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Perioperative Ischemic Optic Neuropathy

A Case Control Analysis of 126,666 Surgical Procedures at a Single Institution

Sarah E. Holy, M.D., Jonathan H. Tsai, M.D.,* Russell K. McAllister, M.D.,† Kyle H. Smith, M.D.‡*

Background: **Ischemic optic neuropathy is the most common cause of perioperative vision loss. The authors sought to determine its incidence and identify risk factors that may contribute to perioperative ischemic optic neuropathy associated with nonophthalmologic surgical procedures at their institution.**

Methods: **Seventeen patients who experienced perioperative ischemic optic neuropathy were included in a retrospective chart review case-control study. The authors matched each patient with two control patients who had a similar surgical procedure but did not lose vision. They analyzed multiple perioperative variables for the case and control groups.**

Results: **From among 126,666 surgical procedures performed during the study period, the authors identified 17 patients with perioperative ischemic optic neuropathy, yielding an overall incidence of 0.013%. There were no hemodynamic variables that differed significantly between the ischemic optic neuropathy patients and the matched control patients.**

Conclusion: **The authors conclude that perioperative ischemic optic neuropathy can occur in the absence of atypical fluctuations in hemodynamic variables during the perioperative period.**

LOSS of vision can be a devastating complication of an otherwise successful nonocular surgical procedure. Perioperative vision loss may be a manifestation of retinal ischemia, cortical infarction, or ischemic optic neuropathy (ION). The most common causes of perioperative vision loss are the two different forms of ION: Anterior ischemic optic neuropathy (AION) and posterior ischemic optic neuropathy (PION).[1,2] The incidence of perioperative ION reported in the literature varies between 0.028 and 1.3%.[3,4] Several published case series in recent years have heightened the awareness of this potential complication among anesthesiologists, surgeons, and ophthalmologists.[1,3–6]

The operations most commonly associated with perioperative ION are coronary artery bypass grafting (CABG) and back or spinal surgery.[7] The cause of the ischemic damage to the optic nerve in this setting is not completely understood, but perioperative anemia, hypotension, facial or orbital edema, and direct pressure on the globe are reported as potential etiologic factors.[8–10] The use of intraoperative vasopressors has also been implicated as a possible contributing factor in the development of ION.[4] Preexisting comorbidities, such as diabetes mellitus, hypertension, hypercholesterolemia, smoking, and heart disease are associated with spontaneous AION, and may also be important variables in the etiology of perioperative ION.[11,12] We initiated this retrospective chart review case-control study to determine the incidence of perioperative ION at our institution, and to evaluate the preoperative, intraoperative, and postoperative risk factors that may contribute to perioperative ION after nonophthalmologic surgical procedures.

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Materials and Methods

After obtaining Institutional Review Board approval (Institutional Review Board, Scott and White Memorial Hospital, Temple, Texas), we initiated this retrospective chart review case-control study by reviewing the documented cases of perioperative ION after nonocular surgical procedures at our institution between January 1998 and December 2004. All patients who report visual loss after surgery in our hospital are examined by one of our faculty ophthalmologists, and any who are suspected of having an optic neuropathy are referred to one neuro-ophthalmologist. This physician examined and confirmed the diagnosis of each patient in this series. We performed a text query of the medical records associated with this physician during the specified time period of the study, identified every inpatient and outpatient record that included "ischemic optic neuropathy" in the diagnosis, and then reviewed each record to identify those instances of ION that occurred in a perioperative setting. This search yielded the 17 cases that are included in this study.

We included in our study all patients who noted visual loss within 1 week of the procedure, and who had documented evidence of acute AION or PION when examined by the neuro-ophthalmologist (table 1). The diagnostic criteria for AION included the presence of optic disc swelling when first examined, a relative afferent pupillary defect, and demonstrable visual acuity or

* Assistant Professor of Ophthalmology, ‡ Associate Professor of Ophthalmology, Department of Ophthalmology; † Associate Professor of Anesthesiology, Department of Anesthesiology; Scott and White Memorial Hospital and Clinic; Scott, Sherwood and Brindley Foundation; and The Texas A&M University Health Science Center College of Medicine; Temple, Texas.

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Address correspondence to Dr. Holy: The Texas A&M University Health Science Center College of Medicine, Scott and White Memorial Hospital and Clinic, Division of Ophthalmology, 2401 South 31st Street, Temple, Texas 76504-7115. sholy@swmail.sw.org.

