INDONESIAN JOURNAL OF ANESTHESIOLOGY AND REANIMATION
Volume 7 (1), January 2025: 1-11

Original Research Article

CEREBRAL OXYGENATION MONITORING DURING CORONARY ARTERY BYPASS GRAFTING AND ITS CORRELATION WITH HEMATOCRIT, MEAN ARTERIAL PRESSURE, AND PARTIAL PRESSURE OF OXYGEN IN ARTERIAL BLOOD

Jai Sharma[1a], **Indu Verma**[1], **Swati Agarwal**[1], **Nivedita Dagar**[1]
[1] Department of Anaesthesiology, Swai Man Singh Medical College, Jaipur, India
[a]**Corresponding author**: jays80425@gmail.com

ABSTRACT

Introduction: Optimal cerebral oxygenation is vital during coronary artery bypass grafting (CABG) to prevent neurological complications like cognitive decline and stroke. Non-invasive monitoring methods include near-infrared spectroscopy (NIRS), electroencephalography (EEG), and transcranial doppler (TCD). It offers real-time rSO2 assessment, detecting critical thresholds and reducing risks during cardiopulmonary bypass (CPB). **Objective:** This observational study aims to investigates cerebral oxygenation changes during CABG and correlations with hematocrit, mean arterial pressure (MAP), blood oxygen levels, CPB flows, and temperature. **Methods**: Seventy-two elective CABG patients underwent CPB with parameters including rSO2, hematocrit, MAP, PaO2, temperature, and pump flows assessed at specific time points: T1: Baseline pre-anesthesia; T2: Post-anesthesia induction (FiO2 100%); T3: Post-anesthesia induction (FiO2 50%); T4: CPB initiation; T5: CPB at 35°C; T6: CPB at 32°C; T7: CPB rewarming (36°C); T8: Post-CPB weaning (FiO2 100%); T9: Post-CPB weaning (FiO2 50%). **Results**: The mean baseline values for rSO2 were 72.14 for the right side and 71.90 for the left side. Upon initiating CPB at 35°C, a significant maximum reduction in rSO2 of 10.5% was observed, which remained below baseline during the hypothermia phase. The rSO2 values began to increase during the rewarming phase, nearly reaching baseline levels after CPB. A post hoc analysis indicated that changes in rSO2 were correlated with variations in hematocrit (correlation coefficient = 0.518), MAP (correlation coefficient = 0.399), and PaO2 (correlation coefficient = 0.001). **Conclusion:** This study explored the fluctuations in rSO2 during CABG with CPB and examined its correlations with hematocrit, MAP, PaO2, CPB flows, and temperature. The findings highlight significant correlations among these variables, providing insights into factors influencing cerebral oxygenation during cardiac surgery.

Keywords: Cardiopulmonary Bypass; Coronary Artery Bypass Grafting (CABG); Cerebral Oxygenation; Coronary artery disease; Near-Infrared Spectroscopy (NIRS)

ABSTRAK

Pendahuluan: Oksigenasi serebral yang optimal sangat penting selama operasi bypass arteri koroner atau *coronary artery bypass grafting* (CABG) untuk mencegah komplikasi neurologis seperti penurunan kognitif dan stroke. Metode pemantauan non-invasif termasuk spektroskopi inframerah dekat atau *near-infrared spectroscopy* (NIRS), elektroensefalografi (EEG), dan *transcranial doppler* (TCD). Metode ini menawarkan hasil penilaian rSO2 secara *real-time*, mendeteksi nilai ambang kritis dan mengurangi risiko selama bypass kardiopulmoner atau *cardiopulmonary bypass* (CPB). **Tujuan:** Studi observasional ini bertujuan untuk menganalisis perubahan oksigenasi serebral selama operasi CABG dan korelasinya dengan hematokrit, tekanan arteri rata-rata (MAP), kadar oksigen darah, aliran CPB, dan suhu. **Metode:** Tujuh puluh dua pasien CABG elektif menjalani CPB dengan parameter antara lain; rSO2, hematokrit, MAP, PaO2, suhu, dan aliran pompa yang dinilai pada titik waktu tertentu: T1: *Baseline* pra-anestesi; T2: Post-induksi anestesi (FiO2 100%); T3: setelah induksi anestesi (FiO2 50%); T4: Inisiasi CPB; T5: CPB pada 35°C; T6: CPB pada 32°C; T7: Pemanasan ulang CPB (36°C); T8: *Weaning* setelah CPB (FiO2 100%); T9: *Weaning* setelah CPB (FiO2 50%). **Hasil:** Nilai rata-rata *baseline* untuk rSO2 adalah 72,14 pada sisi kanan dan 71,90 pada sisi kiri. Setelah memulai CPB pada suhu 35°C, penurunan

