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ORIGINAL ARTICLE

Changes in intraocular pressure during cardiac surgery with and without cardiopulmonary bypass

Hironobu Hayashi · Masahiko Kawaguchi · Kyoko Hasuwa · Satoki Inoue · Masahiro Okamoto · Toyoaki Matsuura · Shigeki Taniguchi · Hitoshi Furuya

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Abstract
Purpose Data on intraocular pressure (IOP) during cardiac surgery with cardiopulmonary bypass (CPB) and anesthetic management are limited. This study was conducted to investigate changes in IOP during cardiac surgery with and without CPB.
Methods IOP was intraoperatively measured in patients undergoing elective cardiac surgery with ($n = 35$) or without ($n = 11$) CPB. Measurements were performed using a Tonopen® XL hand-held tonometer at the following five time points in patients with CPB: (1) 30 min after anesthesia induction (baseline), (2) prior to CPB, (3) 60 min after the beginning of CPB, (4) before cessation of CPB, and (5) at the end of operation; and in patients without CPB: (1) 30 min after anesthesia induction (baseline), (2) prior to anastomosis, (3) during left anterior descending artery anastomosis, (4) during left circumflex or right coronary artery anastomosis (head-down position), and (5) at the end of operation.
Results In patients with CPB, IOP values at points 3 and 4 were significantly decreased compared with baseline values and returned to baseline levels at point 5. In patients without CPB, values were significantly increased and peaked at point 4 in the head-down position compared with baseline and prior to anastomosis.
Conclusion Results indicate that during cardiac surgery, IOP values decreased during CPB and increased during anastomosis in the head-down position in patients without CPB.

Keywords Intraocular pressure · Cardiac surgery · Cardiopulmonary bypass

H. Hayashi · M. Kawaguchi (✉) · K. Hasuwa · S. Inoue · H. Furuya
Department of Anesthesiology, Nara Medical University, 840 Shijo-cho, Kashihara, Nara 634-8522, Japan
e-mail: drjkawa@naramed-u.ac.jp

M. Okamoto · T. Matsuura
Department of Ophthalmology, Nara Medical University, 840 Shijo-cho, Kashihara, Nara, Japan

S. Taniguchi
Department of Thoracic and Cardiovascular Surgery, Nara Medical University, 840 Shijo-cho, Kashihara, Nara, Japan

Introduction

Postoperative ophthalmological complications after cardiac surgery with cardiopulmonary bypass (CPB) are rare and poorly understood but are devastating, in part because they often produce severe visual loss and in part because once vision is lost, it rarely recovers. The incidences of postoperative ophthalmological complications after cardiac surgery with CPB have been reported to be 0.06–25.6% [1–6]. This complication is caused by multiple physiologic changes during CPB, including systemic hypotension, cerebral hypoperfusion, arterial embolism, and hypothermia. The most common site of ischemic injury to the visual pathway is the optic nerve. Anterior ischemic optic neuropathy appears to be more common than posterior ischemic optic neuropathy in patients undergoing cardiac surgery with CPB [1–3, 6]. CPB induction may also cause changes in intraoperative IOP, which potentially may be related to postoperative ophthalmological complication, and it can affect IOP determinants, which include aqueous humor fluid dynamics, choroidal blood and vitreous volume, and extraocular muscle tone. In fact, there have been several reports on IOP

changes during CPB [7–11], which indicated increased IOP in some reports [8, 10, 11] and unchanged IOP in others [7, 9, 11]. Increased IOP was considered a risk factor in decreased perfusion pressure to the optic nerve and can result in ischemic optic neuropathy [12]. However, these investigations were conducted in the 1970–1990s, when patient management and settings for CPB may have been different from today. Therefore, it is unknown whether IOP during cardiac surgery changes or not under recent conditions and anesthetic management employed in CPB. This study was conducted to investigate changes in IOP during cardiac surgery with and without CPB.

Materials and methods

After institutional approval at Nara Medical University, Nara, Japan, written informed consent was obtained from all patients. Forty-six patients undergoing elective cardiac surgery with ($n = 35$) or without ($n = 11$) CPB from March 2007 to September 2008 were enrolled in the study. Patients with preoperative glaucoma or who underwent intraoperative deep hypothermic circulatory arrest (<28°C) and selective cerebral perfusion were precluded. Patient ages ranged from 40 to 84 (mean 65) years. There were 23 men and 23 women.

