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Postoperative Visual Loss

Experts, Data, and Practice

CME This editorial accompanies the article selected for this month's *Anesthesiology* CME Program. After reading the article and editorial, go to http://www.asahq.org/journal-cme to take the test and apply for Category 1 credit. Complete instructions may be found in the CME section at the back of this issue.

PLEASE allow me to paraphrase recent plaintiffs' experts in cases related to postoperative visual loss (POVL) in patients who had undergone spine surgery while positioned prone.

The anesthesiologist clearly caused this patient's vision loss by letting the mean arterial blood pressure drop below 60 mmHg for more than 5 min.

This patient is now blind because the anesthesiologist let the patient's hematocrit level decrease to less than 24% during the case.

The person providing the patient's anesthesia failed to avoid prolonged pressure on the eye, leading to the patient's ischemic optic neuropathy and blindness.

No one wants any patient to experience a catastrophic perioperative event such as permanent blindness. However, these statements (and others similar to them) are devastating to the anesthesiologists who have provided apparent good care to patients undergoing spine procedures, only to find that their patients have awakened with near complete loss of vision in one or both eyes.

What is wrong with these statements? First, these plaintiff "experts" are making statements for which there are no supporting data. Second, good anesthesiologists experience loss of confidence and, in some cases, shattered practices while these legal actions are being resolved. Last, these statements detract attention and effort from the development of studies to determine the etiologies of, and possible preventive measures for, perioperative blindness. Our patients deserve better.

Postoperative visual loss is a real problem in patients undergoing cardiac and spine surgical procedures. POVL occurs more frequently in cardiac surgical patients. There seem to be multiple etiologies for POVL in cardiac surgical patients (*e.g.*, embolic, thrombotic, oncotic, and ischemic reasons), several of which may be related to the surgical procedures and operative techniques themselves.[1] These patients have numerous visual pathologic consequences, including central retinal artery occlusion and ischemic optic neuropathy (ION). Because there are many factors that may contribute to the development of POVL in cardiac surgical patients, it is a difficult problem to study.

In contrast, POVL in patients undergoing spine surgery entices us with the potential to discern etiologic factors in a group of patients who have less variation in visual pathology and outcomes. Dr. Lee *et al.*[2] report these findings this month in their review of the first 6 yr of cases submitted to the American Society of Anesthesiologists Postoperative Visual Loss Registry. This registry was established in 1999 after anesthesiologists and others voiced concern that the frequency of POVL seemed to be increasing, particularly in patients undergoing spine surgery. In their comprehensive report of 93 cases of POVL in patients undergoing spine surgery, the authors note that 89% of the patients developed ION, with the other 11% of patients experiencing central retinal artery occlusion. Most patients with central retinal artery occlusion had evidence of ocular trauma and unilateral visual loss, suggesting that in some instances, positioning or other potentially controllable factors may have played a role. Many of the patients with ION had bilateral visual loss, suggesting that one or more systemic factors, including inherent patient-specific factors, may have been present. In the patients with ION, the highest-risk group included patients who had anesthetics lasting more than 6 h and estimated blood loss of greater than 1 l. The authors were unable to find any factor under the direct control of anesthesiologists that led to a high frequency of ION. Specifically, patients who developed ION had intraoperative mean arterial blood pressures and hematocrits that ranged widely, with patients at each extreme of these parameters developing ION.

What is the typical natural history of ION in spine surgery patients? After awakening, the patients often note an inability to see, citing that they can only perceive gray shadows (usually in response to objects in motion). An ophthalmologic examination may show an edematous optic disc if the ischemia is associated with the anterior portion of the optic nerve, but more often there are no acute funduscopic findings. The papillary light

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reflex may be reduced or absent, but this finding, too, often is difficult to discern. Imaging of the visual pathways by magnetic resonance or computed tomography is usually fruitless but may be worthwhile to confirm the absence of other pathologic changes (*e.g.*, cerebral infarction or hemorrhage). As demonstrated by Dr. Lee *et al.*, vision rarely is improved over time from the initial impaired postoperative status.

A recently published American Society of Anesthesiologists Practice Advisory[3] provided several important findings:

The use of deliberate hypotensive techniques during spine surgery has not been shown to be associated with the development of perioperative visual loss.

At this time, there is no apparent transfusion threshold that would eliminate the risk of perioperative visual loss related to anemia.

These findings seem to be confirmed by data from the current article and negate the veracity of the first two statements above that are paraphrased from plaintiffs' experts. The current article also reports that none of the 83 spine surgery patients who developed ION had periorbital or ocular findings suggestive of intraoperative trauma or externally applied pressure, thus negating the third statement paraphrased above.

Unfortunately, the current article does not advance our understanding of potential etiologic factors that cause ION in spine surgery patients except for the confirmation that the highest-risk patients are those who undergo anesthetics greater than 6 h in duration and who experience intraoperative blood loss of more than 1 l. How can we best use this information? First, we should consider informing patients in this high-risk group that there is a small, unpredictable risk of POVL. Second, we should collaborate with our surgical colleagues to consider staging spine procedures in these high-risk patients in an effort to reduce prolonged, bloody procedures in patients who are positioned prone. This latter suggestion has been endorsed by the North American Neuro-Ophthalmology Society and supported by the North American Spine Society.

Opportunities at the bench side to study POVL are severely limited by the lack of animal models that have eye and visual pathway characteristics and anatomy similar to that of humans. Newer or improved imaging modalities (*i.e.*, positron emission tomography) may offer the ability to better distinguish between ION occurring in the anterior or posterior optic nerve, but it is unclear whether imaging techniques can provide the information needed to make a positive impact on the frequency of ION. In the meantime, we must rely on multi-institutional case–control and difficult-to-perform prospective studies of this rare but catastrophic event to determine potential risk factors and practice changes that may lead to reductions in its frequency.