Available at https://e-journal.unair.ac.id/IJAR | **DOI:** https://doi.org/10.20473/ijar.V7I12025.1-11

maksimum rSO2 sebesar 10,5% diamati, dimana tetap berada di bawah baseline selama fase hipotermia. Nilai rSO2 mulai meningkat selama fase pemanasan ulang, hampir mencapai tingkat baseline setelah CPB. Analisis post hoc menunjukkan bahwa perubahan rSO2 berkorelasi dengan variasi hematokrit (koefisien korelasi = 0,518), MAP (koefisien korelasi = 0,399), dan PaO2 (koefisien korelasi = 0,001). **Kesimpulan:** Studi ini mengeksplorasi fluktuasi rSO2 selama CABG dengan CPB dan meneliti korelasinya dengan hematokrit, MAP, PaO2, aliran CPB, dan suhu. Temuan ini menyoroti korelasi signifikan di antara variabel-variabel ini, memberikan wawasan tentang faktor-faktor yang mempengaruhi oksigenasi serebral selama operasi jantung.

Kata kunci: *Cardiopulmonary Bypass*; *Coronary Artery Bypass Grafting* (CABG); Oksigenasi serebral; penyakit arteri koroner; *Near-Infrared Spectroscopy* (NIRS)

Article info: Received: June 18, 2024; Revised: August 9, 2024; Accepted: December 3, 2024; Published: January 30, 2025

INTRODUCTION

Optimal cerebral oxygenation is crucial for maintaining brain function and preventing cerebral injury. The observation of cerebral oxygenation during Coronary artery bypass grafting (CABG) surgery has gained increasing attention in recent years as a means of identifying and mitigating potential cerebral oxygen imbalances. Despite progress, cerebral complications during and after CABG with Cardiopulmonary bypass (CPB) pose risks due to the brain's vulnerability to ischemic events, leading to cognitive decline, stroke, and mortality (1). A significant correlation exists between regional cerebral oxygen desaturation and neurological complications after cardiac surgery (2). Monitoring regional cerebral oxygen saturation (rSO_2) has predictive value in decreasing the incidence of early postoperative cognitive decline (3). Therefore, maintaining optimal cerebral oxygenation during CABG is crucial to alleviate these risks.

Numerous non-invasive cerebral oxygenation monitoring techniques exist, including near-infrared spectroscopy (NIRS), Electroencephalography (EEG), and transcranial Doppler (TCD) ultrasound. EEG has proven useful for early detection of imbalances between cortical tissue oxygen supply and demand (4). However, EEG monitors are not highly specific for ischemic injuries, as such imbalances are not always caused by blood flow variations (4). Other factors, such as non-convulsive seizures or prior sub-clinical traumatic cortical injuries, can also influence the readings (5). TCD complements EEG by assessing blood flow velocity in cerebral arteries, aiding in emboli detection (4). However, maintaining a stable probe position during surgery is challenging, as it involves securing the probe in place with a sterile sleeve using a band strapped around the patient's head (4). NIRS emerges as a superior alternative since near-infrared light can easily penetrate the skull, it enables real-time assessment of rSO2 using sensors placed on the patient's forehead (6). By monitoring both hemispheres, this technology can distinguish between global and unilateral causes of hypoperfusion, such as changes in head position or unilateral vessel occlusion (4). Additionally, it does not rely on pulsatile blood flow, making it particularly advantageous during cardiopulmonary bypass procedures (4). In addition to being noninvasive, NIRS has a response time of 10.9 seconds (7) about to changes in CBF. NIRS is established as a comprehensive and effective neuromonitoring modality in cardiovascular surgeries, surpassing the limitations associated with EEG and TCD.

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Interrupted blood circulation during CABG makes the brain vulnerable to ischemic incidents, manifesting as temporary impairments or severe conditions like as stroke. An rSO2 level below 45% or a decrease of 25% from individual baseline values is considered a critical threshold, indicating a higher risk of adverse neurological outcomes (6).

Numerous unresolved questions remain about the effects of hemodilution, hypothermia, and PaO2 on cerebral oxygenation. This study aimed to investigate alterations in cerebral oxygenation in patients undergoing CABG with CPB and examine potential correlations between cerebral oximetry measurements and factors such as hematocrit levels, mean arterial pressure, arterial oxygen levels, CPB flows, and temperature.

