressure; paO2: partial pressure of oxygen in arterial; blood;

**Table 4:**
Shows the postoperative variables and the incidence of neurological Complications. Postoperative data

| S No | Variable | Values |
|---|---|---|
| 1 | Mediatinal chest cube drainage (ml) | 695.86 ± 613.08 |
| 2 | Packed red blood cells (ml) | 740.69 ± 592.19 |
| 3 | Fresh frozen plasma (ml) | 473.97 ± 341.04 |
| 4 | Platelet concentrate (ml) | 93.10 ± 80.75 |
| 5 | Patients requiring IABP postoperatively | 4/40 |
| 6 | Incidence of atrial fibrillation | 6/40 |
| 7 | Incidence of mediastinal exploration | 4/40 |
| 8 | Duration of mechanical ventilation (h) | 25.50 ± 22.92 |
| 9 | Duration of ICU stay (h) | 78.20 ± 47.87 |
| 10 | Neurological complication | 2/40 (Delusion of persecution, Delerium) |

IABP: Intraaortic balloon pump; ICU: intensive care unit

## Discussion

Near infrared spectroscopy is commonly used during cardiac surgery to monitor cerebral oxygen saturation (rSO2). [14] With the advent of NIRS which can continuously measure the intravascular regional brain saturation, even during CPB, cerebral oximetric values are considered as surrogate indices to ensure cerebral wellbeing during surgery. Also, cerebral saturation has been extensively studied as a marker of postoperative cognitive dysfunction [15,16], delirium[17] and longer hospital stays[15]. However, only few studies have studied the determinants of cerebral oxygenation during cardiac surgery, especially during CPB. Cerebral saturation depends on mean arterial pressure, hematocrit, temperature during CPB, PaO2 and partial pressure of carbon dioxide in blood(PCO2) [18-20]. In this study we studied the determinants of regional cerebral oxygenation and the correlation between oximetric values and the factors under evaluation.

The mean baseline oximetric values in our patients, who were Asian in origin, were 64.35±7.08 and 64.97±6.12 for right and left side, respectively. A recently published outcome study by Mohandas et al [21], mentioned the baseline rso2 values for right and left as 65.78 and 66.32 which are almost similar to our baseline rso2 values. Although the patients in this study were also Asian the mean age of patients was 36.32 years (mean of control and intervention groups) compared to our patients whose mean age was 57.42 years. There is a predominance of males (95%) in our study group compared to Mohandas et al where 44% patients were females[21].

Another study [22] of 1000 patients (age range, 21 to 91 years and females 32%) in a different population subset found the baseline values to be 67± 10 which is in accordance to our findings. The mean difference between right and left values in this study was zero. There was no statistically significant difference between right and left rso2 values in our study but it was not zero as well may be because of small sample size.

After induction of general anaesthesia when the patient was on 100% oxygen there was a significant increase of 5% in rso2 values compared to baseline even when the hematocrit and MAP decreased significantly compared to their baseline values. Thus increasing the partial pressure of oxygen in blood (PaO2) may increase the rso2 values significantly in the pre-CPB period atleast in patients who have no neurologic disease. Mohandas et al in their study attributed this initial increase of rso2 values to anaesthesia-induced reduction in cerebral oxygen demand. If that was the case, the rso2 values would not have dropped proportionately when FiO2 levels were decreased to 50% in our study because the suppression would have been uniform. There was no statistically significant change in MAP and hematocrit during these two (T2 and T3) time points.

During cardiac surgery the maximum decrease in the rso2 values (12%) occurred at the time of initiation of CPB. This is quite explainable as at the initiation of CPB there is sudden hemodilution, mean arterial pressure falls suddenly to ≤50% and CPB full flows are not yet achieved. Another study which also demonstrated a decrease in rso2 values on CPB failed to pinpoint the stage of CPB [21]. The oximetric values improved significantly once full CPB flows were established but were less than the baseline values during the whole CPB period. During hypothermic CPB (lowest temperature 32°C) there was not much variation in the cerebral oxygenation. The differences that occurred were due to changes in MAP and hematocrit changes. Since all our patients underwent hypothermic CPB the imbalance between CMRO2 and CBF would have been less. Kadoi et al [23] compared normothemic and hypothermic CPB and concluded that rso2 values were stable throughout the perioperative period in patients undergoing hypothermic CPB. Although, they did not mention the values of rso2, the graph did show some decrease in rso2 values during CPB which might not have been significant. After full rewarming there was an increase in rso2 values as occurred in our study also? But some studies mention there is a decrease in the rso2 values with the onset of rewarming. Based on the conclusion of a study done by Chowdhury et al [24] we routinely increase the CPB flows at the time of rewarming to match the increased oxygen requirements of the brain. This might be an explanation for increase in rso2 values after full rewarming in our patients. In the study of Kadoi et al[16] they used an infusion of phenylephrine to maintain higher CPB pressures which may have influenced oximetry values at rewarming. On the contrary in our study the MAP slightly decreased during rewarming.

T1:Baseline before induction of anaesthesia(FiO2 21%); T2:After induction of anaesthesia with a FiO2 of 100% ; T3:After induction of anaesthesia with a FiO2 of 50% ; T4: At the initiation of CPB; T5:On CPB during cooling at 35°C ; T6:On CPB during hypothermia at 32°C; T7:On CPB during rewarming at 36°C; T8:After weaning from CPB with a FiO2 of 100%; T9:After weaning from CPB with a FiO2 of 50%.