In summary, Dr. Lee *et al.*, along with the American Society of Anesthesiologists, should be congratulated for providing data that at least help us to identify patients undergoing spine surgery who fall into a high-risk group for POVL. Ironically, their data also tell us what we do not yet know about POVL. The current article provides important information that, if examined responsibly by plaintiffs' "experts," should reduce the frequency of unsubstantiated claims and promote a more informed and fair resolution of legal actions.

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The American Society of Anesthesiologists Postoperative Visual Loss Registry

Analysis of 93 Spine Surgery Cases with Postoperative Visual Loss

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CME This article and its accompanying editorial have been selected for the *Anesthesiology* CME Program. After reading both articles, go to http://www.asahq.org/journal-cme to take the test and apply for Category 1 credit. Complete instructions may be found in the CME section at the back of this issue.

Background: **Postoperative visual loss after prone spine surgery is increasingly reported in association with ischemic optic neuropathy, but its etiology is unknown.**

Methods: **To describe the clinical characteristics of these patients, the authors analyzed a retrospectively collected series of 93 spine surgery cases voluntarily submitted to the American Society of Anesthesiologists Postoperative Visual Loss Registry on standardized data forms.**

Results: **Ischemic optic neuropathy was associated with 83 of 93 spine surgery cases. The mean age of the patients was 50 ± 14 yr, and most patients were relatively healthy. Mayfield pins supported the head in 16 of 83 cases. The mean anesthetic duration was 9.8 ± 3.1 h, and the median estimated blood loss was 2.0 l (range, 0.1–25 l). Bilateral disease was present in 55 patients, with complete visual loss in the affected eye(s) in 47. Ischemic optic neuropathy cases had significantly higher anesthetic duration, blood loss, percentage of patients in Mayfield pins, and percentage of patients with bilateral disease compared with the remaining 10 cases of visual loss diagnosed with central retinal artery occlusion ($P < 0.05$), suggesting they are of different etiology.**

Conclusions: **Ischemic optic neuropathy was the most common cause of visual loss after spine surgery in the Registry, and most patients were relatively healthy. Blood loss of 1,000 ml or greater or anesthetic duration of 6 h or longer was present in 96% of these cases. For patients undergoing lengthy spine surgery in the prone position, the risk of visual loss should be considered in the preoperative discussion with patients.**

This article is accompanied by an Editorial View. Please see: Warner MA: Postoperative visual loss: Experts, data, and practice. ANESTHESIOLOGY 2006; 105:641–2

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†† Providers' Packet for Physicians Completing Case Report Forms. Available at: www.asaclosedclaims.org or http://depts.washington.edu/asaccp/eye/providers/packet.pdf. Accessed October 26, 2005.

POSTOPERATIVE visual loss (POVL) is a relatively uncommon but devastating complication that is most often associated with cardiac, spine, and head and neck operations. Estimates for spine and cardiac surgery are as high as 0.2%[1] and 4.5%,[2] respectively. During the mid-1990s, hospital risk managers, anesthesiologists, ophthalmologists, and surgeons voiced concern that POVL seemed to be increasing, particularly for spine surgery.[1,3,4] Most of the ophthalmologic lesions were caused by ischemic optic neuropathy (ION) and were not consistent with an etiology of globe compression. ION more commonly occurs spontaneously and has been associated with atherosclerotic risk factors,[5] with adverse effects of antihypertensive medications,[6] and, more recently, with sildenafil.[7] The unexpected occurrence and serious nature of ION after nonocular surgery warranted further investigation of patient characteristics and perioperative events.

Because the number of POVL cases from a single institution is very low, a multi-institutional database is required to obtain sufficient numbers for meaningful analysis of common perioperative characteristics or events. In response to this problem, the American Society of Anesthesiologists (ASA) Committee on Professional Liability established the ASA POVL Registry in 1999 to collect detailed information on cases of POVL occurring after nonocular surgery. This report provides an in-depth analysis of 93 cases associated with spine surgery from the ASA POVL Registry. Patient characteristics and perioperative anesthetic and surgical events are described.

Materials and Methods

Study Population

After approval by the Institutional Review Board at the University of Washington (Seattle, Washington), voluntary enrollment of POVL cases by physicians occurring within 7 days after nonocular surgery began in 1999. Cases were submitted on a detailed data collection form available from the University of Washington or from the ASA Closed Claims Project.†† All data were de-identified with respect to patient, physicians, and institutions.

Patient and Perioperative Characteristics

Information collected included patient demographics, medical history including the presence of risk factors for vascular disease (obesity, hypertension, coronary artery disease/myocardial infarction, cerebrovascular disease, diabetes, hypercholesterolemia, and tobacco history), current medications, and surgical history. Intraoperative information included procedure description, number of levels fused and/or instrumented, type of headrest and surgical frame, patient position on frame, and frequency of eye checks. Durations of anesthetic, surgery, and prone positioning were recorded. Type of anesthetic, drugs, and fluids administered preoperatively and intraoperatively were included. Estimated blood loss (EBL) and type of blood products administered were obtained along with preoperative and lowest hemoglobin/hematocrit values, and urine output. Use of deliberate hypotension and specific hypotensive agents were noted.

Intraoperative blood pressure was recorded as absolute mean arterial blood pressure and/or systolic blood pressure (SBP) and percentage below baseline. Data were collected for blood pressure values 20%, 40%, 50%, and more than 50% below baseline values. Blood pressure values were entered only if the blood pressure decreased within a predefined range of values for a minimum of 15 consecutive or nonconsecutive minutes. Presence of hypothermia (temperature < 35°C for a minimum of 30 min) was noted, as were any adverse intraoperative events including cardiogenic shock, cardiac arrest, seizures, and direct trauma to the eye.