METHODS

This observational study was conducted at Swai Man Singh Medical College, Jaipur from September to December 2023, following approval from the office of the ethics committee, S.M.S. Medical College and attached hospitals, Jaipur (No. 199/MC/EC/2023 Dated 05th April 2023) and registration with the Clinical Trials Registry India (CTRI/2023/07/055738 Dated 26th July 2023). The study included adult patients scheduled for CABG with CPB under the care of a single surgeon, who met the inclusion criteria. Patients were excluded if they required emergency surgery, underwent off-pump CABG, had evidence of carotid disease, had a history of cerebrovascular accident or syncope, suffered from liver or kidney disease, acute coronary syndrome, or severe uncontrolled hypertension (MAP > 150 mmHg).

A total of 72 patients were selected for the study by purposive sampling. Upon arrival in the operating theatre, the patient's fasting status, written informed consent, and pre-anesthetic assessment results were verified. Standard routine monitors, including Non-invasive blood pressure (NIBP), oxygen saturation (SpO2) probe, and Electrocardiogram (ECG), were applied, and baseline parameters (heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, and oxygen saturation) were recorded. Femoral arterial cannulation for invasive blood pressure monitoring and right internal jugular vein cannulation were performed under local anesthesia.

Two NIRS sensors (ForeSight Elite large sensor, model FSESL) were positioned bilaterally on the forehead, and rSO2 was constantly measured with the ForeSight Tissue Oximeter Monitor (Edwards Life Sciences). Measurements of rSO2R, rSO2L, hematocrit, MAP, and PaO2 were obtained at T1 [Baseline before anesthesia induction (FiO2 21%)]. Preoxygenation was conducted using 100% oxygen for a duration of 3 to 5 minutes. General anesthesia was induced with intravenous midazolam (0.05 mg/kg), fentanyl (3-5 mcg/kg), and etomidate (3-5 mg/kg). Endotracheal intubation was facilitated with intravenous rocuronium bromide (1 mg/kg), and the correct position of the tube was confirmed by 5-point auscultation and EtCO2 measurements.

Monitoring of end-tidal gases and nasopharyngeal temperature commenced post-intubation. Mechanical ventilation was configured with a tidal volume of 8 - 10 mL/kg and a 50:50 mixture of air and oxygen to achieve a PaCO2 of 35–40 mmHg. During the pre-CPB period, anesthesia was maintained with sevoflurane (1 MAC), supplemented with fentanyl (2-5 mcg/kg) and midazolam (0.05 mg/kg). Anesthesia management was maintained consistently during and after CPB

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according to institutional protocols, incorporating additional boluses of fentanyl, midazolam, and vecuronium bromide. Blood sugar levels were maintained below 180 mg/dL during the surgery in all patients.

Measurements of rSO2R, rSO2L, hematocrit, MAP, PaO_2, and CPB flows were recorded at various time points:

T1 - Baseline before anesthesia induction (FiO2 21%)
T2 - post-anesthesia induction with fio2 at 100%
T3 - post-anesthesia induction with fio2 at 50%
T4 - start of CPB
T5 - on CPB at 35°C
T6 - on CPB at 32°C
T7 - on CPB after rewarming to 36°C
T8 - post-weaning from CPB with fio2 at 100% after protamine administration
T9 - post-weaning from CPB with fio2 at 50% before sternal closure

The CPB protocol included hypothermic CPB at 32°C using a hollow fiber membrane oxygenator and a 40-micron arterial line filter, with the CPB circuit primed with 1800 ml. During CPB, FiO2 was maintained at 100% and the hematocrit at 25%. Hypothermic CPB (32°C) was initiated at flow rates of 2.5 L/min/m², pCO2 levels were controlled between 35-40 mmHg using alpha-stat management. Intravenous nitroglycerin and dobutamine were administered at dosage 0.5 and 5 µg/kg/min, respectively, during rewarming to aid in weaning from CPB. Hemoglobin levels were maintained between 8-9 g/dL post-CPB. The rewarming process to achieve a nasopharyngeal temperature of 36°C was conducted gradually, with pacing initiated if the heart rate dropped below 60 bpm. After surgery, patients were transferred intubated to the Cardiac Surgery ICU for monitoring of postoperative complications.

Statistical analysis

Continuous variables, including rSO2, MAP, hematocrit, PaO2, and CPB flows, were presented as mean ± SD. Categorical variables were reported as frequencies and proportions. Comparison of changes in continuous variables at different time points were conducted using repeated measures ANOVA (within-subject effects). Pearson's correlation coefficient was used to evaluate the relationship between continuous variables. The independent correlation of rSO2 with other parameters was determined using a generalized estimating equation within a population-averaged model. A p-value of <0.05 was considered statistically significant. All statistical analyses were conducted using the trial version of SPSS 22 software.

RESULTS AND DISCUSSION

The demographic characteristics of 72 patients who underwent cardiac surgery was presented at the Table 1. The average ejection fraction was 52.33 ± 5.33%, whereas the duration for CPB and Cross-clamp was 76 ± 22 and 54.40 ± 13.5, respectively.