Table 1. Patient Characteristics

Patient	Age	Sex	Surgery	Ophthalmic Exam	Final Visual Acuity	Final Diagnosis
1	60	M	CABG	APD OS; pallid edema OS; superior, temporal and inferior HVF defect OS	OD: 20/20 OS: 20/25	AION, OS
2	54	M	CABG	APD OS; optic disc edema OS; superior, nasal and inferior HVF defect OS	OD: 20/30 OS: 20/30	AION, OS
3	64	M	CABG	APD OS; optic disc drusen; inferior altitudinal HVF defect OS	OD: 20/20 OS: 20/20	AION, OS
4	55	M	CABG	Nonreactive pupils OU	OD: No light perception OS: No light perception	AION, OU
5	71	M	Spine	APD OS; pallid edema OU	OD: 20/50 OS: No light perception	AION, OU
6	72	M	CABG	APD OS; superior altitudinal defect OS	OD: 20/30 OS: 20/60	AION, OS
7	60	M	CABG	APD OD; superior altitudinal defect OD; trace pallid edema OD	OD: 20/50 OS: 20/70	AION, OD
8	54	M	CABG	APD OD; disc edema OU; HVF with preservation of only superior field OU	OD: 20/400 OS: 20/20	AION, OU
9	63	M	Spine	APD OD; normal exam initially, late pallor of optic nerve OD	OD: 2/200 OS: 20/25	PION, OD
10	62	M	Bilateral leg fracture repair	APD OD; normal exam initially, late pallor OU	OD: Light perception OS: 2/200	PION, OU
11	77	M	Spine	Trace pupillary reaction OU; normal initial optic nerve exam	OD: Light perception OS: No light perception	PION, OU
12	88	M	Thoracotomy, segmentectomy	Normal initial optic nerve exam OS; late pallor OS	OD: No light perception (secondary to neovascular glaucoma) OS: 20/200	PION, OS
13	61	M	CABG	Bilateral superior HVF defect OU; inferior pallor OU	OD: 20/20 OS: 20/20	PION, OU
14	68	F	CABG	APD OD from old AION; superior altitudinal visual field defect OS; optic disc pallor OS	OD: Hand motion (secondary to old AION) OS: 20/30	PION, OS
15	66	M	Bilateral knee replacement	APD OS; Inferior altitudinal defect OS; difficulty detecting red on color vision testing OS	OD: 20/25 OS: 20/60	PION, OS
16	66	M	Spine surgery	APD OS; normal exam initially; late pallor OS; central scotoma on visual field testing	OD: 20/25 OS: 20/200	PION, OS
17	70	M	Femoral aneurysm repair	No APD documented; optic disc pallor OU; superior confrontation visual field defect OD	OD: 5/200 OS: 20/40	PION, OU

AION = anterior ischemic optic neuropathy; APD = afferent pupillary defect; CABG = Coronary artery bypass graft; HVF = Humphrey visual field; OD = right eye; OS = left eye; PION = posterior ischemic optic neuropathy.

