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Ischemic Optic Neuropathy Following Spine Surgery

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Abstract

Perioperative visual loss (POVL) is a devastating injury that has been reported infrequently after nonocular surgery. The most common cause of POVL is ischemic optic neuropathy (ION). Increasing numbers of cases of ION are being reported after spine surgery, but the etiology of postoperative ION remains poorly understood. After a MEDLINE search of the literature, we reviewed published case reports of ION, specifically those reported after spine surgery performed with the patient in the prone position. Most of the cases involved posterior ION (PION, n = 17), and the remainder anterior (AION, n = 5). Most patients had no or few preoperative vascular disease risk factors. All except one PION and 2 of 5 AION cases reported symptom onset within the first 24 hours after surgery. Visual loss was frequently bilateral (40% of AION, 47% of PION cases). Mean operative time exceeded 450 minutes. The lowest average intraoperative mean arterial blood pressure was 64 mm Hg and the mean lowest intraoperative hematocrit was 27%. The average blood loss was 1.7 L for AION and 5 L for PION patients. PION patients received an average of 8 L of crystalloid solution and 2.2 L of colloid intraoperatively. This compilation of case reports suggests that a combination of prolonged surgery in the prone position, decreased ocular perfusion pressure, blood loss and anemia/hemodilution, and infusion of large quantities of intravenous fluids are some of the potential factors involved in the etiology of postoperative ION. However, levels of blood pressure and anemia intraoperatively were frequently at levels considered acceptable in anesthesia practice. The etiology of postoperative ION remains incompletely understood. Potential strategies to avoid this complication are discussed.

Keywords

ischemic optic neuropathy; optic nerve; spine surgery; visual loss

Visual loss after nonocular surgery is rare, but it is among the most devastating injuries that a patient may sustain.[1] The most frequently reported conditions associated with postoperative visual loss (POVL) are ischemic optic neuropathy (ION) and central retinal arterial occlusion.[2,3] The growing concern about POVL has led to establishment of the ASA Postoperative Visual Loss Registry. Spine surgery patients are the single largest group in the Registry to date[4]; in most of these cases, the diagnosis has been ION. This disturbing trend is of great concern,

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especially to neuroanesthesiologists, because of the rapidly increasing number and complexity of spine operations in the United States.[5]

Two large retrospective studies have determined that the incidence of ION is approximately 1/60,000 to 1/125,000 of all anesthetics.[6,7] The incidence of ION after spine surgery appears to be greater but is difficult to define at present with accuracy because the numbers of patients studied are small. In two retrospective reviews, 4 cases of ION were found in 3450 spine surgeries (0.1%),[8] and we reported 2 cases of ION in 3300 patients after spine surgery (0.06%).[3] A number of factors have been proposed to explain postoperative ION. These include decreased systemic blood pressure, blood loss, anemia or hemodilution, increased intraocular or orbital venous pressure, abnormal autoregulation in the optic nerve (ON) circulation and/or anatomic variation in the blood supply to the ON, the use of vasopressors, the presence of systemic disease such as hypertension, diabetes, and atherosclerosis, and retrobulbar hemorrhage.[9] It seems that one or more of these factors are often involved in an individual patient and in an unpredictable fashion. Although case reports have suggested that ION is due to hypotension and anemia, cases occur without these factors and occur even in patients whose blood pressures and hematocrits (Hct) are in a range traditionally considered by anesthesiologists to be acceptable.[3,4,9,10]

ION generally presents with painless visual loss, visual field deficits, and sluggish pupils. When visual loss is asymmetric or unilateral, a relative afferent papillary defect may be present.[11] Anterior ION (AION) is diagnosed when a swollen optic nerve head is seen upon funduscopic examination at the time of symptom onset.[12] In contrast, the optic disc is normal at the time of initial presentation in patients with posterior ION (PION) because the affected area cannot be visualized funduscopically.[13] Since ION is rare and the number of cases is low at any one institution, there are few clinical studies of this entity, and most current knowledge is derived from case reports of isolated single cases or small series. No compilation of ION cases has been performed to assist in determining characteristics of patients who have developed ION after spine surgery. Large studies have described the characteristics of patients who spontaneously developed AION (ie, not after a surgical procedure),[14] but far fewer descriptions of PION, either spontaneous or postoperative, are available. We reviewed case reports of ION in the literature, specifically in spine surgery patients, in an attempt to synthesize the findings and suggest areas for further study.