The initial mean rSO2 values were 72.14 ± 8.96% for the right side (rSO2R) and 71.90 ± 8.05% for the left side (rSO2L) at T1. Following intubation and administration of 100% FiO2 at T2, both rSO2R and rSO2L demonstrated a statistically significant relative increase, reaching 73.50 ± 9.17% and 73.01 ± 9.17%, respectively. However, a decline in these values was observed at T3 (50% FiO2). At T4, coinciding with the initiation of cardiopulmonary bypass (CPB) at 35°C, there was a significant decrease in both rSO2R and rSO2L.

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Table 1. Demographic profile, comorbidities, number of coronary vessels involved, Ejection fraction, medications, and CPB (N=72)

Variable	Values
Age (years) [Mean ± SD]	53.15 ± 8.05
Weight (kgs) [Mean ± SD]	62.5 ± 7.36
Body surface area (meter^2) [Mean ± SD]	1.69 ± 0.16
Gender	
Male [N (%)]	50 (69.4)
Female [N (%)]	22 (30.6)
Diabetic Patients [N (%)]	24 (33.33)
Hypertensive Patients [N (%)]	38 (52.8)
Smokers [N (%)]	32 (44.44)
History of Unstable angina [N (%)]	9 (1.25)
History of myocardial infarction [N (%)]	16 (22.22)
History of both unstable angina and myocardial infarction [N (%)]	4 (5.56)
Number of involved coronary vessels {Mean ± SD (range)}	2.5 ± 0.5(2-3)
Ejection fraction (%) [Mean ± SD]	52.33 ± 5.33
Patients taking nitrates [N (%)]	63 (87.5)
Patients taking β-blockers [N (%)]	67 (93.056)
Patients taking ACEI/ARII antagonists [N (%)]	38 (52.8)
Cardiopulmonary Bypass time (minutes) [Mean ± SD]	76 ± 22
Cross-clamp duration (minute) [Mean ± SD]	54.40 ± 13.5

During CPB (T5), both rSO2R and rSO2L reached their lowest values, with a maximum relative decrease of 10.5% observed. During hypothermia (32°C) at T6, there was a slight increase in oximetry values, which further improved during rewarming to 36°C at T7. Post-CPB (T8 and T9), oximetry values approached baseline and were not significantly different from those at T1. Both rSO2R and rSO2L exhibited comparable patterns during surgery, with minor differences noted, and no measurements dropping below the 50% threshold [Table 2].

Table 2. Variations in cerebral oximetry values (right and left), hematocrit, MAP, PaO2, and CPB flow rates throughout the surgery

Time points	rSO2R (Mean±SD)	rSO2L (Mean±SD)	Hematocrit (Mean±SD)	MAP (Mean±SD)	PaO2 (Mean±SD)	CPB Flows (Mean±SD)
T1	72.14 ± 8.96	71.90 ± 8.05	40.80 ± 6.88	84.06 ± 12.14	69.63 ± 23.29	_
T2	73.50 ± 9.17	73.01 ± 9.17	38.81 ± 7.53	85.53 ± 14.23	581.80 ± 90.68	_
T3	71.70 ± 8.83	73.71 ± 8.48	36.79 ± 8.43	83.01 ± 16.42	389.21 ± 106.68	_
T4	68.01 ± 8.56	70.50 ± 8.96	29.49 ± 5.6	70.04 ± 19.83	515.30 ± 85.37	3 ± 0.42
T5	64.59 ± 8.96	67.39 ± 9.38	27.50 ± 5.63	67.02 ± 15.46	231.15 ± 102.4	3.06 ± 0.4
T6	65.50 ± 8.78	66.71 ± 9.43	27.19 ± 6.21	67.53 ± 12.78	363.08 ± 87.04	3.02 ± 0.47
T7	66.51 ± 9.82	67.63 ± 8.71	27.09 ± 5.81	69.02 ± 15.87	507.83 ± 77.52	3.07 ± 0.45
T8	68.80 ± 9.42	71.92 ± 8.77	28.80 ± 5.38	71.52 ± 13.38	404.83 ± 66.91	_
T9	69.60 ± 10.3	73.94 ± 9.21	29.81 ± 5.75	75.01 ± 16.19	362.78 ± 99.88	_

MAP: Mean arterial pressure; **PaO$_2$**: Partial pressure of oxygen in arterial blood; **CPB**: Cardiopulmonary Bypass

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The baseline hematocrit at T1 was 40.80 ± 6.88, significantly decreasing at T3. A significant decrease occurred at T4 (36.79 ± 8.43 to 29.49 ± 5.6). No significant changes were observed in hematocrit levels at T5 to T7 during CPB. After complete rewarming at T9, hematocrit increased in comparison to T5 [Table 2]. Initially, at baseline (T1), both rSO2R and rSO2L showed a weak positive correlation with hematocrit. After the administration of anesthesia (at T2 and T3), the correlation for both rSO2R and rSO2L increased to moderate levels. During the CPB stages, which included cooling, hypothermia, and rewarming (T4 to T7), the correlations for both rSO2R and rSO2L fluctuated but showed statistically significant associations, indicating varying influences of hematocrit. Post-CPB, the correlations weakened again, highlighting minimal relationships in later stages [Figure 1].

