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Perioperative Visual Loss in Cardiac Surgery

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Summary statement:

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Visual loss associated with cardiac surgery is due primarily to retinal artery occlusion and ischemic optic neuropathy. This review summarizes current state of knowledge concerning these visually disabling complications.

Keywords

cardiac surgery; ischemic optic neuropathy; microembolism; retinal artery occlusion; thrombosis

Perioperative visual loss (POVL) associated with non-ophthalmic surgery is a rare, serious complication. The main causes are ischemic optic neuropathy, and retinal arterial occlusion,

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^{1, 2} and less frequently, cortical blindness,³ acute glaucoma,⁴ and choroidal and vitreous hemorrhage.⁵ This review specifically relates to visual loss after cardiac surgery, which has received less attention than POVL after spinal surgery, despite higher incidence.^{6,7,8,9} Our purpose is to describe the epidemiology, clinical manifestations, mechanisms, and treatment of perioperative visual loss in cardiac surgery. We focus upon retinal artery occlusion and ischemic optic neuropathy.

Epidemiology

POVL occurs in approximately 1 in 60,000 to 1 in 125,000 anesthetic procedures in the overall surgical population in the United States and more frequently in spine fusion and cardiac surgery vs other operative procedures.^{6,7} Widely differing methodologies and numbers of subjects have resulted in a range of incidence estimates. Shen et al examined ischemic optic neuropathy in the U.S. National Inpatient Sample, reporting that cardiac (8.64 per 10,000, 0.086%) followed by spine (3.09 per 10,000, 0.03%) were the most common operations associated with ischemic optic neuropathy.¹⁰ In cardiac surgery, the incidence of ischemic optic neuropathy was reported as 0.06-0.113%, with retinal artery occlusion 7.7 per 10,000.^{6,11,12,13} The yearly rates of POVL in all types of surgery decreased in the 10-years 1996-2005.^{7,10} However, while cardiac surgery volumes have declined, POVL has remained stable.⁶

Retinal artery occlusion

Clinical Findings

Acute retinal artery occlusion is characterized by painless visual loss, a whitish ischemic retina, and retinal arteriolar narrowing.¹⁴ Central retinal artery occlusion produces diffuse central and peripheral vision loss, while branch retinal artery occlusion usually has preserved central and segmental peripheral visual field loss. Branch retinal artery occlusion may not be immediately apparent if the visual field loss is peripheral or small, resulting in delayed diagnosis. Both events are usually unilateral.

In central retinal artery occlusion, a cherry-red macula (“cherry red spot”) surrounded by an otherwise white retina and attenuated arterioles is a “classic” sign (Fig 1)¹⁵ as retinal swelling obscures the red color of the intact, underlying choroid, except in the fovea, the thinnest part of the retina. In branch retinal artery occlusion the white retina is a wedge widening towards the periphery with apex at the site of arteriolar occlusion (Fig 2).¹⁶ Because retinal whitening is due to edema, it may not be initially apparent, and resolves as the retina atrophies over weeks. In the subacute setting, fluorescein angiography can visualize reduced arteriolar perfusion (Fig 1). After the event, the affected vessels usually reperfuse and angiography may be normal.

Mechanisms of Retinal Artery Occlusion

Three main mechanisms produce spontaneous central retinal artery occlusion: (1) external ocular compression causing arteriolar compression, (2) decreased arterial supply (embolism to retinal arterial circulation, decreased systemic blood flow, vasospasm and/or thrombosis), (3) impaired venous drainage. Spontaneous branch retinal artery occlusion is primarily from

emboli, and less often, vasospasm or thrombosis. There are only three case reports of retinal artery occlusion after cardiac surgery with sufficient detail to provide insight into mechanism,^{17–19} and therefore it is difficult to identify mechanisms most pertinent to POVL following cardiac surgery. The most logical from a pathophysiological perspective are retinal embolization and thrombosis.

