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Research Article

Effects on cardiopulmonary bypass duration and optic nerve sheath diameter changes

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ABSTRACT

Background: Cardiopulmonary bypass (CPB) causes disruption of the blood-brain barrier and cerebral autoregulation for many reasons. The resulting cerebral edema causes an increase in intracranial pressure. Ultrasonographic optic nevre sheath diameter (ONSD) measurement is one of the non-invasive methods that provides information about intracranial pressure. Numerous studies have demonstrated a correlation between ONSD and intracranial pressure (ICP). We aimed to investigate ONSD changes during CPB and the relationship between these changes and CPB duration.

Methods: Twenty six patients aged between 18-75 years, with an ASA score of II or III, which underwent cardiac surgery with CPB are included to the study. ONSD measurements were made throughout surgery and data were recorded.

Results: ONSD values increased significantly at 45 and 90 min of CPB and end of the surgery compared to pre-CPB values for both eyes. There was no significant difference between 45 and 90 min. during CPB. A critical ONSD value was detected in 12 patients during CPB. No patient developed neurologic adverse events in the postoperative period.

Conclusion: ONSD increased during CPB regardless of duration. With the data obtained from our study, we cannot say that the increase in ONSD will be a predictor of postoperative neurological complications.

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1. Introduction

Extracorporeal circulation with Cardiopulmonary bypass (CPB) are utilized during most cardiac surgical interventions. Despite advances in anaesthesia and surgical techniques, CPB still has the potential to cause various complications in certain tissues and organ functions of the body. Such complications can lead to undesirable neurologic events [1]. Hypoxic ischemic events, embolism, changes in the blood-brain barrier, and increased intracranial pressure (ICP) are among the causes of postoperative adverse neurologic outcomes [2,3]. CPB may contribute to ischemia-reperfusion injury and the release of inflammatory substances, disruption of the integrity of the blood-brain barrier and increase in ICP [4]. Some studies have reported that prolonged CPB duration may

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damage the blood-brain barrier and extended duration of CPB alongside cross-clamp (CC) times increases brain damage [5].

The first sign of increased ICP is optic disc enlargement. However, ophthalmoscopic evaluation may not be feasible in all patients [6].

The sheath enveloping the optic nerve maintains continuity with the dura mater and the subarachnoidal space filled with cerebrospinal fluid (CSF), forming a direct connection between the two compartments. Due to the extensible nature of the optic nerve sheath, CSF pressure changes in the diameter of the optic nerve sheath with fluctuations in the anterior retrobulbar compartment approximately 3 mm behind the globe [7]. The ultrasonographic evaluation of ONSD was first proposed in 1987 as a tool to measure ICP [8] .

Numerous studies have demonstrated a correlation between ONSD and ICP [9,10]. Its diagnostic accuracy has been studied in various patient groups [11,12]. Studies in cardiac surgery suggest a positive correlation between ONSD and the duration of extracorporeal circulation [13–16]. Elevated ONSD values are associated with a higher risk of adverse neurological outcomes postoperatively, particularly in patients with prolonged CPB durations. It has been proposed that ONSD measurement could serve as a predictive tool for such outcomes [14,15]. Conversely, Rivas-Rangel et al. [13] reported cases where increased ONSD was not accompanied by symptoms of elevated ICP. ONSD has also been suggested as a potential indicator for assessing intravascular volume status [16]. In another study, Kara et al. [14] highlighted its utility as part of intraoperative monitoring during coronary artery bypass grafting (CABG). However, several studies emphasize the need for further research with larger patient cohorts and extended follow-up periods to clarify the effects of CPB duration on ONSD and identify factors influencing ONSD variations [13,17].

Based on these findings, our primary aim was to investigate the ONSD changes during CPB and the relationship between these changes and CPB duration. Our secondary aim was to evaluate adverse neurological events, delayed awakening (the failure of the standard patient to open her eyes and to respond spontaneously to calls from people without any physical contact or stimulus within 2 hours after termination of anesthesia despite despite our routine anesthesia protocol, normothermia, and stable hematologic and biochemical parameters), hemiparesis, hemiplegia, slurred speech, agitation, or poor response to commands, extubation time, and length of stay in the ICU within the first 24 hours.