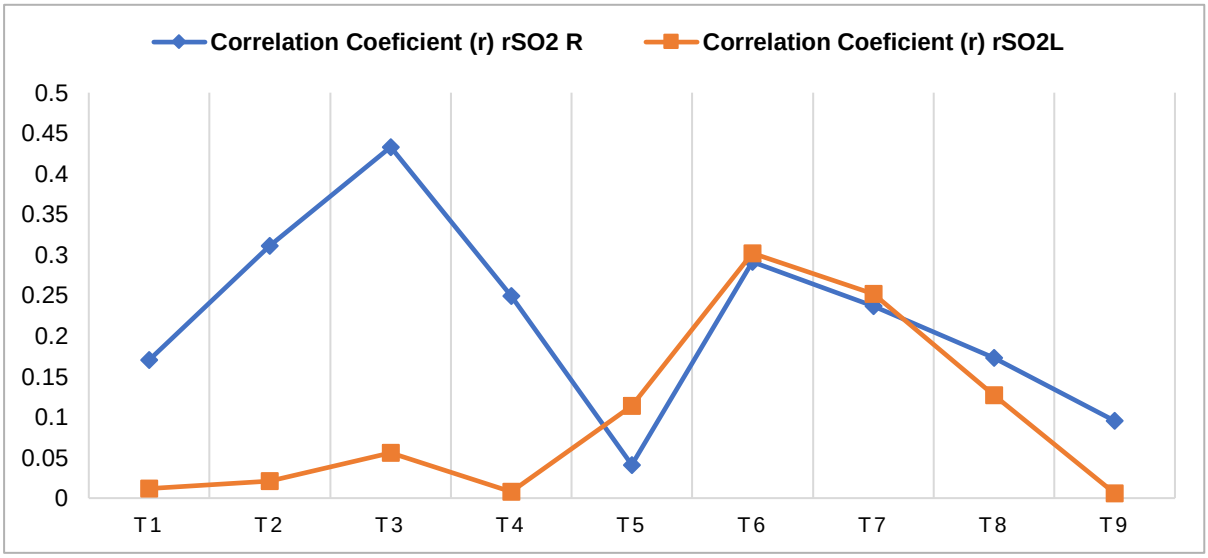

Figure 1. Trend of Pearson correlation coefficients between rSO2 and hematocrit levels (The x-axis represents the correlation coefficient values while the y-axis lists the time points from T1 through T9; **Blue Line** corresponds to the correlation trend between rSO2 on the right side (rSO2R) and hematocrit; **Orange Line** indicates the correlation trend for the left side (rSO2L) with hematocrit)

Mean Arterial Pressure (MAP) decreased significantly at T4 (70.04 ± 19.83 mm Hg) compared to the baseline value at T1 (84.06 ± 12.14 mm Hg). T5 had the lowest MAP (67.02 ± 15.46 mm Hg) during the surgical period. MAP increased from T7 and achieved 75.01 ± 16.19 mm Hg at T9 [Table 2]. Initially, at baseline (T1), both rSO2R and rSO2L showed modest positive correlations with MAP. At T2 and T3, the correlations for both rSO2R and rSO2L largely diminished, although they remained statistically significant. During the CPB process, including the cooling and hypothermia phases, the correlations varied, often showing modest strength. Notably, correlations strengthened slightly during hypothermia for rSO2R and reverted to positive for rSO2L upon rewarming. After CPB, both rSO2R and rSO2L demonstrated more pronounced positive correlations with MAP [Figure 2].