No premedication was given before anesthesia. Prior to anesthesia induction, all patients were monitored with electrocardiogram, finger pulse oximeter probe, and automatic blood pressure cuff. Regional cerebral tissue oxygen saturation index was monitored with near-infrared spectroscopy equipment (INVOS 5100; Somanetics Corp, Troy, MI, USA). Anesthesia induction consisted midazolam and/or propofol, fentanyl, sevoflurane, and neuromuscular blockade to facilitate endotracheal intubation. Anesthesia was maintained with propofol, fentanyl, and sevoflurane with neuromuscular blockade. After the trachea was intubated, semiclosed circuit mechanical ventilation was adjusted to maintain the level of partial pressure of end-tidal carbon dioxide tension between 30 and 40 mmHg using tidal volume set at 8–12 ml/kg and a rate of 8–12 breaths/min. A mixture of air and oxygen was administered. Arterial pressure was monitored via a catheter in the radial artery. A pulmonary artery catheter was inserted through the internal jugular vein to measure mixed venous oxygen saturation, continuous cardiac output, pulmonary artery pressure, and central venous pressure (CVP) in all patients. A transesophageal echocardiography probe was also inserted.

To manage CPB, anticoagulation was achieved with heparin to maintain an activated clotting time >400 s. Standard cannulation for CPB was performed with ascending aortic cannulation and dual-stage cannulation of the right atrium. The CPB circuit was primed with 1.0 L Ringer's lactate and 200 ml 20% mannitol. Nonpulsatile flow (2.4–3.0 L/min/m^2), moderate hypothermia (28–34°C) and a heparin-coated membrane oxygenator (MERA Excelung Prime; MERA, Tokyo, Japan) were used in all patients with CPB. Alpha stat acid–base management of CPB was used. Myocardial protection was achieved by administering intermittent antegrade and occasionally retrograde blood cardioplegia in valve and/or aorta surgery. All patients with CPB were rewarmed to a bladder temperature >35°C before separation from CPB. After separation, heparin was neutralized with 1 mg protamine per 100 U of heparin to achieve an activated clotting time within 10% of baseline.

All patients without CPB underwent off-pump coronary artery bypass graft surgery (OPCAB) performed with the Medtronic Octopus III stabilizing device (Medtronic, Minneapolis, MN, USA) for coronary stabilization and deep pericardial traction sutures for cardiac displacement and presentation. Patients were placed in 15–30° head-down position by tilting the operating table to maintain hemodynamic stability (mean arterial pressure >60 mmHg) during displacement of the beating heart for anastomosis of left circumflex or right coronary artery.

IOP measurements

IOP was measured with a Tonopen® XL hand-held tonometer (Medtronic SOLAN, Jacksonville, FL, USA). This device measures IOP using the Imbert-Fick principle ($P = F/A$, where $P = IOP$, $F =$ the amount of force exerted by the tonometer to flatten a specific area of the eye, and $A =$ the area flattened). The Tonopen is clinically accurate when evaluated against the Goldmann tonometer. Calibration of the Tonopen was carried out in the operating room before each case. IOP measurements were performed on both eyes at the following five time points in patients with CPB: (1) 30 min after anesthesia induction (baseline), (2) prior to CPB, (3) 60 min after the beginning of CPB, (4) before cessation of CPB, and (5) at the end of operation; and in patients without CPB: (1) 30 min after anesthesia induction (baseline), (2) prior to anastomosis, (3) during left anterior descending artery anastomosis, (4) during left circumflex or right coronary anastomosis (head-down position), and (5) at the end of operation. Mean arterial and CVP, body temperature, pH, partial pressure of carbon dioxide in the artery ($PaCO_2$), partial pressure of oxygen in the artery (PaO_2), and hemoglobin were also recorded at the same time points as IOP.