Ophthalmologic Examination Characteristics and Diagnostic Criteria

Detailed information on the ophthalmologic examination for each eye was obtained including type of visual deficit (*i.e.*, decreased visual acuity, visual field deficit, or complete loss of vision), time when visual symptoms were first noted, funduscopic examination, and ophthalmologic diagnosis. Classification of the specific lesion was based on ophthalmologic diagnosis or, if unavailable, findings consistent with standard diagnostic criteria. For central retinal artery occlusion (CRAO), these criteria included a pale ischemic retina with a pathognomonic cherry-red spot at the macula and a relative afferent pupillary defect or reduced pupillary light reflex. A diagnosis of anterior ischemic optic neuropathy (AION) required an early funduscopic examination demonstrating an edematous disc with or without peripapillary flame-shaped hemorrhages and a relative afferent pupillary defect or reduced pupillary light reflex. Criteria consistent with posterior ischemic optic neuropathy (PION) included a normal early funduscopic examination with a relative afferent pupillary defect or an absent pupillary light reflex. Eventual optic nerve pallor was consistent with both AION and PION. Lack of an early funduscopic examination before the appearance of isolated optic nerve pallor was categorized as unspecified ION.

Any treatment and prognosis for recovery of vision was noted, but duration of follow-up varied from initial examination to 4 yr postoperatively. Finally, a summary of events was provided by the physician submitting the case, including any related diagnostic workup (*e.g.*, visual evoked potentials, magnetic resonance imaging or computed tomography of the head and orbits, carotid duplex).

Inclusion criteria for this analysis included any POVL case associated with spine surgery from the ASA POVL Registry with the diagnosis of CRAO, AION, PION, or unspecified ION. Two coauthors (L.A.L. and K.L.P.) reviewed all data forms, and other coauthors (S.R. and N.J.N.) were consulted to resolve a question of appropriate ophthalmologic diagnosis.

Statistical Analysis

Reliability of data were tested by comparing duplicate submissions ($n = 13$) from separate individuals. The κ statistic was acceptable for all categorical variables tested (0.40–0.55 for hypertension, percentage below baseline blood pressure, and recovery of vision; 0.75–1.0 for sex, diabetes, smoking, coronary artery disease, previous myocardial infarction, obesity, instrumentation/fusion, unilateral or bilateral disease, periocular trauma, and loss of vision). However, confidence intervals were wide because of the small sample size. Continuous variables (age, anesthesia duration, EBL, intravenous fluid administration, number of vertebral levels, lowest SBP, and total number of coexisting diseases) all had high intraclass correlation coefficients (0.798–0.999) with narrow confidence intervals, indicating excellent agreement of data submitted by different individuals. Each pair of duplicate submissions was entered only once in the Registry.

Differences between ION and CRAO cases and between AION and PION cases were analyzed using the Student t test with $P \leq 0.05$ considered statistically significant. Median and range were reported as descriptive statistics when distributions were nonnormal, with comparison by Mann–Whitney U test. Differences in proportions were compared using the Z test.

Results

As of June 2005, 93 cases of POVL associated with spine surgery were entered into the ASA POVL Registry that met inclusion criteria, out of a total 131 cases (72%). Other cases included 2 spine cases that did not meet inclusion criteria, 12 cardiac cases, 6 major vascular cases (3 aortic, 2 peripheral vascular, 1 carotid endarterectomy), 5 orthopedic cases, and 13 miscellaneous cases.

Table 1. ASA POVL Registry: Ophthalmic Lesion Associated with POVL after Spine Surgery (n = 93)

Ophthalmic Lesion	Cases, n (% of 93)	No Light Perception, n (% this lesion − row %)
ION	83 (89)	47 (57)
PION	56 (60)	34 (61)
AION	19 (20)	8 (42)
ION unspecified	8 (9)	5 (63)
CRAO	10 (11)	7 (70)

ASA = American Society of Anesthesiologists; AION = anterior ischemic optic neuropathy; CRAO = central retinal artery occlusion; ION = ischemic optic neuropathy; PION = posterior ischemic optic neuropathy; POVL = Postoperative Visual Loss.

Ischemic optic neuropathy was the cause of visual loss in 83 (89%) of these 93 cases, of which 56 were diagnosed with PION, 19 were diagnosed with AION, and 8 were diagnosed with unspecified ION (table 1). Thirty-one cases occurred before 1999, when the ASA POVL Registry was initiated. There were no statistically significant differences between AION and PION cases with respect to demographics, coexisting diseases, surgical characteristics, or anesthetic management (data not shown). Because of the lack of these differences between AION and PION cases, the difficulty in distinguishing AION from PION (particularly in the absence of an early ophthalmologic examination), and the uncertainty whether AION and PION occurring after spine surgery are different disease states with separate etiologies, all AION, PION, and unspecified ION cases were combined under the ION group for comparison with CRAO cases. CRAO accounted for the remaining 10 POVL cases.

Demographics and Coexisting Diseases of Spine Surgery Cases with ION

Operations for the 83 spine cases with ION occurred between 1987 and 2004. There were significantly more males than females (72% *vs.* 28%; $P < 0.05$), and the mean age was 50 ± 14 yr (range, 16–73 yr; table 2). Most patients were relatively healthy (64% ASA physical status I or II), and 96% were undergoing elective surgery. Coexisting diseases, including hypertension, diabetes, tobacco use, coronary artery disease, cerebrovascular disease, increased cholesterol/lipids, and obesity, were present in 4–53% of cases (table 2). At least one of these conditions was present in 82% of cases (n = 68; table 2). Of the 41% of hypertensive patients, 13 used β-blockers, 11 used angiotensin-converting enzyme inhibitors or angiotensin-converting enzyme receptor antagonists, 11 used calcium channel blockers, 11 used diuretics, and 5 used other or unknown medications. No patient had a preoperative history of glaucoma.