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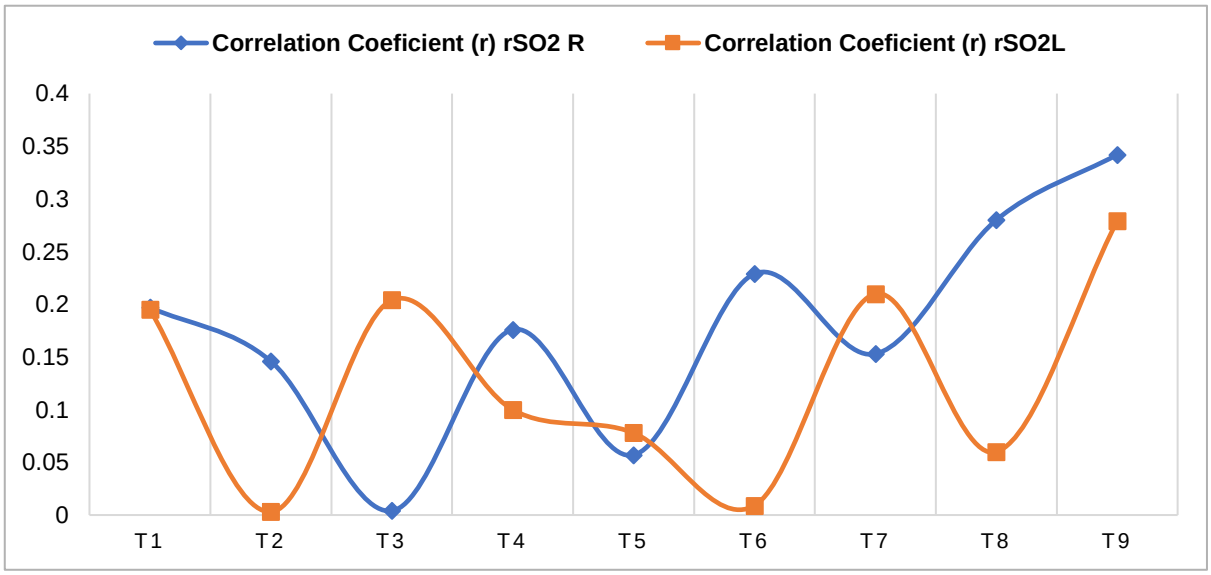

Figure 2. Trend of Pearson correlation coefficients rSO2 and MAP
(The x-axis represents the correlation coefficient values while the y-axis lists the time points from T1 through T9; **Blue Line** corresponds to the correlation trend between rSO2 on the right side (rSO2R) and MAP; **Orange Line** indicates the correlation trend for the left side (rSO2L) with MAP)

The baseline PaO2 at T1 was 69.63 ± 23.29 mm Hg, significantly increasing post-intubation (T2) to 581.80 ± 90.68 mm Hg and then decrease at T3 to 389.21 ± 106.68 mm Hg. T4 showed a significant increase, accompanied by variable values in subsequent time points. T9 exhibited a significant decrease of PaO2 compared to T8 [Table 2]. Initially, at T1, both rSO2R and rSO2L exhibited moderate positive correlations with PaO2, indicating a direct relationship between higher oxygen levels and increased cerebral oxygen saturation. Nevertheless, following the induction of anesthesia at T2 and T3, these correlations diminished for both rSO2R and rSO2L, becoming statistically non-significant, which indicates a reduced influence of PaO2 on rSO2 during these stages. During the different stages of CPB, including cooling, hypothermia, and rewarming, the correlations for rSO2R remained very weak and non-significant. Notably, rSO2L showed a moderate correlation during the cooling phase, though this did not extend to the hypothermia or rewarming phases. After weaning from CPB, both rSO2R and rSO2L showed statistically non-significant positive correlations with PaO2 [Figure 3].

Upon the initiation of CPB (T4), flows averaged 3 ± 0.42 l/min/m2. There was no significant difference in flows at T5 and during hypothermia at T6. At T7 (nasopharyngeal temperature of 36°C), CPB flows increased but were not significantly different from T5 [Table 2]. Our study identified significant correlations between hematocrit and rSO2 on the right (0.518) and left sides (0.338). Similarly, a 1 mm Hg change in MAP resulted in significant changes in rSO2R (0.399) and rSO2L (0.292). However, the correlation between rSO2 and PaO2 was weak and not statistically significant ($p > 0.05$), with rSO2R and rSO2L changing by a factor of 0.001 for every 1 mm Hg change in PaO2. This indicates the possibility of forecasting alterations in cerebral oximetry values based on variables such as hematocrit, MAP, and PaO2 [Table 3].