METHODS

A MEDLINE search was performed for case reports and observational studies on ION with an English abstract using the terms ischemic optic neuropathy, visual loss, blindness, and spine surgery. Our review excluded the papers in which patient data were presented in summary fashion only.[15,16]

Patient demographics, perioperative factors, postoperative course, and outcome were obtained if available. The data were organized into several main categories: type of ION, age and gender, surgery type, perioperative events, possible risk factors and stratification of these factors, postoperative course including time of symptom onset, ocular findings, interventional measures, and outcome. Since baseline risk factors for ION have not been defined, we used the risk stratification designed by Sadda et al for PION.[13] These authors stratified risk based on the number of the following baseline risk factors: hypertension, diabetes mellitus, smoking, hypercholesterolemia, and “cardiac” and “cerebrovascular” history. Cardiac factors encompassed coronary artery disease, congestive heart failure, or arrhythmia. Cerebrovascular factors included history of carotid artery disease, stroke, transient ischemic events, and small vessel disease.

Because of the nature of observational studies, available patient data varied and, therefore, the results are limited to the extent that information could be extracted from case reports. In Table 1, the number of cases from which the averages were calculated was included. This consideration pertains particularly to perioperative events, including calculation of mean arterial pressure (MAP), hemoglobin (Hgb), Hct, and blood loss and volumes transfused, for which reporting of values was not standardized.

RESULTS

Cases were divided into AION or PION, and are summarized in Tables 1 and 2. Five cases of AION from 3 distinct reports and 17 cases of PION from 10 distinct reports were analyzed (see Table 1 for references). The median ages for patients who experienced AION and PION were 53 and 43 years, respectively. All of the patients with AION underwent lumbar spine fusion. Among PION cases, 82% followed lumbar spine fusion, with the remainder following cervical or thoracic. Four patients in the PION group were positioned on a Wilson frame, one on a Toronto frame; for the other 12 patients, no positioning device was described. A Relton-Hall frame was used on one of the AION cases, but no information was provided for the 4 others.

Mean operative time was 522 minutes for AION (5 patients) and 456 minutes (13 patients). The lowest average MAP was 66 (AION) and 64 mm Hg (PION). For AION, the range of lowest MAP was 62 to 78 mm Hg; for PION patients, it was 52 to 85 mm Hg. In 1 AION case, MAP was 78, and systolic blood pressure was reported at 107 mm Hg in another. In two of the case reports of PION, intraoperative MAP was maintained at >75 mm Hg; and in a third case, the authors stated that there was no hypotension. For AION, lowest postoperative mean Hgb was 9.5 g/dL (4 patients; data for intraoperative Hgb were only available for 1 patient). Hct values were reported in 11 PION patients, with a mean lowest Hct of 27% intraoperatively and 29% postoperatively, the latter representing a 34% decrease from the preoperative baseline value. Intraoperative Hct ranged from 21% to 39%. Three patients in the PION series had intraoperative Hct exceeding 30%. The average blood loss reported was 1.7 L (range 0.5–3 L) and 5 L (range 0.8–16 L) for AION and PION cases, respectively. The average volume of crystalloid/colloid solutions administered in the operating room was 6.6/0.8 L (4 patients) and 8.0/2.2 L (9 patients), for AION and PION, respectively.

Sixty percent of the AION patients had been diagnosed preoperatively with diabetes mellitus compared with 27% of PION patients. Hypertension was present in 40% of AION and 53% of PION cases. Coronary artery disease was present in 1 of 5 AION patients and not reported in any of the PION cases. Risk stratification using criteria of Sadda et al[13] showed that the majority of AION cases had only one risk factor, and almost half of the PION cases had no risk factors.

Symptoms were reported within 24 hours after surgery in 40% of AION cases; 59% of the PION cases reported complaints immediately upon awakening from surgery and 88% were symptomatic within 24 hours. When symptoms were reported later, it is not possible to determine if it was symptom onset or diagnosis that was delayed. All of the PION cases, by definition, had normal fundus examination. None of the cases reported a cherry-red macula, which would have indicated retinal vascular occlusion. Altitudinal defects were reported in 27% of PION patients, with another 6% reporting a central scotoma.

