

Cardiopulmonary Bypass in a Glaucoma Patient

Jaswinder Singh, MCh; Rajinder Singh Dhaliwal, MCh; Debasis Das, MCh; Suvitesh Luthra, MCh; Harkant Singh, MCh

Department of Cardiovascular and Thoracic Surgery, Postgraduate Institute of Medical Education and Research, Chandigarh, India

Abstract: Cardiopulmonary bypass (CPB) in a patient with glaucoma is a challenge. The glaucomatous eye is at risk during CPB. We report a case of ostium secundum atrial septal defect that was not amenable to device closure. The unique feature in the patient was the presence of congenital glaucoma. She was blind in the left eye, and the visual acuity in the other

eye was decreased because of glaucoma. She underwent direct closure of the atrial septal defect under CPB and fibrillatory arrest, with intraoperative monitoring of intraocular pressure. There was no change in visual acuity after 1 year of follow-up. **Keywords:** cardiopulmonary bypass, glaucoma. *JECT 2007;39: 53-54*

Neurophthalmologic complications associated with open heart surgery have been reported with increasing frequency up to an incidence of 25% (1). These have been attributed to a variety of physiologic changes occurring during cardiopulmonary bypass (CPB) that include systemic hypotension, embolic phenomenon, cerebral hypoperfusion, hypothermia, and changes in intraocular pressure (IOP). The glaucomatous eye is at a risk during CPB (2). The aim of this report is to highlight that pre-existing glaucoma increases the risk for postoperative ophthalmic complications, and glaucoma should not be overlooked during CPB.

CASE REPORT

A 13-year-old female patient, who was known to have congenital bilateral glaucoma, presented with exertional shortness of breath (NYHA class II) of 2-year duration. The patient had undergone trabeculectomy in the past and was receiving anti-glaucoma medications. Clinical examination suggested the presence of an atrial septal defect (ASD) with a significant left to right shunt. Echocardiography revealed a 27-mm ostium secundum ASD. The ASD was not amenable to closure by percutaneous interventional means. An ocular examination showed a blind left eye and visual acuity of 6 of 60 in the right eye.

The anti-glaucoma medications were continued until the time of surgery. The patient underwent direct closure

of the ASD under CPB and fibrillatory arrest with moderate hypothermia of 32°C. Fibrillatory arrest time was 10 minutes. Care was taken to keep the perfusion pressure >60 mmHg. The IOP was monitored during the course of surgery using a Perkins handheld applanation tonometer (Haag-Streit USA, Inc., Mason, OH). The measurements of IOP were done 10 minutes after intubation of the trachea; 10 minutes after median sternotomy; 10 and 20 minutes after initiation of full flows on CPB; after rewarming to 37°C; and 10 minutes after weaning off CPB. The IOP rose within 10 minutes of achieving full flows on CPB. Gradual return to pre-CPB levels was seen on rewarming to 37°C and at 10 minutes after cessation of CPB (Table 1). The patient was managed with standard intensive care unit (ICU) protocol and was extubated after 4 hours. Anti-glaucoma medications were continued in the postoperative period. The patient had an uneventful postoperative recovery. There was no change in visual acuity in the early postoperative period and at 1 year of follow-up.

DISCUSSION

Contradictory results have been reported regarding IOP changes during CPB. Rapid intraoperative rise in

Table 1. IOP of the patient during surgery.

Time	IOP (mmHg)
Pre-CPB	13.4
10 minutes after intubation	13.4
10 minutes after sternotomy	13.3
On CPB 10 minutes	21.6
On CPB 20 minutes	20.2
On CPB 30 minutes	15.3
On rewarming (37°C)	13.8
Post-CPB 10 minutes	13.6

Address correspondence to: Jaswinder Singh, Department of Cardiovascular and Thoracic Surgery, Postgraduate Institute of Medical Education and Research, Chandigarh 160012, India. E-mail: drjaswindersingh@yahoo.co.in

The senior author has stated that authors have reported no material, financial or other relationship with any healthcare-related business or other entity whose products or services are discussed in this paper.