Description of Operations and Positioning of Spine Surgery Cases with ION

The surgical procedure for most of the spine cases with ION (89%) involved fusion and/or instrumentation on more than one vertebral level in the thoracic, lumbar, or sacral spine (table 3). Approximately one third (39%) of patients had undergone previous spine surgery. All of the patients were positioned prone for a portion of the procedure, except two anterior spine procedures. Ten procedures involved supine/lateral and prone positioning (*i.e.*, anterior–posterior operations). The Wilson frame, Jackson table, and soft chest rolls were used in similar proportions (table 4). Headrests used most commonly were foam pads, Mayfield pins, and donut/gel pads (table 4). Eye checks were documented by the anesthesiologist in 42 cases (51%).

Table 2. ASA POVL Registry Spine Cases with ION: Patient Characteristics (n = 83)

Demographics	n (% of 83 cases)
Age, mean (SD), yr	50 ± 14
Male	60 (72)
ASA I or II	53 (64)
ASA III	24 (29)
ASA IV	2 (2)
Emergency	3 (4)
Coexisting diseases	
Hypertension	34 (41)
Diabetes	13 (16)
Tobacco use	38 (46)
Coronary artery disease	8 (10)
Cerebrovascular disease	3 (4)
Increased cholesterol/lipids	11 (13)
Obesity	44 (53)
≥ 1 Coexisting diseases	68 (82)

American Society of Anesthesiologists (ASA) physical status data do not add up to 100% because of missing data in four cases.

ION = ischemic optic neuropathy; POVL = Postoperative Visual Loss.

Anesthetic Management of Spine Surgery Cases with ION

General anesthesia was used uniformly with a combination of volatile and narcotic (89%), total intravenous

Table 3. ASA POVL Registry: Surgical Characteristics in Spine Cases with ION (n = 83)

Surgical Variable	n (% of 83 cases)
Fusion/instrumentation	74 (89)
Previous spine surgery	32 (39)
Number of vertebral levels	
1	9 (11)
2	19 (23)
3	15 (18)
≥ 4	30 (36)
Unknown number of levels	10 (12)
Vertebral location	
Cervical/cervicothoracic	4 (5)
Thoracic/thoracolumbar	11 (13)
Lumbar	22 (27)
Lumbosacral/sacral	35 (42)
Thoracolumbosacral	5 (6)
Unknown location	6 (7)

ASA = American Society of Anesthesiologists; ION = ischemic optic neuropathy; POVL = Postoperative Visual Loss.

Table 4. ASA POVL Registry: Type of Surgical Frames, Tables, and Headrests in Spine Cases with ION (n = 83)

	n (% of 83 cases)
Type of surgical frame or table	
Wilson frame	25 (30)
Jackson spinal table	22 (27)
Soft chest rolls	17 (20)
Knee–chest tables	7 (8)
Other/unknown tables	12 (14)
Type of headrest	
Foam pad	47 (57)
Mayfield pins	16 (19)
Donut/gel pad	7 (8)
Other/unknown	13 (16)

ASA = American Society of Anesthesiologists; ION = ischemic optic neuropathy; POVL = Postoperative Visual Loss.

anesthesia with propofol and narcotic (2%), and unknown general anesthetic agents (8%). All of the commonly utilized volatile anesthetics (isoflurane [59%], sevoflurane [14%], and desflurane [22%]) and nitrous oxide (29%) were administered.

The mean anesthetic duration was 9.8 ± 3.1 h (table 5), and 94% of cases were 6 h or longer (fig. 1). The mean prone position duration was 7.7 ± 3.1 h. The median EBL was 2.0 l (range, 0.1–25 l; table 5), and 82% of cases had an EBL of 1.0 l or greater (fig. 2). Fluid management varied, with colloid (hydroxyethyl starch or albumin) used in 30% of cases and a mean intravenous crystalloid replacement of 9.7 ± 4.7 l (table 5). Blood was replaced with cell saver (54%), packed erythrocytes (57%), and whole blood (11%). The lowest hematocrit (mean) was 26 ± 5% (table 5), and 17% of cases had a nadir hematocrit of 30% or greater. Urine output was less than 0.5 ml · kg^{-1} · h^{-1} in 24% of cases, with postoperative increased creatinine in six cases and rhabdomyolysis in three.

Blood pressure varied widely for both absolute SBP values and percent below baseline blood pressure. In 33% of cases, the lowest SBPs were greater than 90 mmHg, whereas 20% had the lowest SBP 80 mmHg or less (table 6). In 6% of cases, the lowest mean arterial blood pressure or SBPs were less than 20% below baseline, whereas 34% of cases had the lowest mean arterial pressure or SBP 40% or greater below baseline (table 6). Deliberate hypotension was used in 27% of cases (table 6). Agents most commonly used for deliberate hypotension were labetalol or esmolol (n = 10) and volatile agents (n = 5). Phenylephrine (≥ 1 mg total dose) was administered in 27% of cases. Hypothermia was present in 10% of cases.

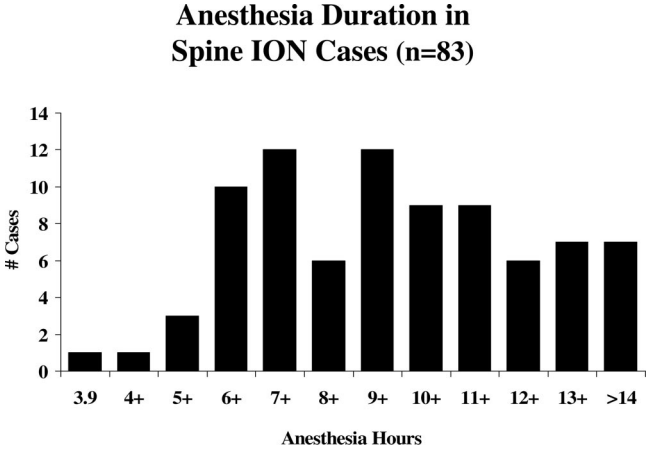

Fig. 1. Anesthetic duration for 83 spine cases associated with ischemic optic neuropathy (ION). The majority of cases (94%) were 6 h or longer in duration.