Overall, the visual acuity improved somewhat in 60% of AION and 65% of PION cases. One PION patient worsened over the first 2 to 3 days postoperatively. Twenty percent of AION cases (1 patient) received high-dose steroids, whereas 29% of PION cases (5 patients) received either high-dose steroids or had a drug-induced increase in blood pressure. The 1 case of AION

and 4 of 5 cases of PION with intervention reported an improvement at final follow-up. Moreover, 33% of AION cases and 58% of PION cases without intervention had improvement.

DISCUSSION

In 1995, Williams et al alerted anesthesiologists about the current state of knowledge of postoperative ION.[2] That report referenced only two case reports of ION after spine surgery. However, it is apparent from our review of more recent case reports that ION is being increasingly noted after spine surgery, especially complex procedures including fusion and/or instrumentation. Moreover, cases after spine surgery currently constitute the single largest group (67%) of patients in the POVL Registry.[4] As a result of medical-legal and other considerations, it is likely that many cases are not reported in the literature. However, retrospective examination of case reports and review of existent case series provide the only current sources of data. Larger and focused case-control studies and prospective examination of risk factors will ultimately better define factors responsible for this devastating complication. Our compilation of patient data from case reports of ION after spine surgery provides a basis for these future studies by identifying potential factors for study.

Our review classified cases as AION or PION, and the majority of cases after spine surgery involved PION. Spontaneous development of AION is the most frequent cause of acute optic neuropathy in patients older than 50 years.[12] The etiology of this disorder remains incompletely understood. Hayreh postulated the existence of watershed zones and abnormal autoregulation in the optic nerve head that may render some patients more susceptible.[11,17] In addition, the presence of a small optic cup-to-disc ratio, indicating a smaller canal from which the optic nerve exits the sclera, is also a significant risk factor.[18] While studies have suggested that patients with vascular risk factors such as diabetes and hypertension are more likely to spontaneously develop ION, others have shown contradictory findings.[14,19-21] In postoperative patients, many cases of AION have been reported after open heart surgery, but there have also been cases after a wide variety of other procedures.[2,9]

One difficulty in dividing cases into AION and PION is the diagnostic confusion in some of the cases. Upon funduscopic examination, edema that spreads anteriorly several days after onset of PION could be mistaken for AION. In previous reviews, this distinction has not been made. However, although the number of cases is low, we think that until better understanding of perioperative ION is obtained from clinical studies, it is appropriate to divide cases into AION and PION, as the anterior and posterior optic nerve differ anatomically, and accordingly, these two entities may well differ pathophysiologically. Indeed, neuro-ophthalmologists, thinking that these are two distinct entities, continue to classify spontaneously developing ION into AION or PION.[13]

For this review, we classified cases as AION or PION depending upon either: 1) documentation as such in the case reports or 2) AION was defined as optic nerve edema at the time of symptom onset, and PION as no optic nerve edema when symptoms were first reported. The presence of a cherry-red spot would exclude patients from AION or PION as this would be a sign diagnostic of a central retinal artery occlusion. AION must have documented disc edema at the time of symptom onset. This edema can also precede visual loss and it persists for several weeks after onset. PION has a normal disc in the acute period. In both diseases pallor eventually develops after several months.

There were some differences between the two groups of patients with AION or PION, although the small numbers of AION patients render the comparisons difficult to interpret. Blood loss tended to be greater in the PION cases. Symptom onset was within 24 hours in nearly all PION cases, but AION did occur beyond the first 24 hours in 60% of the cases. A history of

hypertension was present in about one half of AION or PION patients. Diabetes was found in only 25% of PION cases and in 3 of 5 AION patients.

The increasing number of reports of ION after spine surgery raises the obvious question of what factors specific to spine surgery might be associated with ION. The cases reported tended to be long operative procedures in the prone position, involving hypotension, hemodilution or anemia, blood loss, and infusion of large amounts of intravenous fluids. Intraoperative MAP averaged 65 or 66 mm Hg in the case reports, compared with baseline MAP in the mid 90s. The baseline MAPs are less reliable because they were generally not indicated in the reports. Intraoperative Hct averaged 29% compared with baseline of 42% in the PION patients. Comparison with AION patients is not possible because Hct and Hgb were generally not reported. The decrease in MAP and in Hgb/Hct is consistent with the practice in many centers of mild to moderate deliberate hypotension as well as mild to moderate hemodilution to decrease blood loss and blood usage in major spine surgery; both have been speculated to be associated with ION through as yet unknown mechanisms. In this review, not every patient sustained hypotension or anemia in the intraoperative or postoperative period, and levels of hypotension would generally be considered moderate. This suggests that other factors may be involved in the development of ION. We also noted that patients had significant intraoperative blood loss averaging 1.7 and 5 L for AION and PION patients, respectively. This finding is consistent with the one case-control study to date on postoperative visual loss after spine surgery. Although this study also included patients with visual loss due to retinal vascular occlusion, ION patients predominated, and the ION subgroup was reported to have no differences in intraoperative blood pressure from controls. Patients with postoperative visual loss had significantly greater intraoperative blood loss compared with unaffected patients, and no differences in lowest intraoperative Hct or systemic blood pressure.[16]