Table 2. Surgical Procedures of Patients and Controls

Patient	Surgical Procedure of Patient	Surgical Procedure of Control A	Surgical Procedure of Control B
1	CABG, 3 vessels; bypass used	CABG, 5 vessels; bypass used	CABG, 3 vessels; bypass used
2	CABG, 2 vessels; bypass used	CABG, 1 vessel; bypass used	CABG, 2 vessels; bypass used
3	CABG, 5 vessels; bypass used	CABG, 5 vessels; bypass used	CABG, 5 vessels; bypass used
4	CABG, 3 vessels; bypass used	CABG, 6 vessels; bypass used	CABG, 4 vessels; bypass used
5	Arthrodesis T5–T7, anterior instrumentation T5–T7, removal of herniated disc T6-T7	Left L4-L5 exploration with removal of herniated disc	Multiple-level decompression and arthrodesis, L3–L5
6	CABG, 6 vessels; bypass used	CABG, 6 vessels; bypass used	CABG, 6 vessels; bypass used
7	CABG, 4 vessels; bypass used	CABG, 5 vessels; bypass used	CABG, 3 vessels; no bypass
8	CABG, 4 vessels; bypass used	CABG, 4 vessels; bypass used	CABG, 4 vessels; bypass used
9	Arthrodesis L4–S1; posterior instrumentation L4–S1	Arthrodesis C6–T5; posterior instrumentation C6–T5; posterior laminectomy and fusion	Decompression with bilateral foraminotomies, L2–S1; arthrodesis L2–S1
10	Decompressive fasciotomy, right leg; irrigation, debridement, reduction, repair ankle dislocation; closed reduction and application of external fixator, tibial plateau fracture	Closed reduction, open reduction, internal fixation lateral tibial plateau fracture	Open reduction internal fixation, right intertrochanteric hip fracture with plates and screws; open reduction, internal fixation right lateral malleolar fracture with plates and screws
11	Decompression and foraminotomy L2–L4; arthrodesis T10–L5; posterior instrumentation T10–L5	Decompression and bilateral foraminotomy L2–L4; arthrodesis L2–L5; posterior instrumentation L2–L5	Decompresssion and bilateral foraminotomy, L3–S1; arthrodesis L3–S1; posterior instrumentation L3–S1
12	Left posterolateral thoracotomy, superior segmentectomy	Left exploratory thoracotomy, wedge resection left upper lobe	Right lower lobe wedge resection, right lower lobectomy
13	CABG, 4 vessels; bypass used	CABG, 4 vessels; bypass used	CABG, 3 vessels; bypass used
14	CABG, 3 vessels; bypass used	CABG, 3 vessels; bypass used	CABG, 2 vessels; bypass used
15	Bilateral total knee replacement	Bilateral total knee replacement	Bilateral total knee replacement
16	Posterolateral fusion and internal fixation L4–L5; decompressive laminectomy, L4–L5	Posterolateral fusion and nonsegmental fixation, L4–L5; decompressive laminectomy, L3-L4	Decompressive laminectomy L4 and L5 with bilateral foraminotomies L4–S1
17	Exploration and resection of mycotic femoral artery aneurysm	Exploration and resection of infrarenal abdominal aortic aneurysm, cholecystectomy	Resection of infrarenal abdominal aortic aneurysm and left common iliac artery aneurysm

CABG = coronary artery bypass graft.

visual field loss in the affected eye. The diagnostic criteria for PION included an absence of optic disc swelling or atrophy when first examined, a relative afferent pupillary defect, demonstrable visual acuity or visual field loss, and no other objective evidence of ophthalmologic disease (retinal disease, vitreous hemorrhage, *etc.*) that would explain the loss of vision.

Optic discs with smaller cup-to-disc ratios are said to be "crowded," because the axons exit the eye through a smaller scleral opening. This type of crowded optic disc is frequently called a "disc-at-risk," because of the well-established association between this condition and spontaneous AION.[13] We defined the "disc-at-risk" configuration as a cup-to-disc ratio of 0.2 or less. As it is difficult to accurately determine the cup-to-disc ratio in the presence of disc swelling, the cup-to-disc ratio of the fellow (unaffected) eye was used for statistical analysis in patients with acute disc swelling from AION. In two patients both optic discs were swollen with acute AION. For these patients we recorded the cup-to-disc ratio as unknown.

With the assistance of the Data Architecture and Decision Support Department, we matched each of the 17 patients with 2 control patients who did not report vision loss after surgery. These 34 control patients were identified using a program called Clinical Practice Analysis that uses the extensible markup language platform. This program allows a systems analyst to define a set of parameters and then formulate those into a language called text query language. This language searches all of the dictated documents within our electronic medical record with the stated parameters defined in the text query language. For each of the 17 patients we identified a potential pool of control patients by identifying every other patient of the same sex who had the same type of surgical procedure at our institution during the study period. Each patient was then matched to the two control patients in that pool who were most similar regarding age and date of surgical procedure (table 2).

We reviewed the electronic and written medical records of each patient and control patient, including primary care physician exams before surgery, anesthesia records, postoperative nursing records, operative reports and notes, postoperative physician records, and ophthalmology notes. We collected data for multiple preoperative, intraoperative, and postoperative vari-

ables. The preoperative variables included age; sex; body mass index; mean arterial pressure; hemoglobin concentration; hematocrit; and the comorbidities hypertension, smoking, diabetes, hyperlipidemia, cerebral vascular accident, myocardial infarction, and renal disease. The intraoperative variables included the type of surgical procedure performed, duration of anesthesia and surgery, positioning, lowest mean arterial pressure of ≥ 5 min and the duration of this relative hypotension, lowest hemoglobin and hematocrit, duration of cardiac bypass, estimated blood loss, use of blood products, use of vasopressors, and temperature. Postoperative variables included the presence of facial edema, lowest recorded mean arterial pressure including the duration of this relative hypotension, lowest hemoglobin and hematocrit, and the use of blood products in the recovery period.