Statistical analysis

Sample size was calculated using a power analysis based on the assumptions that: (1) baseline IOP would be

approximately 10–12 mmHg [standard deviation (SD) = 4] based on preliminary study results, (2) in patients with CPB, a reduction in IOP by 50% of baseline was considered of clinical importance, (3) in patients without CPB, an increase in IOP to 21 mmHg (abnormal level) was considered of clinical importance, (4) $\alpha = 0.01$ with a power (1-β) of 0.8. This analysis indicated that 35 patients were needed for the group of patients with CPB and 11 for the group without CPB. Data are presented as mean ± SD. Between-group demographic analyses were performed using Student's unpaired *t* test and chi-square test. Intraoperative values of IOP and parameters were compared using analysis of variance (ANOVA) with repeated measures with Student–Newman–Keuls test for post hoc among measurement points. Values of $P < 0.05$ were considered significant.

Table 1 Preoperative variables of patients and procedures

	With CPB ($n = 35$)	Without CPB ($n = 11$)
Age (years)	65 ± 11	67 ± 8
Sex (male)	16 (46%)	7 (64%)
Body mass index	22.7 ± 3.6	23.2 ± 3.1
Diabetes mellitus	9 (26%)	10 (91%)
Hypertension	21 (60%)	10 (91%)
Anesthesia time (min)	588 ± 163	518 ± 110
Operation time (min)	484 ± 150	399 ± 105
Type of procedure		
Valve	19 (54%)	0 (0%)
CABG	8 (23%)	11 (100%)
Valve + Aorta	3 (9%)	0 (0%)
Valve + CABG	2 (6%)	0 (0%)
Other	2 (6%)	0 (0%)

Data are presented as mean ± standard deviation or as percentage of patients

CPB cardiopulmonary bypass, *CABG* coronary artery bypass grafting

Results

Patients' preoperative variables and surgical procedures are summarized in Table 1. Intraoperative changes in parameters in patients with CPB are shown in Table 2. In patients with CPB, mean arterial pressure and CVPs, body temperature, and hemoglobin were significantly lower 60 min after the beginning of CPB compared with those at baseline and prior CPB. Mean arterial pressure and hemoglobin remained lower until cessation of CPB. PaO_2 was significantly higher 60 min after the beginning of CPB and before cessation of CPB compared with baseline and prior CPB. Intraoperative changes in IOP values in patients with CPB are shown in Fig. 1. No statistical differences were found between IOP values from the right and left eyes at measurement points. The values of left and right baseline IOP were 9.2 ± 2.9 and 9.5 ± 3.7 mmHg, respectively. Bilateral IOP values 60 min after the beginning of CPB (left 5.5 ± 4.3 mmHg, right 6.1 ± 4.3 mmHg) were significantly lower compared with baseline values, and these decreases continued until cessation of CPB and returned to baseline levels immediately after cessation of CPB.

Intraoperative changes in parameters in patients without CPB are shown in Table 3. In patients without CPB, CVP was significantly higher at time point 4 in head-down position compared with baseline and prior to anastomosis, and this increase returned to baseline at the end of operation in the supine position. The other parameters remained unchanged through the operation. Intraoperative IOP changes in patients without CPB are shown in Fig. 2. No statistical differences were found between IOP values from the right and left eyes at all measurement time points. Left and right baseline IOP values were 11.6 ± 3.5 and 11.5 ± 4.7 mmHg, respectively. Bilateral IOP values prior to anastomosis were higher compared with baseline. Bilateral IOP values significantly increased and peaked at time point 4 in the head-down position (left 19.9 ± 4.9 mmHg,

Table 2 Changes in parameters in patients with CPB

Time points	MAP (mmHg)	CVP (mmHg)	BT (°C)	pH	$PaCO_2$ (mmHg)	PaO_2 (mmHg)	Hb (g/dL)
(1)	75 ± 13	8 ± 4	36.0 ± 0.7	7.40 ± 0.05	41 ± 5	220 ± 81	11.5 ± 1.7
(2)	73 ± 13	7 ± 5	35.3 ± 0.5	7.38 ± 0.05	42 ± 4	238 ± 83	10.6 ± 1.5*
(3)	65 ± 11*,#	2 ± 5*,#	32.3 ± 3.0*,#	7.39 ± 0.04	43 ± 4	383 ± 63*,#	7.8 ± 1.0*,#
(4)	67 ± 12*	6 ± 6	35.5 ± 1.7	7.40 ± 0.04	42 ± 4	387 ± 50*,#	8.7 ± 1.1*,#
(5)	70 ± 8	10 ± 3*,#	36.8 ± 0.4*,#	7.40 ± 0.04	42 ± 4	190 ± 71	9.9 ± 1.2*,#