2. Materials and Methods

After Kocaeli University ethics committee approval (GOKAEK-2023/08.22) and informed consent of the patients were obtained, 26 patients, aged between 18–75 years, with ASA scores of II-III, who were planned to undergo CPB-guided open heart surgery, were included in this prospective observational study. Patients with a known ophthalmologic disease, a history of ophthalmologic surgery, known neurologic disease, previous cerebrovascular accident, and intracranial pathology were excluded. Upon arrival at the operating room, patients were administered oxygen at a rate of 5 L/min via a face mask. Preoperative monitoring included electrocardiography (ECG), peripheral oxygen saturation (SpO_2), regional cerebral oximetry (rSO_2), and noninvasive blood pressure measurements. Midazolam at a volume of 0.02 mg/kg and fentanyl at 1 μg/kg intravenously (iv) was administered, followed by local anaesthesia with lidocaine, and radial artery cannulation was performed in the non-dominant hand. Anaesthesia induction was performed with 5–7 μg/kg fentanyl, 2 mg/kg thiopental (if necessary, an additional 1 mg/kg volume was administered until the eyelash reflex disappeared), and 0.8 mg/kg rocuronium. Patients were intubated after preoxygenation and controlled mechanical ventilation was started with a tidal volume of 8 ml/kg according to predicted body weight. Mechanical ventilation parameters were set as I: E ratio 1:2, plateau time 20% of the inspiratory time (Ti), PEEP 5 cm H_2O. The respiratory rate (RR) was initially started as 10/min. RF was adjusted so that the end-tidal carbon dioxide ($EtCO_2$) values were between 35–40 mmHg. Oxygen concentration was increased when SpO_2 dropped below 97% as in our routine clinical practice.

Following anaesthesia induction, a central venous pressure (CVP) catheter was inserted, preferably into the right internal jugular vein. A rectal and nasopharyngeal temperature measurement probes and a urinary catheter were placed. Anesthesia was maintained using a mixture of 40% oxygen and 60% air and desflurane inhalation at a 0.7–1.0 minimum alveolar concentration (MAC). Continuous remifentanil infusion at a rate of 0.1-0.4 μg/kg/min was administered before and after CPB, adjusting the dosage based on the patient's hemodynamic response. In the event of hypotension Mean arterial pressure (MAP) <65 mmHg for ≥1 minute, the following steps are taken in sequence until target blood pressure is achieved: placing the patient in the Trendelenburg position, infusing 250 mL of colloid administering a 5–10 mg intravenous bolus of ephedrine. In hypertension (more than 20% increase in systolic arterial pressure compared to baseline), iv. Bolus fentanyl, and for bradycardia heart rate (HR) <50 beats/min, 0.5 mg iv atropine administration was planned. Following systemic heparinisation and the completion of arterial and venous cannulations, CPB was initiated when the activated clotting time (ACT) level exceeded 480 seconds. Anesthesia was maintained by a desflurane vaporizer integrated into the CPB circuit. A flow rate of 2.2–2.4 L/min/m^2 and perfusion pressure of 60–80 mmHg were maintained during CPB.

Myocardial protection was achieved through the application of hyperkalemic blood cardioplegia. The lungs were not ventilated during the CPB and they were connected to the Bain circuit with a baseline oxygen flow of 200 ml/min. Mild to moderate systemic hypothermia was applied during CPB, and the patient was rewarmed after the last distal anastomosis was completed. Midazolam (0.03 mg/kg) and rocuronium bromide (0.15 mg/kg) were given at the beginning of CPB and during rewarming. Throughout the surgical procedure, an additional in-

travenous bolus of fentanyl (3 μg/kg) was administered during periods when sympathetic stimuli were most pronounced, and the avoidance of hypertension and tachycardia was imperative (skin incision, sternotomy, aortic cannulation, initiation and termination of CPB, rewarming, during and after CPB and skin closure). Following weaning from CPB, the cannulas were removed, and heparin was neutralized with protamine. Fluid, blood, and blood product, inotropic and vasopressor support were determined according to our routine clinical practice based on MAP, CVP, lactate values, arterial and venous oxygen pressure, rSO_2 and hematocrit values, and urine output. At the end of the surgery, the patients were transferred to the cardiovascular surgery intensive care unit. Standard postoperative analgesia protocol was applied to all patients. Patients were extubated when appropriate conditions were met.