Emboli—Perioperative retinal arterial occlusion in cardiac surgery has long been suspected to be primarily embolic.^{13,20} Most cases describe embolization from intravascular injections, the surgical field, or related to cardiopulmonary bypass (CPB), especially left sided cardiac valve surgery.^{21,22} In coronary artery bypass (CABG) surgery, multiple calcific emboli in branches of the central retinal artery are not unusual, resulting in visual field deficits of varying size and location.²³ Although there is considerable evidence of microemboli in cardiac surgery, the visual consequences have not been well documented, possibly because most are asymptomatic.²⁴

The mechanism of embolic retinal artery occlusion may be similar to embolic stroke, which could be associated with use of specific CPB circuits. In 13 randomized trials with 1161 patients a minimized CPB circuit was associated with reduced stroke vs conventional CPB circuit (0.2% vs 2%, respectively; $p = 0.05$).²⁵ Several smaller studies examined microembolic phenomena in the retina with CPB, with findings supporting the hypothesis that mechanisms are similar to those with cerebral embolism.

Rimpilainen et al compared two groups of patients undergoing aortic valve replacement with or without CABG. One was “conventional” and the other “minimized” CPB.²⁶ Retinal microemboli were compared using retinal fluorescein angiograms before and after CPB. Perfusion defects were in 7/19 in the minimized vs 12/19 patients in conventional CPB, but with small sample size, the difference was not statistically significant. Inflammatory markers, coagulation, and endothelial dysfunction increased similarly in both. While potentially clinically advantageous, minimized CPB circuits are not commonly used. Further, an important difference was that minimized CPB was associated with increased post-CPB bleeding and platelet transfusion.^{27, 28} Historically, bubble oxygenators were associated with increased retinal microemboli and postoperative neurocognitive deficits.^{27–31} These oxygenators, however, are rarely used in clinical practice today.

Manipulating a heavily calcified and atheromatous aorta is another important source of cerebral and retinal embolization.^{32,33} Epiaortic ultrasound prior to aortic cannulation may help avoid atheromatous regions not well visualized by transesophageal echocardiography, decreasing embolization.^{34–36} Epiaortic ultrasound was also superior in detecting non-calcific “soft” plaque vs manual examination.³⁷ Furthermore, routine epiaortic ultrasound scanning of the ascending aorta to reduce atheroembolic complications received a class IIA recommendation in the ACCF/AHA Guidelines for Coronary Artery Bypass Graft Surgery.³⁸ But, its efficacy to decrease retinal microemboli is unstudied.

Paradoxical air embolism from the operative site via patent foramen ovale is another potential cause of perioperative retinal vascular occlusion,³⁹ which has not been well studied in humans. In pigs, mechanisms of air embolism damage during CPB included non-

perfusion, vascular leakage and spasm, red blood cell (RBC) sludging, and hemorrhage. Priming with perfluorocarbons blocked many of these mechanisms.⁴⁰

Thrombotic phenomena—Thrombosis in a central or a branch retinal artery associated with hypercoagulability has been reported.^{41–45} In particular, thrombophilic factors such as hyperfibrinogenemia and Factor V Leiden mutation increased risk.⁴⁶ Thrombophilia after cardiac surgery may contribute to postoperative cardiac and cerebral complications.^{47, 48} Using thromboelastography, perioperative hypercoagulability was as high as 43%.^{49,50} In a series of cardiac surgery cases complicated by increased thrombosis, depressed myocardial function, CPB duration longer than three hours, aortic injury, and platelet transfusion were associated factors.⁵⁰

Administration of pro-hemostatic coagulation factors [prothrombin complex concentrate (PCC), recombinant factor VIIa (rFVIIa), or fibrinogen concentrate] in patients with excessive post-CPB bleeding has been increasing. While timely achievement of hemostasis could be life-saving, concerns have been raised about increased thromboembolism. For example, 4-factor PCC has been associated with prolonged elevated vitamin K-dependent coagulation factors,⁵¹ and 5-7% of patients receiving PCC for warfarin reversal develop thromboembolic complications.⁵² Similarly, in 35 randomized clinical trials of 4468 individuals, rFVIIa was associated with a significantly increased risk for arterial thromboembolism particularly in elderly.⁵³ However, no specific link between prohemostatic agents and retinal artery occlusion has been reported, thus, further investigation is needed. Moreover, since morbidity and mortality due to excessive post-CPB bleeding is significantly higher than POVL, appropriate use of these products should not be avoided or restricted in an attempt to decrease POVL even in high-risk patients.