Time points (1) 30 min after anesthesia induction (baseline), (2) prior to CPB, (3) 60 min after the beginning of CPB, (4) before cessation of CPB, and (5) at the end of operation

CPB cardiopulmonary bypass, *MAP* mean arterial pressure, *CVP* central venous pressure, *BT* body temperature, *$PaCO_2$* arterial carbon dioxide partial pressure, *PaO_2* arterial oxygen partial pressure, *Hb* hemoglobin

* $P < 0.05$ vs time point 1 (baseline)

\# $P < 0.05$ vs time point 2

right 19.3 ± 4.9 mmHg) compared with baseline and prior to anastomosis.

Discussion

Our study results show that bilateral IOP values were significantly decreased during operation in CPB patients, whereas they were significantly increased during anastomosis in head-down position in patients without CPB.

IOP is influenced by a great many factors and is mainly determined by the volume of aqueous humor, choroidal blood, and vitreous within the eye exerting an outward pressure; and scleral compliance and extraocular muscle tone exerting inward pressure. The balance between aqueous humor production and drainage is the primary physiological control mechanism regulating IOP. Production of aqueous humor generates a pressure within the eye to maintain its structural integrity and position of the refractive surfaces of the eye. Most aqueous humor is formed in the posterior chamber by the ciliary body supplied by ciliary arteries and circulates through the pupil, enters the anterior chamber, and exits via the trabecular meshwork. From there, the aqueous humor enters the canal of Schlemm and then drains into the orbital venous system through the aqueous veins in the episcleral tissue. Finally, the aqueous drains into central venous system via the internal and external jugular venous systems.

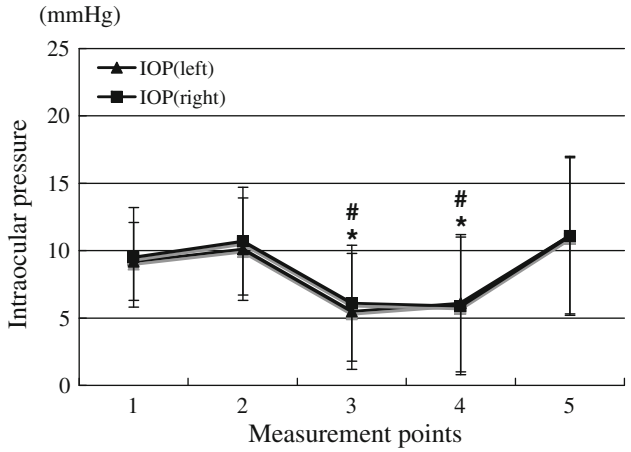

Fig. 1 Intraoperative changes in intraocular pressure (IOP) during cardiac surgery with cardiopulmonary bypass (CPB). Measurement time points (1) 30 min after anesthesia induction (baseline); (2) prior to CPB; (3) 60 min after the beginning of CPB; (4) before cessation of CPB; and (5) at the end of operation. Bilateral IOPs were significantly decreased at time points 3 and 4 and returned at time point 5. $^{*}P < 0.05$ vs point 1. $^{\#}P < 0.05$ vs point 2

The choroid is a highly vascular area with the highest blood flow of any structure in the human body, including the brain and kidneys. Blood to vessels of the choroid is supplied by posterior ciliary arteries originating from the first branch of the internal carotid artery, the ophthalmic artery. The choroidal arteries supply the choriocapillaris from the optic nerve to the ora serrata. Venous drainage of the choroid is through four to seven vortex veins distributed throughout the globe. Vortex veins drain into the superior and inferior orbital veins that are routes to the cavernous sinus. The vitreous mainly consists of water with a fine fibrillar supporting structure. It has been considered that the volume of vitreous and pressure in the vitreous can be reduced by dehydrating the vitreous.