2.1. ONSD measurement

Studies have suggested that novice ultrasound users can become proficient in scanning in as little as 25 examinations, while an experienced sonographer can become proficient in as few as ten scans [18]. Before starting our study, the researcher who will measure ONSD completed the learning process in 25 patients with a radiologist. ONSD measurement was performed by the anaesthesia trainee, who completed the training process in the presence of an expert radiologist (NY). The patient's eyes were closed and covered with a bio-occlusive dressing (Tegaderm®; 3M™ Healthcare). A thick layer of ultrasound gel was applied over Tegaderm®. A linear array ultrasound transducer was gently placed on the gel over the eye. The position of the probe was adjusted to obtain appropriate images. Bilateral ONSD was measured 3 mm posterior to the papilla. ONSD measurements were taken at four-time points: initiation of CPB (T_1), at 45 minutes into CPB (T_2), at 90 minutes into CPB (T_3), and end of surgery (T_4). MAP, arterial blood gas parameters (pH, paO_2, pCO_2, SaO_2, $ScvO_2$, lactate, haemoglobin, and hematocrit), rSO_2, body temperature and end-tidal desflurane ($End\text{-}Tidal_{Desf}$) concentrations were measured at ONSD measurement times. In addition, HR, RF, expired tidal volume (TV Eksp), peak inspiratory pressure (PIP), plateau pressure (PP), dynamic compliance (Cdyn), and CVP values were also recorded at times outside of CPB.

Vivek et al. [15] reported that if the maximum ONSD recorded during CPB exceeded 5.5 mm, the probability of adverse postoperative neurologic outcome was higher. Inspired by this study, an ONSD value greater than 5.5 mm in either the right or left eye was considered critical.

Adverse neurologic event was defined as delayed awakening (the failure of the standard patient to open her eyes and to respond spontaneously to calls from people without any physical contact or stimulus within 2 hours after termination of anesthesia despite despite our routine anesthesia protocol, normothermia, and stable hematologic and biochemical parameters), hemiparesis, hemiplegia, slurred speech, agitation, or poor response to commands. The patients were evaluated by an anesthesia assistant who was not involved in anesthesia management for 24 hours postoperatively.

2.2. Statistical analysis

Based on the ONSD measurements obtained from a 12-person pilot study at pre-CPB (T_1) and 90 minutes during CPB (T_3), a sample size of 23 was calculated using the G*Power 3.1.9.4 program for α=0.05 and Power (1-β)=0.95. Considering a predicted 10% data loss, 26 individuals were planned for the study. Statistical analysis was conducted using IBM SPSS 29.0 (IBM Corp., Armonk, NY, USA). Normal distribution was assessed using the Shapiro-Wilk test. Normally distributed variables were presented as mean ± standard deviation, non- normally distributed variables as median (25th–75th percentile), and categorical variables as frequency (percentage). Friedman's two-way ANOVA and Wilcoxon signed-rank tests were used for dependent group comparisons. Dunn's test was used for the multiple comparisons. A p value < 0.05 was considered statistically significant.

3. Results

In our study, a total of 26 patients were included initially. However, one patient had to be excluded from the study due to the requirement for extracorporeal membrane oxygenation support at the end of surgery, the data of 25 patients were analyzed (Table 1).

Compared to pre-CPB values, ONSD increased significantly in both eyes at 45th and 90th minutes of CPB (for the right eye; p=0.037, p<0.001 and for the left eye; p=0.007, p=0.001, respectively). ONSD values decreased at the end of surgery but were still significantly higher than pre-CPB values (for the right eye; p=0.007, for the left eye; p=0.009). ONSD values showed similar changes at 45th and 90th minutes of CPB (for the right eye; p=0.424, for the left eye; p=1.000). Changes in ONSD during CPB are shown in Figs. 1 and 2. With the introduction of CPB, significant changes were found not only in ONSD but also in MAP, body temparature, Hb, Htc, rSO_2, pH, paO_2, SaO_2, $ScvO_2$, lactate values and inhaled desflurane concentration compared to pre-CPB values (Table 2). We found that 12 patients had ONSD measurements above this critical value, critical ONSD values were determined only during CPB. In the postoperative period, no adverse neurological events developed in any patient.