Two of our ION patients and one control patient had CABG surgery and then required a second emergency procedure the same day. For statistical analysis we chose one procedure and its postoperative data for analysis. Patient 14 had a CABG and then returned to the operating room several hours later to declot a saphenous vein graft. We used intraoperative and postoperative data from the CABG surgery. Patient 2 and a control patient for patient 4 returned to the operating room, 1 h and 5 h, respectively, after their initial CABGs for a repeat CABG. (Patient 2 had hypotension and electrocardiogram changes consistent with myocardial infarction; the control patient had an acute myocardial infarction after his initial surgery.) We used intraoperative and postoperative data from the second procedure.

Patients who have sustained ischemic damage to the optic nerve typically have reduced vision, a relative afferent pupillary defect (if the optic nerve damage is asymmetric), diminished color vision, and visual field defects. To ensure that our control patients had not sustained unrecognized optic nerve damage during their surgery, we attempted to contact each control patient to request that he or she be examined in our ophthalmology outpatient clinic. Twenty-six of the 34 control patients responded to a phone call or letter, four were deceased, and four did not respond for unknown reasons. Sixteen of the control patients agreed to have a complete eye exam by one of the authors in our outpatient clinic. After obtaining informed consent, we performed the following examinations on each of these control patients: Snellen visual acuity test, color vision test with Hardy-Rand-Rittler pseudoisochromatic color plates, pupillary exam, dilated funduscopic exam, and a Humphrey 24–2 SITA–FAST visual field test. These testing procedures are a reliable means of detecting optic nerve damage, even if subtle and asymptomatic.

We had medical record documentation of a postsurgical ophthalmologic exam for 4 of the 18 control patients who were not able or willing to be examined for various reasons (illness, distance needed to travel, or death). These exams included visual acuity, pupillary function, and a dilated fundus exam with documentation of cup-to-disc ratio. We had no postoperative ophthalmologic records for the 14 remaining control patients, but none of these individuals reported visual loss to their physicians after their surgical procedure.

Table 3. Incidence of Perioperative ION

Type of Surgery	AION Cases	PION Cases	Total ION
CABG (n = 2,749)	7 (0.25%)	2 (0.07%)	9 (0.33%)
Spine surgery (n = 1,110)	1 (0.09%)	3 (0.27%)	4 (0.36%)
Other surgery (n = 122,807)	—	4 (0.003%)	4 (0.003%)
Total (n = 126,666)	8 (0.006%)	9 (0.007%)	17 (0.013%)

AION = anterior ischemic optic neuropathy; CABG = coronary artery bypass graft; ION = ischemic optic neuropathy; PION = posterior ischemic optic neuropathy.

Statistical Analysis

All demographic variables and possible risk factors were summarized by group (patient *vs.* control) using descriptive statistics: mean and SD for continuous variables and the frequency (percentage) for categorical variables. We used the conditional logistic regression procedure to compare the patient and control groups in each variable to take into account matching in comparison. *P* value, odds ratio, and 95% confidence interval were presented for each comparison. A *P* value of less than Bonferroni adjusted α (0.0025 = 0.05/number of possible risk factors) indicated a statistical significance. We used SAS 9.1.3 software (SAS Institute, Inc., Cary, NC) for statistical analysis.

Results

There were 126,666 surgical procedures performed at our institution between January 1998 and December 2004. During this time period, there were 17 documented cases of perioperative ION, an overall incidence of 0.013% (table 3). The incidence of perioperative ION after CABG surgery at our institution during this period was 0.33% (9 patients with ION from a total of 2,749 CABG surgeries). The incidence after spine surgery was 0.36% (4 patients with ION from a total of 1,110 spine surgeries). The incidence of perioperative ION for all procedures other than CABG and spine surgery was 0.003%. Of the 9 patients with ION after CABG, 7 patients (78%) were diagnosed with AION. Three of the 4 patients (75%) with ION after spine surgery had PION. In our series of 17 patients with perioperative ION, 16 were men (94%).