Ophthalmologic Findings for Spine Surgery Cases with ION

Of the 83 patients with ION, 55 (66%) had documented bilateral involvement, for a total of 138 affected eyes (table 5). The median onset time of reporting visual loss postoperatively was 15 h (range, 0–168 h), with the exception of one patient who was mechanically ventilated for 2 weeks postoperatively and reported complete blindness 2 days after extubation. Full or partial eye opening was noted immediately postoperatively in 43 patients, inability to open one or both eyes was noted in 12 patients, and eye opening information was missing in 28 patients. There was associated periocular trauma in only one case (table 5). Visual fields were restricted in 134 of 138 affected eyes (97%), and complete blindness with loss of light perception occurred in 64 of 138 affected eyes (47 patients). PION was diagnosed in 67% of all ION cases, AION was diagnosed in 23% of cases, and unspecified ION was diagnosed in 10% of cases (table 1). There was some degree of recovery of vision in 42% of ION cases (table 5), although improvement in vision was often clinically insignificant (*e.g.*, light/dark perception to hand motion only). Follow-up of ophthalmologic examinations was inconsistent and varied from only the initial examination to 4 yr postoperatively.

Spine Surgery Cases with CRAO (n = 10)

The mean age for the 10 patients with CRAO was 46 ± 13 yr (table 5). Horseshoe headrests were used in 3 cases, foam pads were used in 2, and miscellaneous headrests were used in 5. Mayfield pins were not used for any CRAO cases, in contrast to 19% of ION cases ($P = 0.001$). Eye checks were performed in 6 of 10 cases at intervals ranging from 30 min to only once during a 10-h case. Eye checks were not performed in 3 cases (missing data in 1 case). The mean anesthetic duration and median EBL were significantly less in the CRAO compared with the ION group ($P = 0.002$ and 0.001, respectively; table 5). The mean lowest hematocrit was higher in the CRAO than in ION cases, although not significantly different ($P = 0.075$; table 5). Deliberate hypotension was used in 4 of 10 CRAO cases. In contrast to the ION group, there were no cases of bilateral CRAO ($P = 0.001$). Recovery of vision between CRAO and ION

Table 5. Comparison of ION and CRAO Cases from the ASA POVL Registry (n = 93)

	ION (n = 83)	CRAO (n = 10)	*P* Value
Age, mean (SD), yr	50 (14)	46 (13)	0.34*
Anesthetic duration, mean (SD), h	9.8 (3.1)	6.5 (2.2)	0.002*
Estimated blood loss, median (range), l	2.0 (0.1–25)	0.75 (0.5–1.8)	0.001†
Crystalloid infusion, mean (SD), l	9.7 (4.7)	4.6 (1.7)	0.001*
Lowest hematocrit, mean (SD)	26 (5)	31 (6)	0.075*
Bilateral disease, number of cases (% of column)	55 (66)	0 (0)	0.001‡
Any visual recovery, number of cases (% of column)	35 (42)	2 (20)	0.11‡
Mayfield pins, number of cases (% of column)	16 (19)	0 (0)	0.001‡
Ipsilateral periocular trauma, number of cases (% of column)	1 (1)	7 (70)	0.001‡

* *t* test. † Mann–Whitney U test. ‡ Z test.

ASA = American Society of Anesthesiologists; CRAO = central retinal artery occlusion; ION = ischemic optic neuropathy; POVL = Postoperative Visual Loss.

groups was not significantly different ($P = 0.11$). Periocular trauma was documented in 7 of 10 CRAO cases compared with 1 of 83 ION cases ($P = 0.001$; table 5) and included ipsilateral findings of decreased supraorbital sensation, ophthalmoplegia, corneal abrasion, ptosis, or unilateral erythema.

Discussion

The ASA POVL Registry was created because prospective data collection on this low-incidence complication was impractical. It is unclear how the preponderance of spine surgery cases in this analysis relates to the actual proportion of all POVL cases because the ASA POVL Registry lacks denominator data. Incidence of any POVL injury cannot be ascertained. Reporting bias from direct participation in the case and error from retrospective data collection are possible, especially for cases occurring before the start of the Registry in 1999, when information from the medical records may have been more difficult to obtain. Further, the accuracy of the data cannot be verified in these anonymous case submissions. However, data for this analysis were obtained from medical records, and of the 13 cases of duplicate submissions by separate individuals, the κ scores and intraclass correlation coefficients were all acceptable, validating the accuracy of reporting. The perceived increase in POVL in association with spine surgery may be related to multiple factors, including increased awareness of the problem, increased rates of spinal fusion operations over the past decade,[8] or other variables. Our database cannot test these suggestions in a rigorous manner.

The etiology of ION remains unknown, and the majority of the literature on perioperative ION after spine surgery is based on case reports, reviews of case reports, and retrospective studies.[1,3,4,9–11] This analysis of the ASA POVL Registry is the largest, and most detailed to date, of patients with ION after spine surgery (n = 83). The demographics of these patients demonstrate a predominately middle-aged, relatively healthy population, which may reflect the greater than 200% increase in spinal fusion rates in the 1990s for older adults.[8] The finding of 72% male patients in this database is striking given that the National Inpatient Sample data for 1999

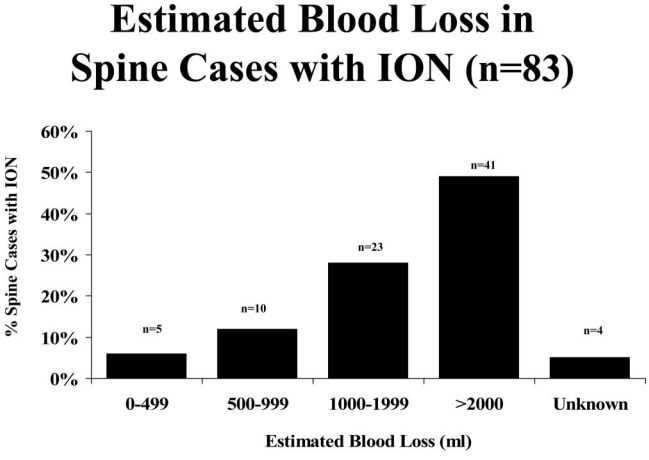

Fig. 2. Estimated blood loss for 83 spine cases associated with ischemic optic neuropathy (ION). In the majority of cases (82%), blood loss was 1,000 ml or greater.