The NIRS values reached the baseline values after weaning off from CPB as did occur in other studies. But these values were achieved at much lower hematocrits and MAPs compared to baseline (23.74 ±2.37 vs 41.68±4.00 and 74.26±11.04 vs 105.94±14.20, respectively). The PaO2 values were much higher (FiO2 100%) after weaning off from CPB suggesting the direct influence of PaO2 on rso2 values. At T9 although the MAP and hematocrit increased the rso2 values decreased because of decrease in PaO2 (FiO2 50%) again reiterating the former explanation. Another factor that could cause an elevation of rSO2 post CPB is post ischemic hyperaemia.

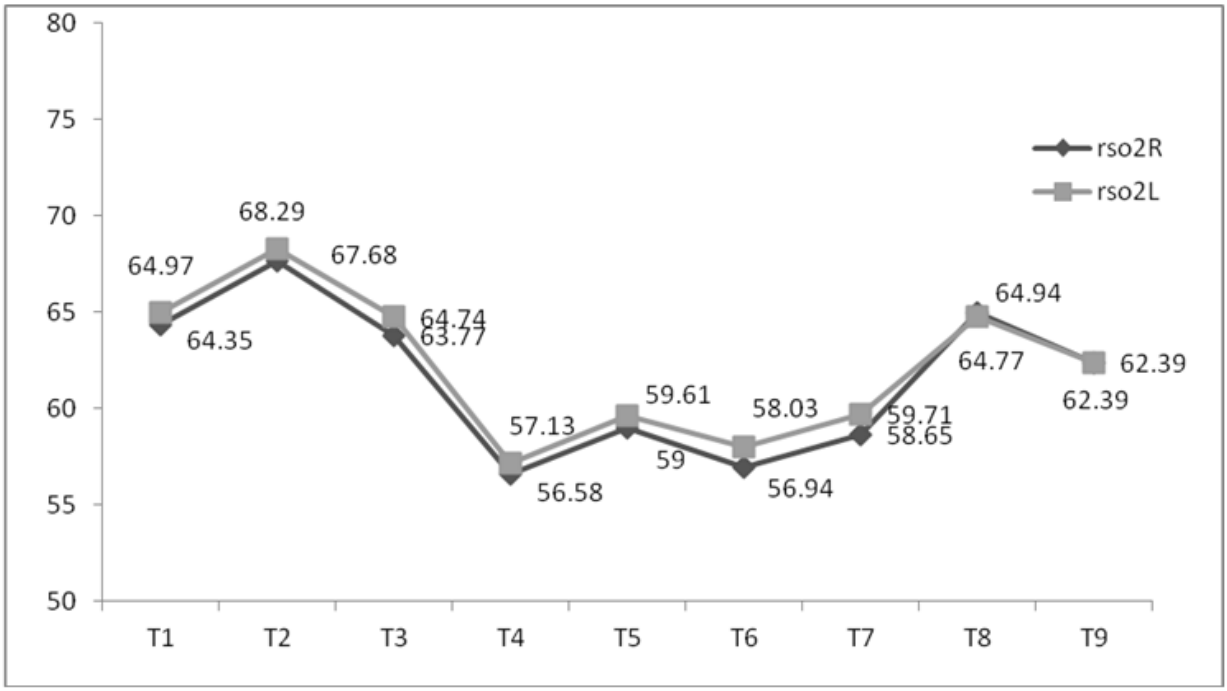


**Figure 1:** Changes in cerebral oxygen saturation (rso2R & rso2 L) at different time intervals during the study period.

The NIRS values increase in response to increase in PCO2 [25] as well but in our study we maintained PCO2 between 35 -40 mmHg to avoid increase in CBF (containing embolic load) that occur at higher PCo2 levels.

In this study we were able to detect a relation between change in rso2 values and different variables. Based upon this post hoc analysis we observed that the change in rso2 value can be predicted as 0.329 X per unit change in hematocrit; 0.133 X per unit change in MAP and 0.005 X per unit change in PaO2. However, the p value for correlation between rso2 and PaO2 was >0.05 (table 3) indicating that the relation is not significant. This is because of the fact that we had high PaO2 levels for most of the duration (T2-T9) of surgery at which the haemoglobin dissociation curve shifts towards the right. Therefore, although not applicable to lower PaO2 levels the correlation between rso2 and PaO2 appears to be valid for PaO2 levels more than 100 mm Hg.

Two patients in our study developed neurological complications one in the form of delusion of persecution and other became delirious. Both the patients recovered on 2nd and 3rd postoperative day, respectively without any further sequelae. Both of these patients underwent a mediastinal reexploration, required IABP support, developed atrial fibrillation and had received excessive blood transfusion.

**Limitation of study**

Our study is an observational study done in relatively healthier subset of patients. Suppression of cerebral oxygen demand would only be constant if anaesthesia levels remained constant, a variable we cannot be sure of in an observational study.

## Conclusion

In our study of patients undergoing cardiac surgery during mild hypothermic CPB cerebral oximetry values were well maintained with maximal decrease of 12% at the time of initiation of CPB. Mild decrease in rso2 occurs with institution of CPB which reverses by the end of rewarming. The rso2 values differ insignificantly during hypothermic CPB. Cerebral oxygenation is likely influenced by hematocrit, mean arterial pressure and partial pressure of oxygen in blood among many other factors in pre, during and post CPB period. This requires confirmation with larger blinded studies.

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