IOP during CPB has been reported; others reported a 6- to 7-mmHg rise in IOP during CPB with perfusion pressure maintained at 60 mmHg, whereas no difference was found in IOP values before and during CPB (1,3,4). IOP regulation during CPB is multifactorial; several factors during CPB reduce aqueous production, particularly hypothermia, hyperosmolality of prime fluid, and reduced perfusion pressure, whereas reduced colloidal osmotic pressure and hemodilution by priming fluid increases aqueous production. Supine position results in a higher increase in IOP in patients with anterior ischemic optic neuropathy (AION). A reduction in the total blood volume of the uvea may also contribute to a decrease in IOP. It is very important to keep the systemic venous pressure low (i.e., 12 mmHg) because for every 1-mmHg increase in episcleral venous pressure, there is an associated 1-mmHg increase in IOP, which is guided by the equation $IOP = F/C + P_v$ (F = force applied over cornea, C = corneal curvature, P_v = episcleral venous pressure). Perioperative anemia is another possible etiologic factor for anterior ischemic optic neuropathy. It is tolerated by most patients, but profound anemia may enhance the adverse effects on oxygen delivery, such as weight gain, tissue edema, use of inotropic agents, and increased CPB time. It has been recommended to keep hematocrit at 25%–30% (5). Hypothermia is protective to the brain, but in some instances may contribute to ischemia (6). Each degree Celsius fall is followed by a 6%–7% decrease in cerebral blood flow (7). Ischemic bipolar cells of the retinal inner nuclear layer is located in the vascular watershed boundary between the retinal and choroidal circulation and may be the most susceptible to hypoperfusion. During CPB, perfusion pressure should be kept around 60 mmHg because arterial pressure in the optic nerve terminal is about one half to one third (i.e., 20 mmHg).

Patients with preexisting glaucoma and elevated IOP may be at a higher risk for postoperative AION. One such patient has been reported to have undergone aggressive intraoperative monitoring and reduction of IOP by withdrawal of aqueous fluid (8). Another case with a past ophthalmologic history of glaucoma that underwent laser iridectomy developed a nasal defect in the left eye after coronary bypass grafting on the sixth postoperative day and underwent optic nerve decompression (2). In our case, we did not have to resort to controlling IOP with invasive means. Various antiglaucoma drugs were instilled locally into the eye; during hypothermic CPB, the pharmacokinetics may be altered through the following mechanisms:

(1) decreased absorption of the drugs administered other than by the intravenous route and (2) altered central nervous system drug penetration (9). Fortunately, in our patient, there was no change in visual acuity after CPB. Patients with increased IOP and a small cup to disc ratio are predisposed to development of AION after CPB (2). In these patients, prevention is the key because there is no specific treatment available for patients with AION. Shapira et al. (1) have recommended various preventive measures that include (1) brief duration of CPB, (2) use of colloid prime, (3) judicious use of fluids and hemoconcentration to reduce perioperative edema, (4) appropriate pCO_2 management to maintain cerebral autoregulation, (5) avoidance of prolonged low perfusion states or hypertension, (6) avoidance of profound hypothermia, (7) judicious use of inotropes and vasoconstricting agents, (8) maintenance of adequate hematocrit and hemoglobin, and (9) intraoperative monitoring and reduction of IOP by prophylactic perioperative installation of agents into the eye. Our case assumes importance because she was already blind in one eye and development of ocular complications would have been a catastrophic event.

In summary, the risk of perioperative visual loss is minimized if glaucoma is not ignored and the above-mentioned preventive measures are observed, thus allowing performance of safe cardiac surgery on CPB.

REFERENCES

1. Larkin DF, Connolly P, Magner JB, Wood AE, Eustace P. Intraocular pressure during cardiopulmonary bypass. *Br J Ophthalmol.* 1987;71:177–80.
2. Shapira OM, Kimme WA, Lindsey PS, Shahian D. Anterior ischemic optic neuropathy after open heart operations. *Ann Thorac Surg.* 1996;61:660–66.
3. Stellpflug H, Busse H, Niedermeier M, Dittrich H. Augenveränderungen während extrakorporaler zirkulation. *Ophthalmologica.* 1979;179:249–57.
4. Lilleaasen P, Horven I. Intraocular pressure levels during extracorporeal circulation in man. *Scand J Thorac Cardiovasc Surg.* 1982;16: 51–3.
5. Tice DA. Ischemic optic neuropathy and cardiac surgery. *Ann Thorac Surg.* 1987;44:677.
6. Johnston WE, Vinten-Johansen J, Dewitt DS, O'Steen WK, Stump DA, Prough DS. Cerebral perfusion during canine hypothermic cardiopulmonary bypass: effect of arterial carbon dioxide tension. *Ann Thorac Surg.* 1991;52:479–89.
7. Reuter JB. Hypothermia: Pathophysiology; clinical settings and management. *Ann Intern Med.* 1978;89:519–27.
8. Ohtake H, Misaki T, Matsunaga Y, Tubota M, Watanabe Y, Shirao Y. Open heart surgery under total extracorporeal circulation in a patient with advanced glaucoma. *Cardiovasc Surg.* 1993;1:190–1.
9. Ballard BE. Pharmacokinetics and temperature. *J Pharm Sci.* 1974; 63:1345–58.