We analyzed all ION cases together (table 4), and then separately analyzed the AION (see table, Supplemental

Table 4. All ION Patients and Controls

Preoperative Variables	Patients (n = 17)	Controls (n = 34)	*P* Value*	Odds Ratio	95% CI	
Age	65.8 ± 8.6	64.4 ± 6.7	N/A	N/A	—	—
Body mass index†	30.6 ± 5.5	29.8 ± 6.3	0.6088	1.028	0.925	1.142
Medical history						
Diabetes	4 (23.5)	7 (20.6)	0.8138	1.179	0.300	4.631
Smoking‡	11 (64.7)	20 (58.8)	0.6556	1.356	0.356	5.171
Hypertension	14 (82.4)	19 (55.9)	0.0791	3.386	0.868	13.208
Coronary artery disease	11 (64.7)	20 (58.8)	0.4292	2.732	0.226	33.003
Myocardial infarction	4 (23.5)	9 (26.5)	0.8232	0.859	0.227	3.251
Stroke	1 (5.9)	5 (14.7)	0.3106	0.295	0.028	3.120
Renal disease	0 (0.0)	0 (0.0)	N/A	N/A	—	—
Hyperlipidemia	10 (58.8)	17 (50.0)	0.5419	1.459	0.433	4.917
Mean arterial pressure§	97.5 ± 8.5	100.4 ± 8.8	0.2705	0.959	0.891	1.033
Hematocrit	41.7 ± 4.3	42.2 ± 3.4	0.6248	0.960	0.814	1.132
Disc-at-risk	9/14 (60.0)	10/20 (50.0)	0.4243	1.794	0.428	7.520
Disc-at-risk with missing imputed as "yes"	12 (70.6)	24 (70.6)	1.0000	1.000	0.301	3.321
Disc-at-risk with missing imputed as "no"	9 (52.9)	10 (29.4)	0.1270	2.611	0.761	8.956
Intraoperative variables						
Mean arterial pressure						
Lowest	56.4 ± 11.1	55.3 ± 14.3	0.7717	1.008	0.957	1.062
Change from preop	−41.1 ± 13.8	−44.8 ± 15.7	0.3212	1.027	0.975	1.082
% Change from preop	−41.8 ± 12.4	−44.5 ± 14.3	0.4203	1.023	0.967	1.083
Duration (in min)	15.9 ± 28.4	7.0 ± 4.7	0.1919	1.070	0.967	1.184
Hematocrit	25.8 ± 4.8	23.4 ± 2.9	0.3223	—	—	—
Lowest	−18.3 ± 4.2	−17.2 ± 3.7	0.3063	1.164	0.861	1.574
Change from preop	−41.6 ± 9.5	−42.1 ± 7.6	0.7043	0.895	0.723	1.107
% Change from preop	—	—	—	0.982	0.894	1.078
Blood products administered	6 (35.3)	9 (26.5)	0.4285	1.877	0.395	8.922
Blood loss amount	880.0 ± 882.6	800.0 ± 979.7	0.5946	1.000	0.999	1.001
Lowest temperature	30.8 ± 2.4	30.0 ± 2.0	0.4431	1.161	0.793	1.701
Surgery time (in h)	4.8 ± 2.7	4.3 ± 2.1	0.3055	1.217	0.836	1.770
Vasopressors	14 (82.35%)	24 (70.59%)	0.2968	2.500	0.447	13.982
Postoperative variables						
Mean arterial pressure						
Lowest	62.1 ± 14.0	69.8 ± 11.1	0.0723	0.950	0.898	1.005
Change from preop	−35.3 ± 17.7	−30.6 ± 13.6	0.3179	0.980	0.943	1.019
% Change from preop	−35.6 ± 15.8	−30.0 ± 12.2	0.2068	0.971	0.928	1.016
Duration (in min)	27.6 ± 26.8	26.2 ± 40.0	0.8942	1.001	0.986	1.016
Hematocrit						
Lowest	25.9 ± 4.0	28.6 ± 5.2	0.1061	0.872	0.739	1.030
Change from preop	−15.7 ± 6.1	−13.4 ± 5.2	0.3005	0.940	0.835	1.057
% Change from preop	−37.0 ± 12.0	−31.8 ± 11.5	0.2532	0.966	0.912	1.025
Blood products administered	10 (58.8)	15 (44.1)	0.3315	1.793	0.552	5.822

* Conditional logistic regression was used. † Body Mass Index = [weight in pounds/(height in inches)^2] × 703. ‡ Current or any prior history of smoking. § Mean arterial pressure = [(systolic blood pressure – diastolic blood pressure)/3 + diastolic blood pressure] (mmHg).