4. Discussion

The data of this study showed that ONSD increased significantly from the beginning of CPB and independently of the duration of CPB. At the end of surgery, ONSD decreased but was still significantly higher than pre-bypass data. No postoperative adverse neurologic events occurred in any patient, including 12 patients who exceeded the critical ONSD value.

In studies conducted in cardiac surgery, it has been reported that ONSD, which is accepted as a non-invasive measurement method of intracranial pressure, increases as the CPB duration increases [13,17]. The

blood-brain barrier is affected by body temperature, blood flow pattern, viscosity, O_2 and CO_2 pressure and cardiopulmonary bypass [19,20]. When the data in Table 2 are analyzed, it is seen that significant changes occur in many parameters and ONSD starting from the 45th minute of CPB and these changes continue throughout CPB. The downward trend in T_4 and the change in ONSD suggest that it is transient.

Table 1. Perioperative data of the patients.

Age, year	62.24 ± 9.87
Weight, kg	79.20 ± 8.65
Gender, M/F, n (%)	7 (28) / 18 (72)
ASA, II/III, n (%)	13 (52) / 12 (48)
BMI (kg/m^2)	26,90 ± 3,69
Surgery, n (%) CABG/Valve repair or replacement/Combined	16 (64) / 7 (28) / 2 (8)
Comorbidity, n (%) Hypertension/Respiratory/Diabetes,	7 (28) / 11 (44) / 7 (28)
Carotid pathology, present/absent, n (%)	2 (8) / 23 (92)
EF, %	60.00 (45.00-60.00)
Fentanyl, mcq	600.00 (575.00-700.00)
Muscle relaxant, mg	90.00 (75.00-100.00)
CC duratiom, min	78.00 (60.00-95.00)
CPB duration, min	132.00 (111.00-146.00)
Cardioplegia, ml	3000.00 (2000.00-4000.00)
Duration of anesthesia, hours	6.00 (5.00-6.75)
End-surgical balance, ml	1350.00 (848.00-1925.00)
Inotrope requirement, n (%) None/single inotrope/two inotropes	8 (32) / 5 (48) / 12 (20)
Blood and blood product requirement, yes/no, n (%)	14 (56) / 11 (44)
Ephedrine requirement, yes/no, n (%)	5 (20) / 20 (80)
Atropine,requirement yes/no, n (%)	0/25
Extubation time, hours	5.00 (4.00-8.00)
Duration of intensive care, hours	48.00 (48.00-60.00)
ONSD >5.5 mm, n (%)	12 (48)
$ONSD_{right,\ meam}$, mm	4.70
$ONSD_{left,\ meam}$, mm	5.10

Values are given as n(%), median (25–75) percentile or mean±SS

ASA: American Society of Anesthesiologists; EF: Ejection Fraction; ONSD: Optic Nerve Sheath Diameter; CABG: Coronary artery bypass graft.

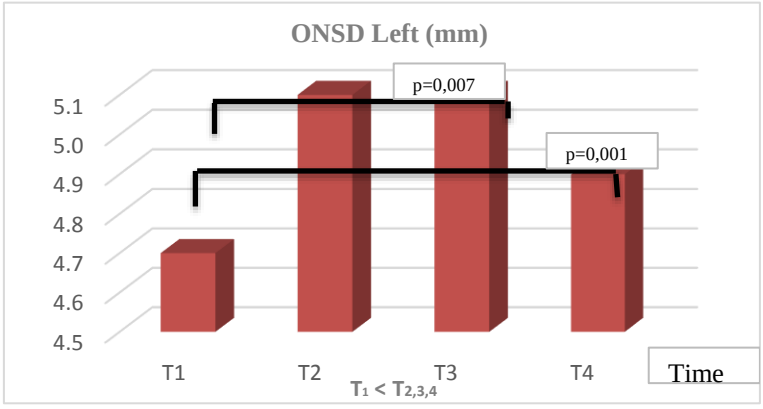

Fig. 1. Changes in ONSD during CPB-Left.