Systematic Study of Risk Factors

To date, there is only one systematic study of risk factors for retinal artery occlusion in cardiac surgery. Among more than 5.8 million cardiac operative procedures from 1998-2013 in the US National Inpatient Sample, there were 4,564 cases of retinal artery occlusion, or 7.77 per 10,000.¹³ Risk factors included history of transient cerebral ischemia, carotid artery stenosis, embolic stroke, hypercoagulability, atrial myxoma, diabetes mellitus (DM) with ophthalmic complications, and aortic insufficiency. Significant operative factors were bleeding, aortic and mitral valve surgery, and septal surgery (Table 1). These results further indicate that conditions that predispose to embolic phenomena, such as carotid disease, open heart procedures and pre-existing abnormal retinal vasculature (diabetic retinopathy) are potential risk factors for retinal artery occlusion in cardiac surgery.

Treatment

Currently available treatment is unsatisfactory. Ocular massage to decrease intraocular pressure and dislodge emboli, if present, to more peripheral arterial branches could be instituted.¹⁴ Intravenous acetazolamide (carbonic anhydrase inhibitor) may decrease CSF production, decreasing intraocular pressure and increasing retinal blood flow. Intravenous thrombolysis may be an option in retinal artery occlusion, however, it is contraindicated after most cardiac surgical procedures. Intra-arterial fibrinolysis via a catheter in the ophthalmic

artery within 6-8 h after spontaneous central retinal artery occlusion was associated with improved visual outcome, but its use remains controversial.⁵⁴

Prevention

A small prospective trial randomized CABG patients to CPB (CABG-CPB) or off-pump (OPCAB).⁵⁵ Retinal microvascular damage was detected in 5 of 9 CABG-CPB, and 0 of 10 OPCAB ($p = 0.01$). Doppler high-intensity transient signals (HITS), indicating cerebral microemboli, were 20 times more frequent in CABG-CPB vs OPCAB ($p < 0.0001$). HITS were almost 15 times more frequent ($p = 0.001$) and S100 (marker of cerebral injury) 2.1 times higher ($p = 0.005$) when retinal microvascular damage was present, demonstrating the protective effect of OPCAB in avoiding cerebral and retinal microemboli. These findings notwithstanding, as large randomized controlled trials failed to demonstrate that OPCAB decreased neurological complications after CABG,^{56,57} more investigation is required before general recommendations can be made.

For CPB, avoiding atheromatous areas in the ascending aorta and the aortic arch are in theory the best means to minimize retinal microembolism during cardiac surgery. The beneficial role of epiaortic ultrasound has reviewed.^{34,35,58} Minimizing aortic manipulation by single clamp application also reduced postoperative cognitive deficits.⁵⁹

Ischemic Optic Neuropathy

Clinical findings

Ischemic optic neuropathy is characterized by painless visual loss in one or both eyes; it most often occurs spontaneously, but also can occur perioperatively. Diagnosis is typically within 24-48 hours after surgery, but may be delayed in sedated patients.⁶⁰ Vision loss is often severe with central and peripheral visual deficits and loss of light perception. Unilateral injury often has a relative afferent pupillary defect (pupil constriction is decreased, appearing to dilate when swinging a bright light from unaffected to affected eye). Ischemic optic neuropathy is categorized according to the segment of optic nerve involved. In anterior ischemic optic neuropathy the optic nerve head is swollen acutely and there can be hemorrhages and cotton wool spots on and around the optic nerve head, followed by late pallor (Fig 3). In posterior ischemic optic neuropathy the optic nerve head appears normal, however, optic atrophy develops over time and the optic nerve head later appears pale.⁶¹ Most of the cases described in cardiac surgery are of the anterior variety (Table 2). Although ancillary tests have not been studied in ischemic optic neuropathy after cardiac surgery, in spontaneous ischemic optic neuropathy, magnetic resonance imaging is frequently normal, although some studies have described enlargement of the nerve from edema or perineural enhancement.^{62,63}