ION = ischemic optic neuropathy.

Digital Content 1, which shows AION cases *vs.* controls, http://links.lww.com/A674), PION (see table, Supplemental Digital Content 2, which shows PION cases *vs.* controls, http://links.lww.com/A675), and CABG subgroups (see table, Supplemental Digital Content 3, which shows CABG cases *vs.* controls, http://links.lww.com/A676). None of the preoperative, intraoperative, or postoperative variables we examined for these subgroups reached statistical significance. None of the 22 control patients who had documented postoperative ophthalmologic examinations had evidence of prior ION.

Discussion

Ischemic optic neuropathy is the most commonly reported cause of perioperative vision loss.[1,2] Our study is the first large series specifically designed to calculate the incidence of symptomatic perioperative ION after nonocular surgery at a single institution over an extended period of time. Perioperative ION is a rare event, as evidenced by the incidence of 0.013% in our series of 126,666 surgical procedures over a 7-yr period. Our study design allowed us to identify ION in patients who reported visual complaints in the perioperative period. We did not have a complete neuro-ophthalmologic examination of all 126,666 patients who had surgery during this period. Therefore, our calculated incidence of 0.013% is actually the incidence of patients with perioperative ION who reported visual loss in the perioperative period. There may have been patients with less significant visual loss who simply failed to notice the change in

their vision, or patients who failed to notify their doctor of their visual symptoms. Similarly, patients who were critically ill after surgery may have had vision loss that was overshadowed by more life-threatening issues and thus went unrecognized. For these reasons, the true incidence of perioperative ION may actually be greater than 0.013%.

Our 0.33% incidence of ION after CABG surgery is higher than the 0.06% incidence reported by Nuttall *et al.*,[5] but lower than the 1.3% incidence of AION after CABG reported by Shapira*et al.*[4] The 0.36% incidence after spine surgery is also higher than the 0.028% and 0.12% incidences previously reported.[3,14] We believe our incidence is higher than has been reported in most other studies because our system of consults and referrals that funnels all patients with perioperative ION to one faculty neuro-ophthalmologist produces a very high capture rate for these events. This would conceivably enhance our ability to collect and analyze data related to perioperative ION.

Our series documented a higher incidence of perioperative ION after spinal surgery (0.36%) and CABG (0.33%) than any other type of surgical procedure (table 3). The risk of perioperative ION appears to be about 100 times greater for CABG or spinal surgery than for all other major surgical procedures combined. Patients in our study who experienced ION after CABG were more likely to have AION (7 of 9 patients, 78%). All of our AION patients, except one, were associated with CABG surgery. This preponderance of AION cases after CABG is consistent with other reports.[4,5] Patients with ION associated with other surgical procedures were more likely to have PION (7 of 9 patients, 78%). Three of the 4 patients (75%) with ION after spine surgery had PION. This tendency for PION, rather than AION, to occur more commonly after spine surgery is consistent with the results of other series.[6,14]

AION results from ischemia to the anterior portion of the optic nerve that receives its vascular supply from the short posterior ciliary arteries and peripupillary choroid. Affected patients present with a wide range of visual acuities and visual field defects. The optic disc is initially swollen, but the swelling gradually evolves into visible optic atrophy over the ensuing month. Spontaneous AION in the nonsurgical setting is classified as either arteritic or nonarteritic. The arteritic form generally occurs in patients over 55 yr of age and is caused by a systemic vasculitis, most commonly giant cell arteritis. In contrast, nonarteritic AION is a multifactorial disease that affects middle-aged patients who have a morphologically small or crowded optic nerve head, the so-called "disc-at-risk" configuration.[13] It is thought that other risk factors, in addition to an underlying "disc-at-risk," create an environment for ischemic damage to the optic nerve. Diabetes, high cholesterol, smoking,[11,12,15] physiologic nocturnal arterial hypotension,[16] an inability to properly autoregulate optic nerve head blood flow,[17] and interindividual variations in the vascular supply of the optic nerve head all may play a role in the ultimate occurrence of spontaneous nonarteritic AION.[18]

We were unable to identify any preoperative, intraoperative, or postoperative variables that increase the risk of perioperative AION. However, all six of the AION patients for whom we could assess the cup-to-disc ratio in the fellow eye had a "disc-at-risk" configuration. We suspect that the other two patients with bilateral AION also had a preexisting "disc-at-risk" configuration, but the presence of this configuration cannot be determined in an acutely swollen optic disc. We know the cup-to-disc ratio for 10 of the AION control subjects, and only 4 of these had a "disc-at-risk" configuration. This is consistent with the reported incidence of "disc-at-risk" in the general population of 15 to 65%.[13] The presence of "disc-at-risk" did not attain statistical significance in our study. In light of the well-documented association between the "disc-at-risk" configuration and spontaneous AION, we believe that this association in perioperative AION would be statistically significant in a larger, more powerful study.