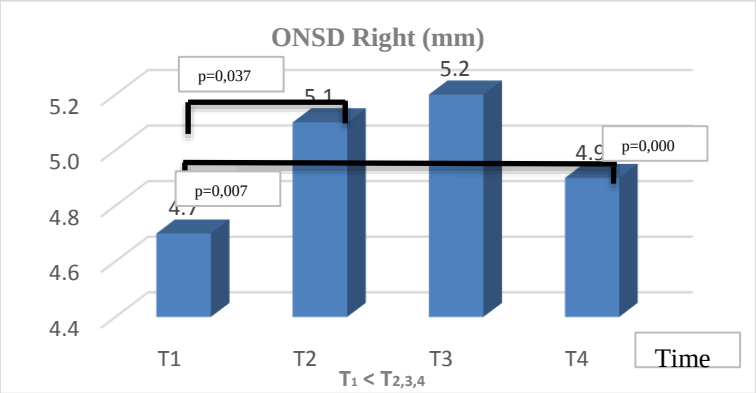

Fig. 2. Changes in ONSD during CPB-Right.

Table 2. Intraoperative changes seen during CPB compared to before and after CPB.

	T_1	T_2	T_3	T_4	p	Pairwise comparison
$ONSD_{right}$, mm	4.7 (4.3-5.0)	5.1(4.75-5.35)	5.2(4.85-5.5)	4.9(4.6-5.2)	<0.001[a]	$T_1 < T_{2,3,4}$
$ONSD_{left}$, mm	4.7(4.4-5.05)	5.1(4.8-5.3)	5.1(4.8-5.4)	5.0(4.7-5.2)	<0.001[a]	$T_1 < T_{2,3,4}$
MAP, mmHg	63.0(60.0-68.0)	57.0(54.0-73.0)	65.0(60.0-70.0)	73.0(68.0-78.5)	<0.001[a]	$T_1 > T_2$ $T_4 > T_{1,3}$ $T_3 > T_2$
Body temperature, °C	36.2(35.9-36.65)	33.0(32.0-33.0)	32.0(32.0-33.0)	36.2(35.9-36.7)	<0.001[a]	$T_1 > T_{2,3}$ $T_4 > T_{2,3}$
Hb, g/dL	12.7(11.1-13.2)	8.9(7.55-9.8)	9.0(8.2-10.1)	9.7(9.2-10.4)	<0.001[a]	$T_1 > T_{2,3,4}$ $T_4 > T_2$
Htc, %	37.5(32.75-42.0)	27.0(23.6-29.9)	27.7(25.65-31.15)	30.0(28.1-32.75)	<0.001[a]	$T_1 > T_{2,3,4}$ $T_4 > T_2$
$rSO_{2\text{-right}}$, %	85.0(78.5-94.0)	78.0(70.0-87.5)	80.0(71.0-86.5)	81.0(72.5-85.5)	0.002[a]	$T_1 > T_{2,3,4}$
$rSO_{2\text{-left}}$, %	84.0(80.5-88.5)	81.0(70.5-85.5)	78.0(71.5-86.0)	81.0(73.0-84.5)	<0.001[a]	$T_1 > T_{2,3,4}$
ph	7.41(7.39-7.44)	7.36(7.34-7.38)	7.35(7.32-7.38)	7.36(7.31-7.39)	<0.001[a]	$T_1 > T_{2,3,4}$
Lactate, mol/L	1.0(0.8-1.3)	1.5(1.15-1.8)	1.9(1.35-2.45)	2.2(1.5-3.95)	<0.001[a]	$T_1 < T_{3,4}$ $T_2 < T_{3,4}$
PaO_2, mmHg	141.0(96.7-170.5)	302.0(246.0-328.0)	296.0(249.50-330.0)	103.0(78.75-158.5)	<0.001[a]	$T_1 < T_{2,3}$ $T_4 < T_{2,3}$
$PaCO_2$, mmHg	36.0(34.70-37.75)	37.9(35.0-41.45)	39.0(37.2-41.25)	38.0(35.65-39.75)	0.003[a]	$T_1 < T_3$
SaO_2, %	98.0(97.0-99.0)	99.0(99.0-99.35)	99.0(99.0-99.5)	98.0(96.75-99.0)	<0.001[a]	$T_1 < T_{2,3}$ $T_4 < T_{2,3}$
$ScvO_2$, %	76.0(71.50-79.0)	84.2(80.3-86.5)	80.0(74.5-86.5)	77.0(70.6-82.55)	<0.001[a]	$T_1 < T_{2,3}$ $T_4 < T_2$
$EndTidal_{Desf}$, %	5.00(4.00-5.00)	4.00(3.50-4.00)	4.00(3.00-4.00)	5.00(4.00-5.00)	<0.001[a]	$T_1 > T_{2,3}$ $T_4 > T_{2,3}$
HR, beat/min	73.0(64.5-81.5)	-	-	89(82.5-98.5)	0.002[b]	
RF, breath/min	12.0(11.0-12.0)	-	-	12.0(12.0-14.0)	0.008[b]	
TVexpirium, ml	560.0(505.0-606.5)	-		550.0(495.5-615.0)	0.820[b]	
PIP, cmH_20	17.0(15.0-20.5)	-	-	19.0(16.0-21.5)	0.272[b]	
PP, cmH_20	15.0(13.0-18.0)	-	-	16.0(13.5-18.0)	0.822[b]	
Cdyn, l/cmH_20	54.0(45.0-71.9)	-	-	52.5(40.0-65.5)	0.484[b]	
CVP, mmHg	12.0(11.0-13.5)	-	-	12.0(10.5-15.5)	0.084[b]	