Table 6. ASA POVL Registry: Lowest Blood Pressure* in Spine Cases with ION (n = 83)

	n (% of 83 cases)
Lowest SBP, mmHg	
> 110	4 (5)
101–110	7 (8)
91–100	17 (20)
81–90	35 (42)
71–80	12 (14)
≤ 70	5 (6)
Unknown	3 (4)
Lowest MAP or SBP as % below baseline, mmHg	
< 20%	5 (6)
20–39%	47 (57)
40–49%	21 (25)
≥ 50%	7 (8)
Unknown	3 (4)
Deliberate hypotension	22 (27)

* Blood pressure ranges were based on 15 min of blood pressure at a given range.

ASA = American Society of Anesthesiologists; ION = ischemic optic neuropathy; MAP = mean arterial pressure; POVL = Postoperative Visual Loss; SBP = systolic blood pressure.

spinal fusion procedures (n = 188,309) demonstrates a 48% male:52% female ratio.‡‡ The influence of sex on ulnar nerve injuries has also been documented in the same proportion (70% male).[12] Previous studies on ulnar neuropathy have suggested that anatomical differences predispose men to this injury, but hormonal differences may be equally as important. Experimental animal models of cerebral ischemia have demonstrated a protective effect of estrogen, and this may contribute to the lower proportion of women with both ulnar and optic nerve injuries.[13]

Although there are few children or teenagers who developed perioperative ION, young age did not render patients immune to this complication. Moreover, one retrospective study from a single institution included two 13-yr-old patients who developed PION after spine surgery.[11] Older patients may be more vulnerable to these injuries than younger patients because there is a natural reduction in optic nerve fibers of approximately 5,000 axon loss per year of life, compared with the 0.8–1.2 million fibers in early childhood.[14,15] However, the occurrence of this complication in teenagers, and in patients with relatively few vascular risk factors, suggests that "normal" anatomical or physiologic variation in the optic nerve blood supply between individuals may place some patients more at risk for this devastating complication than others. Consequently, preoperative identification of patients at high risk for developing ION after spine surgery is not currently possible based on specific patient characteristics or coexisting diseases.

The occurrence of ION in 16 patients whose heads were placed in Mayfield pins with the eyes free of pressure clearly demonstrates that ION occurs in the absence of pressure on the globe. These findings are consistent with the lack of retinal ischemia on ophthalmologic examination in ION. Moreover, the occurrence of ION in both eyes in the majority of cases is more consistent with a systemic etiology, rather than globe compression that usually affects only one eye. The 10 patients with CRAO, a lesion that is known to result from globe compression, all had unilateral disease usually associated with ipsilateral periocular trauma and demonstrated significantly shorter anesthetic durations and lower EBL. Although specialized ophthalmologists have long known that pressure on the globe does not result in isolated ION,[16] these findings provide more convincing evidence to patients, surgeons, and anesthesiologists.

Blood pressure management for the 83 spine cases with ION varied widely, with blood pressure decrements anywhere from less than 20% of baseline (n = 5) to 50% of baseline or greater (n = 7). The relatively high proportion of cases (27%) using deliberate hypotension reflects a long-standing clinical practice of decreasing blood pressure to reduce blood loss during major spine surgery.[17] This technique has not been previously associated with POVL after spine surgery in prospective or retrospective studies on deliberate hypotension,[18,19] although studies with adequate power to detect this infrequent complication are lacking. Autoregulation of blood flow in the cerebral circulation has been well demonstrated in humans, albeit with a high degree of variability in the lower limit of autoregulation (mean arterial pressure < 57 to 91 mmHg).[20,21] It is not clear whether the optic nerve in humans also has the ability to autoregulate in both anterior and posterior regions.[22–24] The occurrence of ION in many cases without apparent hypotension makes the role of blood pressure management unclear. The case–control study by Myers *et al.*[4] of spine patients with POVL did not show any difference in lowest blood pressure between patients who developed visual loss from any lesion (ION, CRAO, and cortical blindness) and those who did not. Future case–control studies of only patients with ION after spine surgery may help to determine whether certain blood pressure reductions are associated with an increased risk of developing this complication.

Whether the finding of moderate anemia in the majority of these cases is a result of the fact that these were predominately major spine procedures with large blood losses, or whether anemia contributes to the development of ION, cannot be discerned by this study. The finding of ION in a patient with a nadir hematocrit of 40% demonstrates that ION occurs in the absence of anemia. Of the 83 spine surgery patients with ION, 14 cases (17%) occurred with a nadir hematocrit of 30% or greater. The effects of hemodilution on the blood flow and oxygen delivery to the optic nerve have not been well studied in either animals or humans.