Large amounts of crystalloid and colloid solutions were infused in both the AION and PION cases, a factor that, to date, has received little attention in the literature.[22] In the prone position, fluid may collect in the regions surrounding the optic nerve, and over time, large fluid infusions may increase venous pressure and decrease perfusion pressure. Combined with other factors such as decreased blood pressure, ischemic injury of the optic nerve may result. This theory has, however, not yet been studied. During surgery in the prone position, it has been shown that intraocular pressure (IOP) increases over time.[23] The relationship between increased IOP and ION in the perioperative period remains obscure; the increase may simply be a surrogate marker. However, it has been suggested that increased IOP reflects, at least in part, the increased venous pressure within the eye, perhaps resulting from perioperative infusion of large quantities of fluid.[24]

Our review of cases did not address the use of various frames to position the patients for surgery, or the use of any specific headrests, because in most cases, details of positioning were missing. Positioning the head below the level of the body could also increase IOP and enhance fluid collection around the optic nerve. When a patient is positioned on a Wilson frame, the head tends to be lower than the body, especially when the frame is elevated to maximal or near-maximal height. Also, compression of the abdomen, particularly in obese patients positioned in some types of frames, potentially could increase venous pressure further. We are unable to assess the impact of obesity since body weights were generally not reported.

This retrospective review of case reports of ION after spine surgery cannot determine the cause (s) of the visual loss. There is no comparison group of patients undergoing the same surgery who did not sustain ION. Long surgery, hypotension, hemodilution, and administration of large amounts of intravenous fluids are common occurrences during spine surgery. In addition, our conclusions are limited by the accuracy of the information reported by the authors of the case reports. In some instances, values for blood pressure and Hgb/Hct were not provided. Only

one case report included the duration of hypotension and hemodilution. The length of time that these alterations are present remains an unknown influence upon the occurrence of perioperative ION.

In summary, review of recent case reports of ION after spine surgery suggests that both AION and PION, often bilateral, continue to occur in patients with few vascular risk factors. In most cases, especially with PION, visual loss almost always is apparent within the first 24 hours after surgery. The length of surgery, levels of hypotension and hemodilution, as well as quantities of fluid and blood administered generally fell into a range typically encountered by anesthesiologists caring for these patients. While the necessity to maintain perfusion pressure in the optic nerve is self-evident, whether deliberate hypotension should be used in spine surgery remains controversial. On the basis of these reports, we recommend that anesthesiologists establish a protocol for management of blood pressure and blood and fluid replacement in consultation with their spine surgeons, and decide whether indications for deliberate hypotension outweigh the potential risks. In addition, positions that cause the head to be below the level of the body or that compress the abdomen or chest should be avoided. While not the decision of the anesthesiologist alone, staging of long complex procedures, eg, anterior-posterior spinal fusions, seems a reasonable alternative to decrease blood loss and fluid requirements.

