Anesthesiology 2006; 105:652–9

The American Society of Anesthesiologists Postoperative Visual Loss Registry

Analysis of 93 Spine Surgery Cases with Postoperative Visual Loss

Lorri A. Lee, M.D., Steven Roth, M.D.,† Karen L. Posner, Ph.D.,‡ Frederick W. Cheney, M.D.,§ Robert A. Caplan, M.D.,‖ Nancy J. Newman, M.D.,# Karen B. Domino, M.D., M.P.H.***

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

* Associate Professor, ** Professor, Departments of Anesthesiology and Neurological Surgery (Adjunct), ‡ Research Associate Professor, Departments of Anesthesiology and Anthropology (Adjunct), § Professor and Chair, Department of Anesthesiology, University of Washington. † Associate Professor, Department of Anesthesia and Critical Care, University of Chicago, Chicago, Illinois. ‖ Clinical Professor, Department of Anesthesiology, University of Washington, Staff Anesthesiologist, Virginia Mason Medical Center, Seattle, Washington # Professor, Departments of Ophthalmology, Neurology, and Neurological Surgery, Emory University, Atlanta, Georgia.

Received from the Department of Anesthesiology, University of Washington, Seattle, Washington. Submitted for publication January 6, 2006. Accepted for publication March 24, 2006. Supported by the American Society of Anesthesiologists, Park Ridge, Illinois, All opinions expressed are those of the authors and do not reflect the policy of the American Society of Anesthesiologists.

Address correspondence to Dr. Lee: Department of Anesthesiology, Harborview Medical Center, Box 359724, 325 Ninth Avenue, Seattle, Washington 98104. lorlee@u.washington.edu. Individual article reprints may be accessed at no charge through the Journal Web site, www.anesthesiology.org.

†† 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.

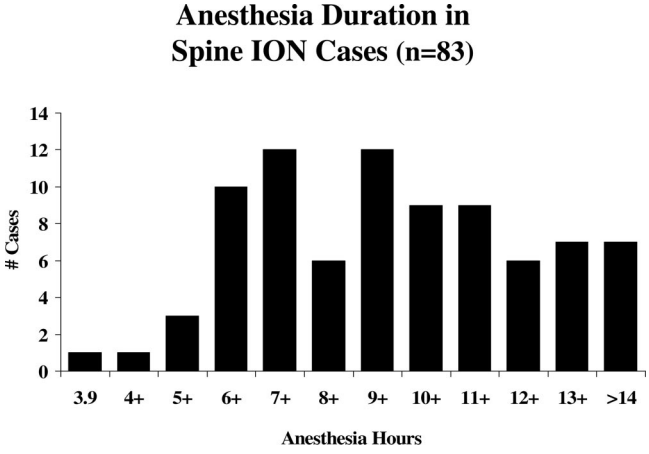

Fig. 1. Anesthetic duration for 83 spine cases associated with ischemic optic neuropathy (ION). The majority of cases (94%) were 6 h or longer in duration.

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

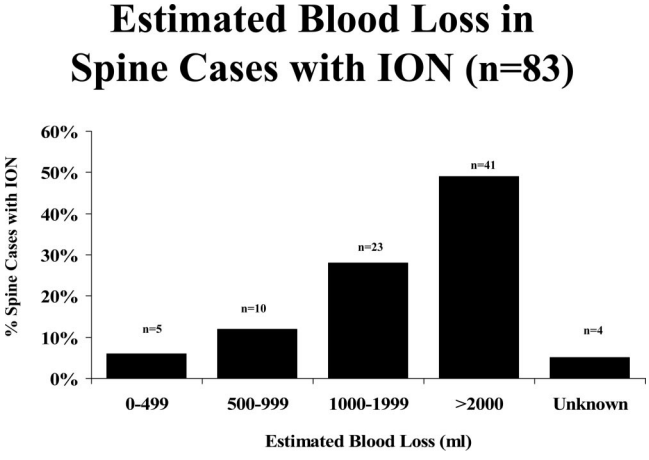

Fig. 2. Estimated blood loss for 83 spine cases associated with ischemic optic neuropathy (ION). In the majority of cases (82%), blood loss was 1,000 ml or greater.

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.