Of note, two interrelated factors regarding the surgical procedure were common to most cases. EBL of 1,000 ml or greater occurred in 82% of cases, and anesthetic duration of 6 h or longer was present in 94%. One of these two factors was present in all but three ION cases. Myers *et al.*[4] also found that long duration and large EBL were associated with POVL after spine surgery, but their data combined all causes of POVL, including ION, CRAO, and cortical blindness. Although there is not yet enough information to confirm a relation between surgical duration, magnitude of blood loss, and the risk of POVL, there is an opportunity for further clinical study. This could be accomplished by comparing outcomes in conventional single-stage surgery to outcomes in staged surgery. Such a study would require considerations of the added costs of staged surgery and the potential for increased risks from perioperative complications such as infection, pneumonia, and deep venous thrombosis.

The ASA POVL Registry does not allow us to establish a definite etiology for perioperative ION, but it is noteworthy that 72% of all ION cases in the Registry were

‡‡ National and Regional Statistics from the National Inpatient Sample. Available at: http://hcup.ahrq.gov/HcupNet.asp. Accessed February 15, 2006

associated with spine surgery in the prone position. This observation is consistent with the hypothesis that the venous pressure within the optic nerve may become increased during prone surgery, perhaps due to venous engorgement. The plausibility of this hypothesis is supported by the observation that intraocular pressure increases when awake and anesthetized patients are placed in the prone position.[25–27] Blood flow in the posterior optic nerve may be particularly susceptible to increased venous pressure because the arterial vessels that supply the posterior optic nerve are small end-vessels from the surrounding pia.[28] Further support for this hypothesis comes from case reports of ION that have occurred in patients with increased venous and intracranial pressure after radical neck operations with bilateral internal jugular vein ligation.[29,30] These reports suggest that high venous pressure and interstitial tissue edema may compromise blood flow in the optic nerve. Histopathologic studies of PION in one patient with severe blood loss and in two patients after bilateral radical neck dissection demonstrated central hemorrhagic infarctions several millimeters posterior to the lamina cribrosa to several millimeters anterior to the optic nerve canal—an area supplied by the small pial vessels.[31–33] A related hypothesis is that ION is a "compartment syndrome of the optic nerve" created by increased venous pressure and interstitial fluid accumulation within the relatively nondistensible space of either the semirigid lamina cribrosa at the optic nerve head or the bony optic canal. Proponents of this hypothesis have frequently recommended a head-up body position and colloid-based fluid resuscitation in prone spine surgery to decrease the potential interstitial edema around the optic nerve. Body position could not be reliably discerned from these cases. However, the use of colloid in 30% of these ION cases and in many case reports and case series suggests that its role in prevention of ION remains undetermined.[1,9]

It is notable that ION almost always occurred without any accompanying evidence of vascular injury in other critical organs, such as the heart or brain, even in patients with preexisting coronary atherosclerosis, diabetes, and hypertension. This observation suggests that the optic nerve vasculature may be uniquely vulnerable to hemodynamic perturbations in the prone position in some patients.

In summary, more than two thirds of the cases in the ASA POVL Registry were related to spine surgery in the prone position, and 89% of these cases were associated with ION. Most spine surgery patients with ION were relatively healthy and had a wide range of nadir hematocrits and blood pressure management that may reflect a multifactorial etiology. EBL of 1,000 ml or greater or anesthetic duration of 6 h or longer was present in 96% of these cases. For patients undergoing lengthy spine surgery in the prone position, the risk of visual loss should be considered in the discussion of perioperative risks.

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Multifactorial Etiology of Postoperative Vision Loss

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In Reply:—
We appreciate the interest of Drs. Benumof and Mazzei in our case report of visual loss after an uneventful spine surgery in the prone position in an otherwise healthy patient. [1] They expressed concerns about several issues. In particular, their emphasis on "adequacy" of eye-check and padding provides us with the opportunity to address an important issue that we perhaps did not adequately explain in our report, leading to their misunderstanding. Adequate padding of the head in the prone position to prevent pressure on the eyes is important, and in the case we reported, we used a standard foam support (Gentletouch Headrest Pillow, Orthopedic Systems Inc., Union City, CA), with a cut-out for the eyes. Eye-checks can be performed accurately by pressing down on the side of the foam cushion without altering the correct positioning. The patient is of Caucasian extraction with a high nasal bridge. Moreover, the nasal bridge is of no consequence provided that the eyes are positioned correctly in the cut-out portion of the foam cushion.
Of utmost concern is their perseveration regarding the type of foam cushion, the height of the nasal bridge, and the method of eye-checks, which underscores a fundamental lack of understanding, and a common misconception about the pathophysiology of visual loss after spine surgery. [2] The ophthalmologic diagnosis in our case report, similar to most cases of visual loss after prone spine surgery, was posterior ischemic optic neuropathy. The lesion lies posterior to the lamina cribosa, sparing the retina, and has never been shown to be related to pressure on the globe. [3] Visual loss from pressure on the globe, on the other hand, is secondary to central retinal artery occlusion with or without anterior ischemic optic neuropathy. These patients will frequently show signs of external periorbital bruising or proptosis, with evidence of retinal ischemia, which our patient did not. Moreover, in some patients who develop visual loss after prone spine surgery, Mayfield head pins were used instead of foam cushions, removing all doubts about pressure on the eyes. (ASA Postoperative Visual Loss Registry, unpublished data, 2001). The emphasis on pressure on the eyeballs in the context of postoperative visual loss is akin to the man looking for his keys under the lamppost after dropping them on the lawn; he sees a bright spot but he won't find the keys. While we can all applaud efforts to improve patient safety with foam cushions of better designs (the Dupaco Prone-View foam cushion is certainly a good one), overemphasis on this aspect will divert our attention and focus away from the real pathophysiology and prevention of postoperative

visual loss from ischemic optic neuropathy.
As for their final point, we would advise Drs. Benumof and Mazzei to read the original discussion in our case report again [1], where we had raised similar questions, the answers to which are currently unavailable, and should form the focus of concerted research efforts.
Arthur M. Lam, M.D., F.R.C.P.C.
Lorri A. Lee, M.D.