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REFERENCES

1. Lauer KK. Visual loss after spine surgery. J Neurosurg Anesthesiol 2004;16:77–79. [PubMed: 14676574]
2. Williams EL, Hart WM Jr, Tempelhoff R. Postoperative ischemic optic neuropathy. Anesth Analg 1995;80:1018–1029. [PubMed: 7726399]
3. Roth S, Barach P. Post-operative visual loss: still no answers yet. Anesthesiology 2001;95:575–576. [PubMed: 11575526]
4. Lee LA. ASA Postoperative Visual Loss Registry: preliminary analysis of factors associated with spine operations. ASA Newsl 2003;67:7–8.
5. Deyo RA, Nachemson A, Mirza SK. Spinal-fusion surgery: the case for restraint. N Engl J Med 2004;350:722–726. [PubMed: 14960750]
6. Warner ME, Warner MA, Garrity JA, et al. The frequency of perioperative vision loss. Anesth Analg 2001;93:1417–1421. [PubMed: 11726416]
7. Roth S, Thisted RA, Erickson JP, et al. Eye injuries after non-ocular surgery: a study of 60, 965 anesthetics from 1988-1992. Anesthesiology 1996;85:1020–1027. [PubMed: 8916818]
8. Stevens WR, Glazer PA, Kelley SD, et al. Ophthalmic complications after spinal surgery. Spine 1997;22:1319–1324. [PubMed: 9201834]
9. Roth, S.; Gillesberg, I. Injuries to the visual system and other sense organs. In: Benumof, JL.; Saidman, LJ., editors. Anesthesia and Perioperative Complications. Vol. 2nd. Mosby; St. Louis: p. 1999-377.
10. Lee LA. Postoperative visual loss data gathered and analyzed. ASA Newsl 2000;64:25–27.
11. Hayreh SS. The 1994 Von Sallman Lecture: the optic nerve head circulation in health and disease. Exp Eye Res 1995;61:259–272. [PubMed: 7556490]
12. Kelman, SE. Ischemic optic neuropathies. In: Miller, NR.; Newman, NJ., editors. Walsh and Hoyt's Clinical Neuro-ophthalmology. Vol. 5th. Williams & Wilkins; Philadelphia: 2000. p. 549-594.

13. Sadda SR, Nee M, Miller NR, et al. Clinical spectrum of posterior ischemic optic neuropathy. Am J Ophthalmol 2001;132:743–750. [PubMed: 11704036]

14. Anonymous. The Ischemic Optic Neuropathy Decompression Trial Research Group: optic nerve decompression surgery for non-arteritic anterior ischemic optic neuropathy is not effective and may be harmful. JAMA 1995;273:625–632. [PubMed: 7844872]

15. Cheng MA, Sigurdson W, Tempelhoff R, et al. Visual loss after spine surgery: a survey. Neurosurgery 2000;46:625–631. [PubMed: 10719859]

16. Myers MA, Hamilton SR, Bogosian AJ, et al. Visual loss as a complication of spine surgery: a review of 37 cases. Spine 1997;22:1325–1329. [PubMed: 9201835]

17. Hayreh, SS. Current Concepts in Ocular Blood Flow in Glaucoma. Pillunat, LE.; Harris, A.; Anderson, DR., et al., editors. Kugler; The Hague, Netherlands: 1999. p. 3-31.

18. Danesh-Meyer HV, Savino PJ, Sergott RC. The prevalence of cupping in end-stage arteritic and nonarteritic anterior ischemic optic neuropathy. Ophthalmology 2001;108:593–598. [PubMed: 11237915]

19. Anonymous. Characteristics of patients with nonarteritic ischemic optic neuropathy eligible for the Ischemic Optic Neuropathy Decompression Trial. Arch Ophthalmol 1996;114:1366–1374. [PubMed: 8906027]

20. Jacobson DM, Vierkant RA, Belongia EA. Nonarteritic anterior ischemic optic neuropathy: a case-control study of potential risk factors. Arch Ophthalmol 1997;115:1403–1407. [PubMed: 9366670]

21. Hayreh SS, Joos KM, Podhajsky PA, et al. Systemic diseases associated with nonarteritic anterior ischemic optic neuropathy. Am J Ophthalmol 1994;118:766–780. [PubMed: 7977604]

22. Dilger JA, Tetzlaff JE, Bell GR, et al. Ischaemic optic neuropathy after spinal fusion. Can J Anaesth 1998;45:63–66. [PubMed: 9466031]

23. Cheng MA, Todorov A, Tempelhoff R, et al. The effect of prone positioning on intraocular pressure in anesthetized patients. Anesthesiology 2001;95:1351–1355. [PubMed: 11748391]

24. Lee LA, Lam AM, Roth S. Causes of elevated intraocular pressure during prone spine surgery [letter]. Anesthesiology 2002;97:759. [PubMed: 12218559]

25. Alexandrakis G, Lam BL. Bilateral posterior ischemic optic neuropathy after spinal surgery. Am J Ophthalmol 1999;127:354–355. [PubMed: 10088754]