Values are given as median (25–75) percentile.

a: Friedmen's two way ANOVA with Dunn's test; b: Wilcoxon Signed rank test.

ONSD: Optic nerve sheath diameter; MAP: Mean arterial pressure; rSO_2: Regional serebral oxygen saturation; HR: Heart rate; RF: Respiratory frequency; TV: Tidal volume; PIP: Peak inspiratuar pressure; PP: Plateau pressure; Cdyn: Dynamic compliance; CVP: Central venous pressure.

T_1: 10 min before initiation of CPB; T_2: 45th min of CPB; T_3: 90th min of CPB; T_4: End of surgery.

The parameters in Table 2 including MAP, body temperature, Hb and Htc, right and left rSO_2, paO_2, SaO_2, $ScvO_2$ and end-tidal desflurane levels show significant differences at the 45th and 90th minute of CPB. Each of these changes can cause fluctuations in cerebral perfusion and may lead to an increase in ONSD due to anatomical relationship [21,22]. Our findings do not support the idea that there is a positive correlation between ONSD and CPB duration reported in previous studies [13,16]. Several of the studies have reported that further research with larger patient populations and longer patient follow-up is needed to identify the effect of CPB and CPB duration on ONSD as well as to identify factors related to variations in ONSD [13,17].

The fact that ONSD did not continue to increase at T_3 and showed a decreasing trend at T_4 suggests that ONSD, and thus ICP, is only associated with acute exacerbation of CPB. We may be mistaken if we think that increases in ONSD are only a reflection of changes in cerebral perfusion caused by CPB. Because in an animal study, hemodilution changes in the optic nerve and intraoperative ischemic damage were found to lead to optic neuropathy and optic nerve damage [23].

Hypotension, arrhythmia, hypercoagulopathy and hypothermia during surgery and CPB, decreased blood flow with hypothermia, prolonged CPB times and use of vasopressor agents increase ischemia in the optic area [24,25]. It has been reported that changes in ONSD may also be detected in neuropathies involving the optic nerve [26–28] .Further studies are needed to differentiate whether the changes in ONSD during CPB are really a reflection of impaired cerebral perfusion or whether it is really a damage to the optic nerve.