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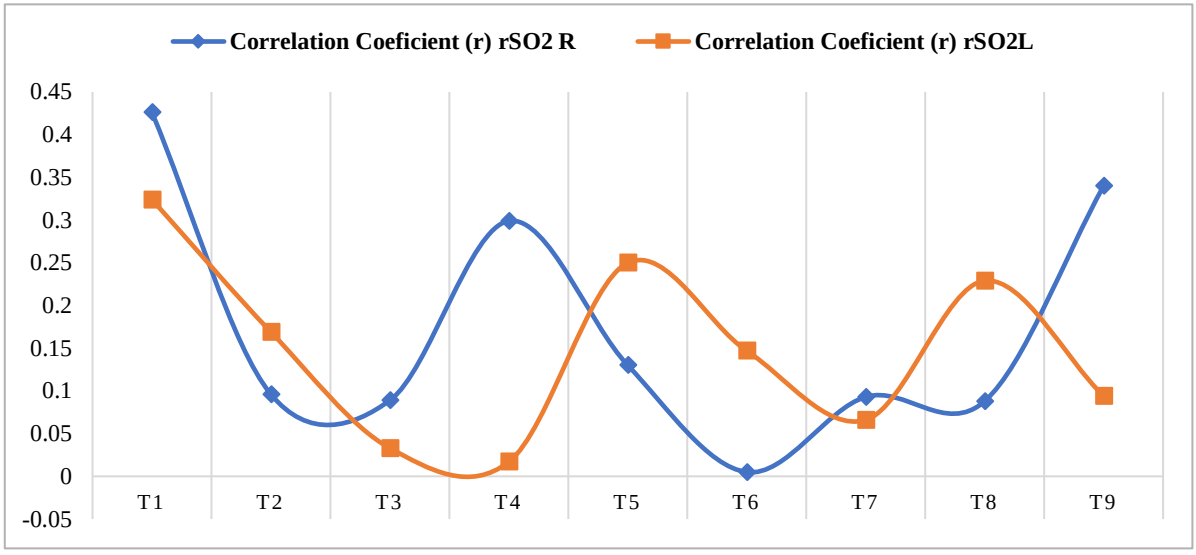

Figure 3. Trend of Pearson correlation coefficients rSO2 and PaO2
(The x-axis represents the correlation coefficient values while the y-axis lists the time points from T1 through T9; **Blue Line** corresponds to the correlation trend between rSO2 on the right side (rSO2R) and PaO2; **Orange Line** indicates the correlation trend for the left side (rSO2L) with PaO2.)

Our findings demonstrated a moderate positive correlation between rSO2 and Hematocrit on both the right (r = 0.518) and left sides (r = 0.338), with statistical significance (P < 0.001 for both sides) [Table 3]. This corresponds with several other studies highlighting the importance of hematocrit in preserving cerebral oxygenation after cardiac surgeries. Yamamoto et al., (8) established a significant correlation between hematocrit levels and cerebral oxygen saturation during both the pre-CPB and CPB phases in pediatric cardiac surgery. Similarly, in a study conducted by E.E. Ševerdija et al., (9), discovered that a decrease in hematocrit levels during CPB resulted in a significant reduction in mean cerebral tissue oxygenation. These consistent findings across multiple studies underscore the need for strategies to manage hematocrit levels during CPB, including blood transfusions, to prevent significant drops in cerebral oxygenation.

The positive correlations between rSO2 and MAP observed in our study (r = 0.399 for rSO2R and r = 0.292 for rSO2L, both P < 0.001) [Table 3] indicate that blood pressure significantly influences cerebral oxygen saturation. Numerous studies showing that adequate perfusion pressure is essential for maintaining cerebral oxygenation during surgery. Mansouri et al., (10) reported a significant relationship between MAP and cerebral oximetry in pediatric cardiac surgery, indicating that increasing MAP during CPB enhances brain perfusion and oxygenation. Pan et al., (11) analyzed 141 patients and discovered that rSO2 increased with an increase in MAP during CPB, with a correlation in children but not in neonates or infants.

We found very weak and non-significant correlations between rSO2 and PaO2 [r = 0.001 for both rSO2R (P = 0.896) and rSO2L (P = 0.792)], indicating that within the observed range, PaO2 does not exert a substantial direct influence on cerebral oxygen saturation. This finding aligns with research by Sarvesh Pal Singh et al., (12) , which identified a correlation coefficient of 0.005 between rSO2 and PaO2

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and observed minimal effects of PaO2 fluctuations on cerebral oxygenation compared to changes in MAP and hematocrit. This supports the idea that PaO2 plays a less direct role in influencing rSO2 during surgery.

Table 3. Comparison between rSO2 and various variables using a Generalized Estimating Equation (GEE) on a population-averaged model

Variable tested	Correlation coefficient	P value
rSO2R and hematocrit levels	0.518	**<0.001***
rSO2L and hematocrit levels	0.338	**<0.001***
rSO2R and MAP	0.399	**<0.001***
rSO2L and MAP	0.292	**<0.001***
rSO2R and PaO2	0.001	0.896
rSO2L and PaO2	0.001	0.792

*Based on the Wald Chi-Square test, Significant if the p-Value < 0.05
rSO2R: regional oxygen saturation on the right side of the frontal lobe; **rSO2L**: regional oxygen saturation on the left side of the frontal lobe; **MAP**: mean arterial pressure; **PaO2**: partial pressure of oxygen in arterial blood.