26. Brown RH, Schauble JF, Miller NR. Anemia and hypotension as contributors to perioperative loss of vision. Anesthesiology 1994;80:222–226. [PubMed: 8291715]

27. Dunker S, Hsu HY, Sebag J, et al. Perioperative risk factors for ischemic optic neuropathy. J Am Coll Surg 2002;194:705–710. [PubMed: 12081060]

28. Katz DM, Trobe JD, Cornblath WT, et al. Ischemic optic neuropathy after lumbar spine surgery. Arch Ophthalmol 1994;112:925–931. [PubMed: 8031272]

29. Lee LA, Lam AM. Unilateral blindness after prone lumbar surgery. Anesthesiology 2001;95:793–795. [PubMed: 11575556]

30. Lee AG. Ischemic optic neuropathy following lumbar spine surgery. J Neurosurg 1995;83:348–349. [PubMed: 7616283]

31. Roth S, Nunez R, Schreider BD. Visual loss after lumbar spine fusion. J Neurosurg Anesthesiol 1997;9:346–348. [PubMed: 9339408]

32. Murphy MA. Bilateral posterior ischemic optic neuropathy after lumbar spine surgery. Ophthalmology 2003;110:1454–1457. [PubMed: 12867409]

33. Abraham M, Sakhuja N, Sinha S, et al. Unilateral visual loss after cervical spine surgery. J Neurosurg Anesthesiol 2003;15:319–322. [PubMed: 14508173]

TABLE 1

Patient Characteristics

	AION (5)		PION (17)	
No. of cases and Reference	**Dilger 1998 Katz 1994 (3 patients) Stevens 1997**		**Abraham 2003 Alexandrakis 1999 Brown 1994 Dunker 2002 (5 patients) Katz 1994 Lee 2001Lee 1995 Murphy 2003 Roth 1997 (2 patients) Stevens 1997 (3 patients)**	
Patient demographics				
% male	40		71	
Median age (yr)	53		43	
Age range (yr)	41–65		12–79	
Surgery location				
Cervical	0%		6%	
Thoracic	0%		24%	
Lumbar	100%		82%	
	Mean	**Range**	**Mean**	**Range**
Perioperative events				
Operative time (min)	522 (5)	360–720	456 (13)	180–600
Preop mean arterial pressure (mm Hg)	91 (1)		95 (6)	73–106
Intraop lowest mean arterial pressure (mm Hg)	66 (2)	62–78	64 (6)	52–85
Preop hemoglobin	15 (4)	14–16.4	13 (4)	9.9–15.6
Postop lowest hemoglobin	9.5 (4)	8–11.7	11 (4)	10.6–12
% change hemoglobin	−39 (4)	−24–48	−22 (3)	−17–27
Intraop lowest hemotocrit (%)	32 (1)		27 (10)	21–39
Preop hematocrit (%)	NR		42 (9)	32–50
Postop lowest hematocrit (%)	28 (1)		29 (11)	21–40
% change hematocrit	NR		−34 (9)	−20–50
Blood loss (L)	1.7 (5)	0.5–3	5 (11)	0.4–16
Blood transfused (L)	0.9 (3)	0.5–1.1	1.5 (11)	0.4–4

	AION (5)		PION (17)	
No. of cases and Reference	**Dilger 1998 Katz 1994 (3 patients) Stevens 1997**		**Abraham 2003 Alexandrakis 1999 Brown 1994 Dunker 2002 (5 patients) Katz 1994 Lee 2001Lee 1995 Murphy 2003 Roth 1997 (2 patients) Stevens 1997 (3 patients)**	
Volume crystalloids/colloid (L)	6.6/0.8 (4)	3.8–10/0.5–1	8.0/2.2 (9)	4–14/1.5–3.5
Preoperative history				
Coronary artery disease	20%		0%	
Hypertension	40%		53%	
Chronic anemia	0%		0%	
Diabetes mellitus	60%		27%	
Carotid disease/prior stroke	0%		0%	
Ocular disease	0%		6%	
Obesity	20%		12%	
Other	40%		29%	
No significant history/not indicated	20%		24%	
Vascular risk factors				
No risk factors/not indicated	20%		47%	
1 risk factor	60%		29%	
2 risk factors	0%		24%	
3 risk factors	20%		0%	
4 risk factors	0%		0%	
Time of symptoms				
Upon awakening	0%		59%	
Within 24 hours	40%		29%	
Postop day 2–5	20%		12%	
Postop day 5–9	20%		0%	
>Postop day 9	20%		0%	
Ocular findings				