There have been several reports regarding IOP changes during cardiovascular surgery with CPB, although the results differ depending on the reports (Table 4) [7–11]. Levy et al. [7] examined intraoperative IOP in patients with CPB and showed that IOP remained unchanged during CPB and systemic hypotension was associated with a fall in IOP. Stellpflug et al. [8] showed that IOP increased during CPB with a decrease in arterial pressure. Lilleaasen et al. [9] reported that although IOP increased at the onset of CPB, during the other phases, it did not change

Table 3 Changes in parameters in patients without CPB

Times	MAP (mmHg)	CVP (mmHg)	BT (°C)	pH	$PaCO_2$ (mmHg)	PaO_2 (mmHg)	Hb (g/dL)
(1)	65 ± 9	6 ± 3	35.7 ± 0.7	7.35 ± 0.03	43 ± 2	205 ± 72	10.8 ± 1.8
(2)	66 ± 11	5 ± 3	35.5 ± 0.9	7.35 ± 0.03	43 ± 2	200 ± 48	9.9 ± 2.0
(3)	68 ± 9	7 ± 5	35.2 ± 1.3	7.35 ± 0.04	43 ± 1	196 ± 46	10.1 ± 2.2
(4)	70 ± 8	18 ± 7$^{*,\#}$	36.0 ± 0.9	7.33 ± 0.06	43 ± 3	210 ± 51	10.0 ± 1.1
(5)	69 ± 11	9 ± 4	36.1 ± 1.0	7.35 ± 0.04	41 ± 2	167 ± 67	10.1 ± 1.3

Time points: (1) 30 min after anesthesia induction (baseline), (2) prior to anastomosis, (3) during left anterior descending artery anastomosis, (4) during anastomosis of left circumflex or right coronary artery (head-down position), and (5) at the end of operation

CPB cardiopulmonary bypass, *MAP* mean arterial pressure, *CVP* central venous pressure, *BT* body temperature, $PaCO_2$ arterial carbon dioxide partial pressure, PaO_2 arterial oxygen partial pressure, *Hb* hemoglobin

* $P < 0.05$ vs time point 1 (baseline)

$^{\#}$ $P < 0.05$ vs time point 2

significantly compared with preoperative values. Larkin et al. [10] reported significant elevations in IOP during CPB, with a reduction of CVP to approximately zero and maintenance of mean arterial pressure at 60 mmHg. Abbott et al. [11] demonstrated a significant increase in IOP soon after the onset of CPB when crystalloid solution was used for priming, with unchanged IOP when colloid solution was used.

Contrary to the unchanged or increased IOP in previous reports, our study demonstrated a significantly decreased IOP during CPB. The reason for this discrepancy is not clear but may be attributed to variations of anesthetic techniques, CPB settings, and hemodynamic status. Intravenously and inhaled anesthetic agents are thought to decrease IOP in a variety of ways intraoperatively. Propofol used in this study, but not in previous studies [7–11], may explain the discrepancy. Mowafi et al. [13] demonstrated that IOP was significantly lower during laparoscopic surgery under propofol anesthesia compared with isoflurane. Furthermore, variations in CPB settings, including priming solution, temperature, oxygenator, and hemodynamic parameters including mean arterial pressure, CVP, hemoglobin level, and serum osmolality during CPB among the reports might have affected the results, although exact influence of each parameter on IOP remains undetermined.