PION is the result of ischemia to the posterior portion of the optic nerve that receives its blood supply from the pial capillary plexus. Patients experience sudden painless vision loss or visual field defects. The optic disc initially appears normal, but optic atrophy invariably ensues. PION is not associated with a "disc-at-risk" configuration. Spontaneous PION is distinctly less common than spontaneous AION. Like AION, there is an arteritic form of PION secondary to systemic vasculitic disease, most commonly giant cell arteritis.[19] Rarely reported nonarteritic causes of spontaneous PION include migraine,[20] hemodialysis,[21] spontaneous carotid artery dissection,[22] and *Aspergillus* infection.[23] However, PION from any cause other than giant cell arteritis or trauma with sustained hypotension is exceedingly rare, except in the perioperative setting.

Perioperative PION is reported after spine surgery,[24–27] orthopedic surgery,[28] neck surgery,[29–33] heart surgery,[5] and abdominal surgery.[34,35] Intraoperative variables that are reported to play a role in the pathogenesis of PION include hypotension, anemia, and elevated intraocular pressure with prone positioning during spinal surgery.[26,36] Many patients also have coexistent vascular risk factors such as diabetes, coronary artery disease, and hypertension.[24] Vision loss has also been reported in children and healthy adults who had none of these risk factors.[26,37]

In our analysis of perioperative PION, we found no significant difference in any of the preoperative, intraoperative, or postoperative variables we evaluated when the PION patients were compared with their matched control patients. Based on other case reports in the literature, we expected to find that patients who lost

vision from perioperative PION would have more intraoperative or postoperative hemodynamic problems than the control patients, who had the same operations but did not lose vision. Interestingly, we found no significant difference in blood pressure values or hematocrit levels in the two groups. In addition, intraoperative vasopressor use was similar between the patients and control groups.

One significant strength of our study that differentiates it from most other reported studies of perioperative ION is that each of our patients was matched with two control patients of similar age who had the same operation at about the same time in the same institution. With this matching process we attempted to eliminate the confounding effect of these factors and more accurately assess the effects of the studied hemodynamic variables. Our results should be interpreted with the understanding that a study with only 17 patients and 34 control patients is significantly underpowered to detect a small difference between patients and controls. Because perioperative ION is so uncommon, finding large numbers of patients for evaluation would require a much longer period of time or a multicenter study. The fact that ours is not a multicenter study, however, is one of the strengths of this study. We are more confident in the validity of our reported incidence of this complication because all data were collected from a single institution, and all of the patients were examined by a single neuro-ophthalmologist.

It is interesting to note that 16 of the 17 patients (94%) with perioperative ION in our series were men. A higher incidence of perioperative ION among men is reported in some patient series, but the marked preponderance of men in our series is greater than in any previous reports.[1,4,5] The reason for this sex-related disparity is unknown and may simply be a statistical anomaly, but the disparity may be a function of differences in anatomy, preexisting comorbidities, or vascular physiology.

In conclusion, our study demonstrates that perioperative ION is an uncommon complication of nonocular surgical procedures, with an overall incidence of 0.013%. Given that the mechanisms and risk factors for ION are poorly understood, the risks of vision loss should be considered in the preoperative discussion with a patient who is anticipating a surgical procedure where this rare complication seems to have the highest frequency: Spine surgery and surgery requiring cardiopulmonary bypass. We found no evidence that the management of our patients who had perioperative ION, at least regarding the hemodynamic variables we evaluated, deviated from the perioperative course of similar patients undergoing similar procedures at our institution who did not lose vision. We conclude that perioperative ION can occur in the absence of any unusual or atypical fluctuations in these hemodynamic variables during the perioperative period. We recognize the limited power of this study and that larger, more powerful studies are required to better understand the pathogenesis of this condition.

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