Previous systematic reviews and meta-analyses have demonstrated that an ONSD value exceeding 5.00 to 5.70 mm is associated with an ICP value above 20 mmHg [29,30]. Vivek et al. [15] investigated the relationship between postoperative neurological complications, ONSD, and CPB durations and stated that if the maximum ONSD recorded during CPB exceeded 5.5 mm, there was a higher likelihood of postoperative adverse neurological outcomes. However, 33 of 44 patients with no adverse neurological outcome had ONSD above 5.5 mm. The authors stated that a maximum ONSD anytime during CPB has a high sensitivity, specificity, diagnostic accuracy, as well as predictive value of negative test [15]. In the study of Taşkın et al. [17] no cut-off value of ONSD and no adverse neurological outcomes were mentioned. Kara et al. [14] reported that no critical ONSD value was obtained in any patient, and no perioperative neurological complications were observed. The authors stated that during extracorporeal circulation, ultrasound-guided ONSD measurement is an easy, inexpensive, and low-complication method that can be used as a part of monitoring during cardiac surgery and may be a predictor of increased ICP [14,17].

In a study conducted in pediatric patients undergoing congenital cardiac surgery, a positive correlation was found between ONSD values measured at 24 hours postoperatively and the duration of CPB. However, ONSD increase was also observed in patients without signs of increased ICP in this study. The authors stated that these results are insufficient to draw definitive conclusions, and more studies are needed to determine the factors that cause ONSD change [13]. In accordance with Rivas-Rangel et al. [13] , the factors responsible for ONSD change caused by CPB are not elucidated sufficiently, and more comprehensive studies are required.

In our study there was no adverse neurologic event in any patient at 24 hours postoperative follow-up. Based on these data from our study, we do not think that there is a positive association between the critical ONSD value and adverse neurologic events and that the critical ONSD value may be a predictor of adverse neurologic events. However, in our study, ONSD values at 45 and 90 minutes of CPB were found to be similar and therefore our primary hypothesis could not be confirmed. When we compare the results of studies conducted on this subject, both similarities and differences can be found due to methodological differences. For example, in the study by Taşkın et al. [17] , the ONSD was measured just before the start of the CPB, and at the 30th minute, 60th minute and 90th minute of the CPB. During the CPB, ONSD and nICPONSD values increased over time, and there was a statistical difference between the 0th, 30th, 60th and 90th minutes. All comparisons were made with the initial values (T_0). In our study, when we compared with the pre-bypass values, a significant increase was found during the CPB period, but when the changes were examined according to the CPB period, no significant difference was found between the 45th and 90th minutes of the CPB.

In the study by Kara et al. [14]. ONSD measurements were performed immediately before surgery, after intubation, 15 minutes after cross-clamping, after removal of the cross-clamping, and at the end of the operation and mean ONSD values at all stages during surgery were statistically significantly higher than the mean basal measurement.

Our study was performed with a small number of patients and that preoperative and postoperative ONSD measurements were not performed may be considered among the limitations of our study. The fact that the follow-up of postoperative adverse neurologic events was not limited to 24 hours and the patient was not followed up for neurologic complications in the late postoperative period is another limitation.

Patients cannot be limited to a 24 hour observation period for postoperative undesirable neurological complications; therefore, follow-up may be required in the later postoperative period as well. In our pilot study, we did not include pre-anesthesia ONSD measurement in the methodology, as performing this measurement in two awake patients caused discomfort. This may represent another limitation of our study.

5. Conclusions

CPB during open-heart surgery elevates ONSD independently of duration. However, our data do not support ONSD as a reliable predictor of postoperative neurologic complications. Further research is warranted to elucidate the multifactorial mechanisms underlying ONSD variability and its clinical implications.

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Conflict of Interest

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this manuscript.

Data Availability

The datasets created and/or analyzed during the current study are not publicly available, but are available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

This study was approved by the ethics committee of Kocaeli University (approval number: GOKAEK-2023/08.22; date: 04.05.2023). Written informed consent was obtained from the participants and/or legal guardian(s) of the patients. All methods were performed in accordance with relevant guidelines and regulations. No violation of Helsinki Declaration was taken place during informed consent and data acquisition period.

Author Contributions

Tulay Cardakozu: *investigation, methodology, data curation, writing – original draft.*
Nur Nazire Yucal: *conceptualization, supervision, formal analysis, writing – review & editing, project administration.*
Ali Ahmet Arikan: *resources, validation, clinical support.*
Sevim Cesur Okan: *methodology, visualization, writing – review & editing.*
Oguz Omay: *surgical investigation, patient recruitment, resources.*

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