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Multifactorial Etiology of Postoperative Vision Loss

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Author Information

In Reply:—

Drs. Cheng and Tempelhoff are correct that postoperative visual loss (PVL) occurs in the absence of negligence by the anesthesiologist. The origin of PVL appears to be multifactorial, and further studies are needed to define the mechanisms. Hence, we opined in the editorial that more data are needed to design strategies for prevention of this devastating complication. Because of the poorly-defined nature of this injury, whether patients should be informed of the risk preoperatively remains a matter of judgment for the individual anesthesiologist and surgeon.

Drs. Cheng and Tempelhoff think it is “indisputable” that we should routinely ask patients about their vision postoperatively. While this seems an easy procedure to implement and a worthwhile recommendation, data are lacking to mandate such a practice. Patients waking up after anesthesia may not be fully responsive, pupil signs can be erroneous, and visual fields might not be assessable. Anesthesiologists do not routinely perform fundoscopy to examine the optic nerve. In the setting of posterior ischemic optic neuropathy, the fundoscopic exam will, early on at least, likely be inconclusive. Also complicating any postoperative visual examination is the fact that symptom onset may occur anywhere from immediately postoperatively to several days later. Serial examinations would be needed. In our opinion, the inavailability of qualified personnel, the questionable cost-benefit ratio, and the low incidence of PVL render this approach, currently, generally not feasible.

The main thrust of Drs. Benumof and Mazzei's comments relate to the device used to position the head during spine surgery. Anesthesiologists are well aware of the necessity to avoid external pressure on the patients’ eyes. Benumof and Mazzei argue that the device they developed allows continuous observation of the eyes without the need to manipulate the foam cushion. Having used this headrest, we agree that it has this advantage. However, we feel that the anesthesiologist should consider also periodically palpating the eyes directly, every 5–30 min, and documenting this on the patient's operative record. With respect to the conventional square foam headrest, this device is, in fact, suitable for the majority of patients. Placing a hand under the foam to intermittently feel the eyes is easily done. That the foam has to be “peeled” or “pressed away” as Benumof and Mazzei point out, is not a major problem because the eyes are in the open portion of the headrest. Moreover, one could argue that the process of moving the foam and perhaps even lifting the head from time to time adds further assurance that the eyes

are not subjected to external pressure. In any event, we think these comments regarding the headrest distract the focus from the primary issues raised in the case report of Drs. Lee and Lam, because direct pressure is rarely the cause of postoperative visual disturbances.

We agree with Drs. Benumof and Mazzei that data on the effects of head down tilt and neosynephrine on the ocular circulation are not available. Whether it is advisable to use vasoconstrictors to maintain higher perfusion pressures to the eye in the prone position cannot currently be stated.

Steven Roth, M.D.

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limited. Previous studies in adults show that the optimal intrathecal morphine has a much wider range, and that the dose is dependent on the anticipated degree of postoperative pain for the type of operation. For example, the suggested effective dose of intrathecal morphine is 500 μg for knee surgery (2), whereas it is 50 μg for transurethral prostate resections (3). Pediatric "high" spinal morphine dose used for oncology, spinal surgery, cardiac surgery, and frontal encephalocele repair suggests a threefold range: from 10 to 30 μg/kg (4,5). In a recent unpublished pediatric study, we found that 2 μg/kg intrathecal morphine provides excellent postoperative analgesia in patients undergoing hypospadias repair. We therefore believe that pediatric effective "low" morphine dose needs to be determined independently for each type of surgery.

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In Response:

We agree with the authors (1) that different doses of morphine may be administered intrathecally. However, our study (2) simply describes our experience with the use of intrathecal morphine in a dose of 4–5 μg/kg. We have also listed the surgical procedures where this dose was administered. Larger doses of intrathecal morphine tend to have a greater incidence of side effects (3) and usually require monitoring of the patient in an intensive care unit (ICU). About 90% of the patients in our study (2) were monitored on the regular floor and the others were admitted to the ICU as per the institutional policy of admitting all neurosurgical patients to the ICU and not due to the fact that intrathecal morphine was administered. Using a uniform dose also helps to formulate the timing and dosage of rescue analgesics.

Gall et al. (3) concluded that intrathecal morphine in a dose of 5 μg/kg can be a useful adjunct in the management of postoperative pain after spine surgery (very painful) for idiopathic scoliosis.

Hypospadias surgery in our institution is almost always performed as a day surgery procedure, which makes the use of intrathecal morphine not feasible.

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Etiology of Postoperative Visual Loss Not Always as Obvious as It Appears to Be

To the Editor:

A recent case report by Roth et al. (1) described unilateral central retinal artery occlusion in a patient in a prone position with a Dupaco Opti-Gard eye shield in place to "protect the eyes." Although inadvertent mechanical compression of the globe would seem to be the most likely etiology for blindness in this case, an alternative mechanism cannot be excluded. Optic nerve perfusion pressure is equal to the mean arterial pressure minus intraocular pressure (IOP) or venous drainage pressure, whichever is greater. There was only a modest decrease in mean arterial pressure reported during the procedure, but perfusion pressure could have been substantially decreased by changes in IOP. Direct pressure upon the globe is just one potential cause of dramatic increases in IOP.

Several studies have suggested that profound (as much as 10-fold) increases in IOP during general anesthesia in patients in the prone position even when the head is secured by pins and the eyes are presumably protected from mechanical compression (2,3). The absence of increased IOP postoperatively does not reliably exclude the possibility of significant intraoperative perturbations in IOP. Although less likely, the corneal abrasion and bruising noted postoperatively could be caused if a sedated patient were rubbing at a blind eye. External compression of the globe is an infrequent cause of visual loss for patients having surgery in the prone position (4). The risk factors for postoperative visual loss have only been partially identified and when these complications occur, the etiology often cannot be precisely identified.

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