	AION (5)	PION (17)
No. of cases and Reference	**Dilger 1998** **Katz 1994 (3 patients)** **Stevens 1997**	**Abraham 2003** **Alexandrakis 1999** **Brown 1994** **Dunker 2002 (5 patients)** **Katz 1994** **Lee 2001Lee 1995** **Murphy 2003** **Roth 1997 (2 patients)** **Stevens 1997 (3 patients)**
Pupil		
Afferent pupil defect	100%	33%
Non-reactive pupil	0%	12%
Funduscopy		
Disc edema	100%	0%
Normal	0%	100%
Hemorrhage	20%	0%
Cherry-red macula	0%	0%
Retinal ischemia	0%	0%
Visual defects		
Altitudinal defects	0%	27%
Central scotoma	0%	6%
Vision loss		
Monocular involvement	60%	53%
Binocular involvement	40%	47%
% both eyes involvement equal	20%	24%
Postoperative care		
% receiving intervention	20% (1)	29% (5)
Outcomes		
Some improvement at follow-up	60%	65%
No improvement at follow-up	20%	24%
Worsening at follow-up	0%	0%
Not indicated	20%	6%
% intervention with improvement	100% (1)	80% (5)

	AION (5)	PION (17)
No. of cases and Reference	**Dilger 1998** **Katz 1994** (3 patients) **Stevens 1997**	**Abraham 2003** **Alexandrakis 1999** **Brown 1994** **Dunker 2002** (5 patients) **Katz 1994** **Lee 2001Lee 1995** **Murphy 2003** **Roth 1997** (2 patients) **Stevens 1997** (3 patients)
% no intervention with improvement	33% (2)	58% (7)

Note: The numbers in parenthesis adjacent to mean or median values are the numbers of patients for which data were provided. References for the table: [22, 25-33].

TABLE 2

Individual Values for Age, Blood Pressure, Hemoglobin or Hematocrit, Blood Loss, and Quantities of Fluids Administered to the Patients

	Age (yr)	Lowest Intraoperative MAP	Lowest Intraoperative Hct	Lowest Postoperative Hct	% Change Preop to Intraop Hct	Blood Loss (L)	Crystalloids (L)	Colloid (L)
PION								
Alexandrakis 1999	68	no hypotension	ni	ni	ni	3		
Abraham 2003	24	ni	ni	ni	ni	0.35		
Brown 1994	13	52	ni	ni	ni	8	10.3	
Dunker 2002	66	73	32%	32%	−22%	2.4		
Dunker 2002	43	60	31%	31%	−26%	2		
Dunker 2002	12	60	26%	26%	−40%	2.5		
Dunker 2002	57	70	24%	24%	−41%	16		
Dunker 2002	44	(diastolic = 45)	26%	26%	−30%	ni		
Katz 1994	49	62	ni	ni	ni	2	3	
Lee 2001	58	85	39%	40%	−22%	0.8	8	
Lee 1995	48	55	ni	ni	ni	ni		
Murphy 2003	33	55	ni	ni	ni	1.2	4	
Roth 1997	44	63	27%	27%	−38%	ni	14	
Roth 1997	24	60	24%	24%	−50%	ni	6.4	
Stevens 1997	79	75	25%	31%	ni	2.2	2.5	1.5
Stevens 1997	27	ni	ni	36%	ni	5	9.5	1.5
Stevens 1997	37	ni	21%	21%	−35%	9	10	3.5
AION								
Dilger 1998	44	(systolic = 107)	ni	9.5	−40%	3	10	1
Katz 1994	41	62	ni	11.7	−24%	2	7	
Katz 1994	60	62	ni	8.6	−48%	2	5.5	0.5
Katz 1994	65	63	ni	8	−43%	0.5	3.8	
Stevens 1997	56	78	ni	ni	ni	0.85		
				(Hemoglobin)	(% change to postop Hgb)			

	Age (yr)	Lowest Intraoperative MAP	Lowest Intraoperative Hct	Lowest Postoperative Hct	% Change Preop to Intraop Hct	Blood Loss (L)	Crystalloids (L)	Colloid (L)

Ni = not indicated; MAP = mean arterial pressure; Hct = hematocrit; Hgb = hemoglobin.

For AION, data were not available for changes in Hct.