Mechanisms

Ischemic optic neuropathy (either anterior or posterior) comes in two forms, arteritic or non-arteritic. Arteritic usually occurs in older patients and is caused most often by temporal arteritis. In contrast, non-arteritic anterior optic neuropathy usually affects those with a morphologically small or crowded optic nerve head, the “disc-at-risk.”⁶⁴ For unclear

reasons, most ischemic optic neuropathy in cardiac surgery has been anterior, in contrast to ischemic optic neuropathy associated with spine surgery which is predominantly posterior.^{65–68}

Fluorescein angiography showed dye leakage in the optic nerve head suggesting increased vascular permeability,⁶⁹ and early onset of optic disk edema in anterior ischemic optic neuropathy.⁷⁰ However, another study showed delayed filling of the prelaminar optic disk in 76% of anterior ischemic optic neuropathy subjects, not found in normal eyes, suggesting that delayed filling is primary.⁷¹ Hayreh attributed anterior ischemic optic neuropathy to individual variations in optic nerve blood supply.⁷² Anatomic studies and the variability of visual loss support this theory. A watershed concept, i.e., impaired perfusion and distribution within a posterior ciliary artery sets up the optic disk to infarction—is controversial. Arnold and Hepler showed that delayed filling of watershed zones was more frequent in normal eyes than in anterior ischemic optic neuropathy.⁶⁹ Thus, reduced perfusion pressure in the para-optic branches of the short posterior ciliary arteries results in optic disk hypoperfusion, not a watershed event (Fig 4).⁷³ The number of cases of human ischemic optic neuropathy with documented histopathological study is limited.⁷⁴ In anterior ischemic optic neuropathy, histological studies found infarction mainly in the retrolaminar region.⁷⁵ This implicates the short posterior ciliary arteries as the source of decreased blood flow.

A small optic disk (low cup-to-disk ratio, C/D, Fig 4) may predispose to anterior ischemic optic neuropathy. C/D is a component of routine comprehensive eye examinations, and patients may be aware if it has been explained to them by the eye care provider. A ratio < 0.2 is associated with increased risk of spontaneous ischemic optic neuropathy^{76–79} via mechanical axoplasmic flow obstruction, stiff cribriform plate, and decreased availability of neurotrophic factors to retinal ganglion cells.^{78–80} While well established in spontaneous cases of ischemic optic neuropathy, the relationship between small optic disc size and perioperative ischemic optic neuropathy in cardiac surgery is undetermined.

Risk factors

Several retrospective case control studies have yielded insight into the mechanisms and risk factors for ischemic optic neuropathy in cardiac surgery (Table 2). In a single institution retrospective review⁸¹ of 602 patients undergoing cardiac surgery with CPB under moderate systemic hypothermia with pulsatile flow and a membrane oxygenator, eight of 602 (1.2%) developed anterior ischemic optic neuropathy. CPB time was longer (252 vs 164 min) and minimum hematocrit was lower (18 vs 21%) in patients with anterior ischemic optic neuropathy vs unaffected. Patients with ischemic optic neuropathy had greater mean 24-h postoperative weight gain (18 vs 11%) and required more vasoactive drugs to maintain adequate blood pressure. This study may be criticized as a single institution design, small sample size, and the low hematocrit level during CPB (below the usually acceptable range today). Nuttall et al's retrospective case-control study included over 28,000 cardiac surgery procedures at Mayo Clinic from 1976–1994, with 17 developing ischemic optic neuropathy (0.06%). Nine were bilateral; 5 (29%) had posterior ischemic optic neuropathy, with the remainder having anterior ischemic optic neuropathy. Small C/D was in 5 (29%).¹¹ Associations with ischemic optic neuropathy included lower minimum postoperative

hemoglobin level, clinically severe vascular disease, preoperative coronary angiogram within 48 h of surgery, longer CPB, red blood cell transfusions, and non-RBC blood component transfusions. This study was limited by its single institutional design and lack of differentiation between posterior and anterior ischemic optic neuropathy, as well as lack of correction for multiple comparisons. Holy et al⁸² reviewed more than 126,000 surgical cases (cardiac and non-cardiac) between 1998 and 2004, reporting 17 cases of ischemic optic neuropathy. Nine were in cardiac surgery out of a total of 2749 operations (0.33%), significantly higher than Nuttall et al. However, the authors could not determine any perioperative specific risk factors, except for low C/D in six out of eight patients with anterior ischemic optic neuropathy. Kalyani et al studied visual loss after 9701 cardiac surgeries over 9 years. Specific risk factors could not be determined from the 11 ischemic optic neuropathy patients (0.11%).¹²