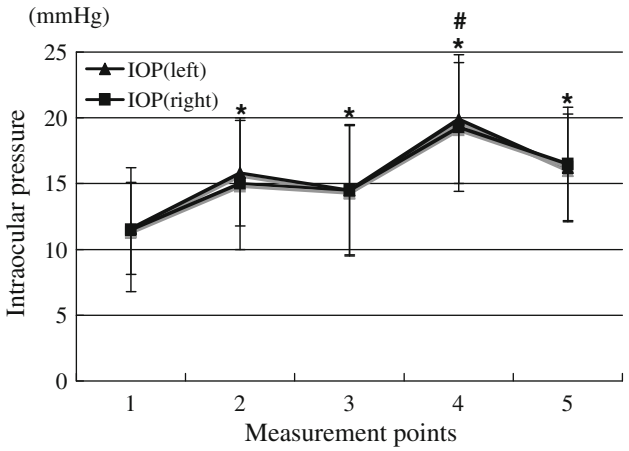

Fig. 2 Intraoperative changes in intraocular pressure (IOP) during cardiac surgery without cardiopulmonary bypass. Measurement time points: (1) 30 min after anesthesia induction (baseline); (2) prior to anastomosis; (3) during left anterior descending artery anastomosis; (4) during anastomosis left circumflex or right coronary artery (head-down position); and (5) at the end of operation. Bilateral IOPs were significantly increased and peaked at time point 4 in head-down position. $^{*}P < 0.05$ vs point 1. $^{\#}P < 0.05$ vs point 2

The actual mechanisms through which bilateral IOP values were significantly decreased during CPB in this study are unclear. However, possible explanations are as follows: First, changes in balance of aqueous humor production and drainage might have affected changes in IOP. Aqueous humor drainage might be increased due to a decrease in CVP during CPB. In fact, CVP was significantly decreased 60 min after the beginning of CPB. Aqueous humor production can also be decreased following a decrease in ophthalmic and ciliary artery blood flow, although we did not measure blood flow to the ciliary body, which produces aqueous humor. Second, a reduction of choroidal blood volume might have resulted in a decrease of IOP. Choroidal blood volume might be affected by ciliary artery blood flow and venous drainage to cavernous sinus, which can be influenced by a variety of physiological and pharmacological conditions. However, these premises are speculative. Further study would be required to clarify the exact mechanisms of decreased IOP during CPB.

Our results indicate a significant increase in IOP values during anastomosis in the head-down position compared with baseline (measurement time point 1) and before anastomosis (measurement time point 2) in patients without CPB. Increased IOP values reached abnormal range (>21 mmHg) [14] in the head-down position in five of 11 patients without CPB. The primary mechanism for this increase may be related to increased CVP in the head-down position. Increased CVP might result in a decrease in aqueous humor drainage and increase in choroidal blood volume. Draeger et al. [15] found that IOP increased immediately after head-down positioning in awake volunteers and suggested that IOP changed parallel to venous pressure. In addition to measurement time point 4 at the

Table 4 Previous reports investigating intraocular pressure during cardiopulmonary bypass

Study	Priming solution	Oxygenator	Temperature	Anesthesia	IOP
Levy et al. [7]	Crystalloid	Bubble	28°C	ND	Unchange
Stellpflug et al. [8]	ND	Bubble	ND	Neuroleptic	Increase
Lilleaasen et al. [9]	Crystalloid	Bubble	27–30°C	Morphine, diazepam, 50% N_2O	Unchange
Larkin et al. [10]	Crystalloid	Membrane	28°C	50% N_2O, intrathecal morphine	Increase
Abbott et al. [11]	Crystalloid	ND	28°C	BZ, enflurane, N_2O, fentanyl, morphine	Increase
	Colloid				Unchange
Hayashi et al. (this study)	Crystalloid	Membrane	28–34°C	Propofol, fentanyl, sevoflurane	Decrease

ND no available data, *N_2O* nitrous oxide, *BZ* benzodiazepine, *IOP* intraocular pressure

head-down position, IOP values were slightly but significantly increased at measurement time points 2, 3, and 5 compared with baseline (measurement time point 1), although blood pressure and CVP values remained unchanged. The reasons for this increase in IOP values are unknown. However, the use of catecholamines and volume loading for preparation of heart displacement during anastomosis might have affected IOP values, although increased IOP values were almost within normal range.

In summary, we investigated changes in IOP during cardiac surgery with a recently employed CPB and anesthetic management and without CPB. The results showed that bilateral IOP values were significantly decreased during CPB, whereas they significantly increased during anastomosis in the head-down position in patients without CPB. However, it is unknown whether these changes in IOP during cardiac surgery can contribute to ophthalmological complications, including ischemic optic neuropathy. Further studies are required to clarify the physiological mechanisms of IOP changes and the relationship between IOP changes and postoperative ophthalmological complications in patients undergoing cardiac surgery.

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