Baseline measurements (T1) in our study indicated consistent mean rSO2 levels of 72.14 ± 8.96% on the right side and 71.90 ± 8.05% on the left side. A study by Singh et al (12) involving 40 patients reported baseline rSO2 values of 64.35 and 64.97 for the right and left sides, respectively, whereas another study by Mohandas et al., (13) reported baseline rSO2 values of 65.78 and 66.32 for the right and left sides, respectively. The average difference between the right and left rSO2 values in our study was negligible. The non-zero difference observed could be attributed to the limited sample size. Post-intubation and exposure to 100% FiO2 (T2) significantly increased mean rSO2 (73.50 ± 9.17% on the right, 73.01 ± 9.17% on the left) despite a reduction in mean HCT (38.81 ± 7.53% from 40.80 ± 6.88). The average MAP showed a significant increase during this period. Contrary to Sarvesh Pal Singh et al., (12), who attributed an increase in PaO2 solely to this initial rise in rSO2 values, we find this explanation insufficient as increased MAP cannot be ignored according to our observation, this underscores the need for a comprehensive approach addressing multiple physiological parameters.

Upon the initiation of CPB (T4), there was significant decrease in mean rSO2 was 68.01 ± 8.56% on the right and 70.50 ± 8.96% on the left, associated with lower Hematocrit (29.49 ± 5.6%) and MAP (70.04 ± 19.83 mm Hg), despite adequate PaO2 (515.30 ± 85.37 mm Hg). During CPB (T5-T7) with hypothermia, mean rSO2 levels stabilized (64.59 ± 8.96% to 66.51 ± 9.82% on the right, 67.39 ± 9.38% to 67.63 ± 8.71% on the left), indicating adaptations to modified conditions despite fluctuating Hematocrit and MAP. Full CPB flows and reduced imbalance between cerebral metabolic oxygen consumption (CMRO2) and cerebral blood flow (CBF) during hypothermia might contribute to these adaptations.

After CPB (T8-T9), mean rSO2 approached baseline values (68.80 ± 9.42% to 69.60 ± 10.3% on the right, 71.92 ± 8.77% to 73.94 ± 9.21% on the left), which were achieved at significantly lower hematocrit and MAP compared to baseline (29.81 ± 5.75 vs 40.80 ± 6.88 and 75.01 ± 16.19 vs 84.06 ± 12.14, respectively). Higher PaO2 values post-CPB, especially with 100% FiO2, suggest an impact of PaO2 on rSO2. However, some studies have reported a decline in rSO2 during early rewarming stages. In an animal study conducted by Ostadal et al., (14), it was reported that brain oxygen saturation levels were significantly higher in the hypothermia

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group compared to the normothermia group. One possible explanation for this could be increased oxygen demand in normothermia. In their study, Moerman et al., (15) concluded that lower cerebral oxygen saturation is associated with lower flows during CPB. Enhanced CPB flows during rewarming to meet the brain's heightened oxygen requirements might account for the observed increase in rSO2 values post-full rewarming in our patients.

The findings of our study align with existing literature, emphasizing the complex interplay between hematocrit, MAP, and cerebral oxygen saturation during CABG with CPB. The moderate positive correlations between rSO2 and both hematocrit and MAP highlight their critical roles in ensuring adequate cerebral oxygen delivery and perfusion. The weak correlations between rSO2 and PaO2 indicate that while maintaining adequate PaO2 is necessary, it may not be the primary determinant of cerebral oxygen saturation.

The homogeneous patient population and exclusion criteria may limit the application to all CABG patients. Moreover, environmental factors and technology limitations may influence measurement accuracy. Although correlations were identified, causative relationships were not established, necessitating further research.

CONCLUSION

This study investigated the variations in rSO2 during CABG with CPB and analyzed its relationships with hematocrit, MAP, PaO2, CPB flows, and temperature. The findings highlight significant relationships between these variables, providing insights into factors influencing cerebral oxygenation during cardiac surgery.

Acknowledgment

The authors extend their gratitude to the surgery, nursing, and perfusion teams of the Cardiothoracic and Vascular Surgery department at SMS Medical College in Jaipur.

Conflict of Interest

The authors declare that there are no conflicts of interest among them.

Funding

This research did not receive any specific funding from public, commercial, or non-profit organizations.

Author's Contributions

JS, IV, SA, and ND contributed to the study's concept and design, data collection and processing, data analysis and interpretation, manuscript drafting, and revision for significant intellectual content, and provided administrative, technical, or material support. They also contributed to the literature review and gave final approval of the manuscript for submission.

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