Rubin et al published the largest series in cardiac surgery using the National Inpatient Sample (1998-2013). More than 5 million discharges met inclusion criteria with 794 (0.014%) cases of ischemic optic neuropathy. Incidence was 1.43/10,000 procedures per year. Carotid artery stenosis, stroke, diabetic or hypertensive retinopathy, macular degeneration, glaucoma, and cataract were associated with ischemic optic neuropathy while female sex and uncomplicated DM type 2 were inversely associated. The observation that degenerative eye diseases are associated with ischemic optic neuropathy is particularly interesting as it suggests there may be both local and systemic risk factors.⁶ The limitations include possibility of over and under-coding using medical claims data, and inability to independently confirm diagnoses.¹⁰

Treatment

In some patients, spontaneous improvement in vision has been reported. No effective treatment exists for spontaneous or perioperative ischemic optic neuropathy. Buono and Foroozan summarized the lack of evidence that treatment altered the course of the disease.⁶⁵ A few cases of treating perioperative ischemic optic neuropathy have been reported. Acetazolamide inhibits carbonic anhydrase, decreasing aqueous humor formation and lowering intraocular pressure, potentially improving blood flow to the optic nerve head and retina.⁸³ Corticosteroids may reduce axonal swelling, but in the postoperative period they may increase wound infection and complicate glucose management, therefore, their use must be carefully weighed against risks.⁸⁴ Increasing blood pressure or hemoglobin may be appropriate when ischemic optic neuropathy is diagnosed in conjunction with significant decreases in blood pressure and anemia.

Diminishing Risks and Prevention

Anatomic variation in the circulation of the optic nerve may potentially predispose patients to ischemic optic neuropathy. The location of potential watershed zones in the anterior and posterior circulation and the presence of disturbed autoregulation, even in normal patients,⁸⁵ are of concern but at present cannot be predicted clinically. Patients with known C/D < 0.2 should be informed that there is an increased risk to develop ischemic optic neuropathy, however, it is undetermined if surgical management should be altered (e.g., performing off-bypass surgery).

While a suggested risk factor for ischemic optic neuropathy after spine surgery,⁸⁶ there is presently no evidence that the length of cardiac surgery impacts ischemic optic neuropathy, however, longer bypass time may be a factor. Thus in patients perceived to be of higher risk, it may be advisable to minimize CPB time. With regard to hypotension, it can potentially lead to decreases in perfusion pressure in the optic nerve and to ischemic injury because of either anatomic variation in the circulation or abnormal autoregulation and an inability to adequately compensate for decreased perfusion pressure. The degree of hypotension that is potentially dangerous is difficult to quantitate because of lack of data.⁸⁷ Human studies generally show preserved blood flow in the anterior optic nerve at clinically used or even lower ranges of perfusion pressure.⁸⁸ It is not currently feasible to measure blood flow in the human retrolaminar optic nerve. In animal studies, blood flow is preserved in various layers of the optic nerve, including the retrolaminar area, at a mean arterial blood pressure as low as 40 mm Hg.⁸⁹

There have been no studies to suggest what constitutes an adequate blood pressure to prevent ischemic optic neuropathy.⁹⁰ Patients with other conditions that may be associated already with a “nerve at risk” (e.g., carotid artery stenosis, stroke, diabetic or hypertensive retinopathy, macular degeneration, glaucoma)⁶ may require higher perfusion pressures.

Hemodilution is common during cardiopulmonary bypass.⁹¹ The critical lowest hemoglobin level or the duration for which it needs to exist to lead to this complication is not known.⁹² In miniature pigs, blood flow in the optic nerve head measured by laser Doppler imaging was maintained during isovolumic hemodilution with a 30% decrease in hematocrit. Moreover, O₂ tension at the vitreal surface increased 15%.⁹³ Although Lee et al⁹⁴ demonstrated that extreme decreases in hematocrit (15%) and mean arterial pressure (50 mm Hg) in adult pigs reduced blood flow to the optic nerve, histology or optic nerve function were not studied, and the pig eye circulation significantly differs from humans in that, among other differences, the external carotid supplies the majority of the posterior ocular circulation, and there is no single central retinal artery that vascularizes the retina or supplies a portion of the optic nerve.⁹⁵ Roth et al showed that hemodilution and extreme head down tilt in rats significantly reduced visual evoked potentials and electrical activity originating in retinal ganglion cells, and increased glial reactivity in the optic nerve.⁹⁶ These results are not readily extrapolated to cardiac surgery, because head down position, if used, is not ordinarily sustained. Nor do we recommend that visual evoked potentials be used during cardiac surgery. The disadvantages include need for minimal inhalational anesthetic agent, technical issues in dark adaptation of the retina, and unknown sensitivity or specificity to detect ischemic optic neuropathy.⁹⁷

The theory that massive intravascular fluid resuscitation could be a pathogenic factor in perioperative ischemic optic neuropathy remains speculative, but does have some merit.⁸¹ The American Society of Anesthesiologists POVL Study Group found that the odds ratio for developing ischemic optic neuropathy in spine surgery was increased as percent colloid of nonblood replacement decreased.⁸⁶ However, this finding may also be closely related to prone positioning in spine surgery, which obviously is not used in cardiac surgery.

Vasoconstrictors are commonly used in cardiac surgery patients. Hayreh theorized that spontaneous ischemic optic neuropathy was related to excessive secretion of vasoconstrictors, which in turn could lower optic nerve perfusion to dangerously low levels. However, the hypothesis was based on the development of non-anterior ischemic optic neuropathy in patients who sustained massive hemorrhage.⁹² Shapira et al showed an association between prolonged use of epinephrine or long bypass time and ischemic optic neuropathy in patients undergoing open heart surgery.⁸¹ Lee and Lam presented four cases of ischemic optic neuropathy in critically ill patients with significant systemic illness who required prolonged use of vasopressors and inotropic agents to maintain blood pressure and cardiac output.⁹⁸ However, α -adrenergic receptors are not located in the optic nerve and the blood-brain barrier prevents entry of systemically administered vasopressors. Given the widespread use of vasoactive medications during and after cardiac surgery, a direct link between vasopressors and ischemic optic neuropathy appears less likely. We do not recommend any change in care nor restrictions on vasopressors in cardiac surgery patients with high risk for ischemic optic neuropathy.

Conclusions:

Perioperative visual loss after cardiac surgery is a rare complication usually caused by ischemic optic neuropathy or retinal arterial occlusion. These complications can result in devastating consequences with very few treatment options to reverse the damage. A single institution survey reported that patients prefer to be informed of the risk of visual loss in spine surgery.⁹⁹ It seems advisable to have the discussion with patients that are at higher risk of developing POVL. It may be preferable for anesthesiologists and surgeons to develop systematic means to inform patients of the risks of retinal artery occlusion and ischemic optic neuropathy.

For retinal artery occlusion, potential risk factors include carotid artery stenosis, embolic stroke, hypercoagulability, myxoma, diabetes mellitus with ophthalmic complications, and aortic insufficiency. Perioperative factors were bleeding, aortic and mitral valve surgery, and septal surgery, and in particular, conditions that increase embolism such as manipulation of an atheromatous aorta. For ischemic optic neuropathy, potential risk factors include carotid artery stenosis, stroke, diabetic or hypertensive retinopathy, macular degeneration, and glaucoma. The roles of hypotension, length of surgery, hemodilution, vasopressors, and fluid administration are uncertain.

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Figure 1:

Acute central retinal artery occlusion. The fundus image (left) shows narrowing of retinal arterioles, retinal whitening, and the characteristic cherry red spot (left of center, blue arrow). The red arrow in the left image shows the retinal pallor. The fluorescein angiogram (**right**) demonstrates absence of flow out of the central retinal artery (red arrow), and the areas of decreased perfusion in the retina (blue arrows). (Picture provided by Diana Do, MD, Department of Ophthalmology, Stanford University)

Figure 2:

Acute branch retinal artery occlusion. The fundus image shows the characteristic wedge-shaped retinal whitening (blue arrow) in the region of occlusion (in this case, a branch of the supero-temporal retinal artery) corresponding to the area of non-perfusion. (Cho HK, Cerebral small vessel disease in branch retinal artery occlusion. *Investigative Ophthalmology and Visual Science* 2016;57: 5818-5824 used with permission).

Figure 3: Anterior ischemic optic neuropathy. The left fundus image shows the characteristic disc swelling (dark blue arrow) in the acute phase, and the splinter hemorrhages in the retina (light blue arrows). The right fundus image demonstrates optic atrophy (blue arrow) of the chronic phase.

Figure 4:

The blood supply to the optic nerve is primarily from the ophthalmic artery. The posterior optic nerve is supplied by the ophthalmic artery posteriorly and posterior ciliary arteries anteriorly. The central region supply is limited to the branches that penetrate deeply. The optic nerve head is supplied by the circle of Zinn, which is from the short posterior ciliary arteries, and from choroidal vessels. The cup-to-disc ratio is the central cup diameter divided by diameter of the entire disc. (Bioussé V, Newman NJ. Ischemic Optic Neuropathies. New England Journal of Medicine. 2015;372:2428-36 used with permission)

Table 1:

Risk Factors for Retinal Artery Occlusion in Cardiac Surgery from Rubin et al, 2017

Pre-operative Risk Factors	Surgery/Intraoperative Risk Factors
Transient cerebral ischemia	Bleeding
Carotid artery stenosis	Aortic valve surgery
Embolic stroke	Mitral valve surgery
Hypercoagulability	Septal surgery
Atrial myxoma	
Diabetic eye complications	
Aortic insufficiency	

Table 2:
Summary of study findings in case control studies of ION in cardiac surgery.

Abbreviations: AION = anterior ischemic optic neuropathy; CABG= coronary artery bypass grafting; CPB = cardio-pulmonary bypass; DM = diabetes mellitus; ION = ischemic optic neuropathy; PION = posterior ischemic optic neuropathy; RBC= red blood cells

Authors	Year	Type	Study size	AION or PION	Findings
Shapira et al	1996	Single institution, retrospective case control	602 total, all CPB	8, all AION	<ul style="list-style-type: none"> • CPB time longer AION vs non affected (252 vs 164 min) • Minimum hematocrit lower AION vs non affected (18 vs 21%) • Flow indices, perfusion pressures, and Pco₂ did not differ. • AION greater mean 24-h postoperative weight gain (18 vs 11%) and required more vasoactive drugs to maintain hemodynamics.
Nuttall al	2001	Single institution, retrospective case control	28,000 total, all CPB	12 AION 5 PION Total 17	<ul style="list-style-type: none"> • Cup-to-disk ratio < 0.3 in 5 cases (AION) • Associations with ION: lower minimum postoperative hemoglobin, clinically severe vascular disease, preoperative angiogram within 48 h of CPB, longer CPB duration, red blood cell transfusions, and non-RBC blood component transfusions.
Holy et al	2009	Single institution, retrospective case control	2749 CABG, all CPB	7 AION 2 PION Total 9	<ul style="list-style-type: none"> • No significant differences between cases and controls for CABG (small sample size, and 2 controls per case)
Kalyani et al	2012	Single institution, retrospective case control	9701 total, all CPB	8 AION 3 PION Total 11	<ul style="list-style-type: none"> • No significant differences between cases and controls (small sample size, and 3 controls per case)
Rubin et al	2017	Retrospective case control using National Inpatient Sample	5.5 million, CPB in 75% of affected	794 total; no distinction AION/PION	<ul style="list-style-type: none"> • Increased risk with: carotid artery stenosis, stroke, diabetic or hypertensive retinopathy, macular degeneration, glaucoma, and cataract • Female sex and uncomplicated DM type 2 inversely associated with ION.