The authors thank Nayak L. Polissar, Ph.D. (Mountain-Whisper-Light Statistical Consulting, Seattle, Washington), for his paid consultation for the statistical analysis of agreement of data between duplicate submissions. The authors thank Lynn Akerlund for her contributions as project coordinator and for her secretarial assistance, and John Campos, M.A., for technical assistance with creation and maintenance of the database. They are members of the Closed Claims Project research staff in the Department of Anesthesiology, University of Washington, Seattle, Washington. The authors also thank the physicians and patients who submitted cases to the American Society of Anesthesiologists Postoperative Visual Loss Registry for their dedication to furthering research in this area.

References

1. Stevens WR, Glazer PA, Kelley SD, Lietman TM, Bradford DS: Ophthalmic complications after spinal surgery. Spine 1997; 22:1319–24
2. Shaw PJ, Bates D, Cartlidge NE, Heaviside D, French JM, Julian DG, Shaw DA: Neuro-ophthalmological complications of coronary artery bypass graft surgery. Acta Neurol Scand 1987; 76:1–7
3. Katz DM, Trobe JD, Cornblath WT, Kline LB: Ischemic optic neuropathy after lumbar spine surgery. Arch Ophthalmol 1994; 112:925–31
4. Myers MA, Hamilton SR, Bogosian AJ, Smith CH, Wagner TA: Visual loss as a complication of spine surgery: A review of 37 cases. Spine 1997; 22:1325–9
5. Anonymous: Characteristics of patients with nonarteritic anterior ischemic optic neuropathy eligible for the Ischemic Optic Neuropathy Decompression Trial. Arch Ophthalmol 1996; 114:1366–74
6. Hayreh SS, Zimmerman MB, Podhajsky P, Alward WL: Nocturnal arterial hypotension and its role in optic nerve head and ocular ischemic disorders. Am J Ophthalmol 1994; 117:603–64
7. Pomeranz HD, Bhavsar AR: Nonarteritic ischemic optic neuropathy developing soon after use of sildenafil (Viagra): A report of seven new cases. J Neuroophthalmol 2005; 25:9–13
8. Deyo RA, Gray DT, Kreuter W, Mirza S, Martin BI: United States trends in lumbar fusion surgery for degenerative conditions. Spine 2005; 30:1441–5
9. Ho VT, Newman NJ, Song S, Ksiazek S, Roth S: Ischemic optic neuropathy following spine surgery. J Neurosurg Anesthesiol 2005; 17:38–44
10. Buono LM, Foroozan R: Perioperative posterior ischemic optic neuropathy: Review of the literature. Surv Ophthalmol 2005; 50:15–26
11. Chang SH, Miller NR: The incidence of vision loss due to perioperative ischemic optic neuropathy associated with spine surgery: The Johns Hopkins Hospital Experience. Spine 2005; 30:1299–302
12. Warner MA, Warner ME, Martin JT: Ulnar neuropathy: Incidence, outcome, and risk factors in sedated or anesthetized patients. ANESTHESIOLOGY 1994; 81:1332–40
13. Jover T, Tanaka H, Calderone A, Oguro K, Bennett MV, Etgen AM, Zukin RS: Estrogen protects against global ischemia-induced neuronal death and prevents activation of apoptotic signaling cascades in the hippocampal CA1. J Neurosci 2002; 22:2115–24
14. Balazsi AG, Rootman J, Drance SM, Schulzer M, Douglas GR: The effect of age on the nerve fiber population of the human optic nerve. Am J Ophthalmol 1984; 97:760–6
15. Mikelberg FS, Drance SM, Schulzer M, Yidegiligne HM, Weis MM: The normal human optic nerve: Axon count and axon diameter distribution. Ophthalmology 1989; 96:1325–8
16. Hollenhorst RW, Svien HJ, Benoit CF: Unilateral blindness occurring during anesthesia for neurosurgical operations. AMA Arch Ophth 1954; 52:819–80
17. McNeill TW, DeWald RL, Kuo KN, Bennett EJ, Salem MR: Controlled hypotensive anesthesia in scoliosis surgery. J Bone Joint Surg Am 1974; 56: 1167–72
18. Malcolm-Smith NA, McMaster MJ: The use of induced hypotension to control bleeding during posterior fusion for scoliosis. J Bone Joint Surg Br 1983; 65:255–8
19. Patel NJ, Patel BS, Paskin S, Laufer S: Induced moderate hypotensive anesthesia for spinal fusion and Harrington-rod instrumentation. J Bone Joint Surg Am 1985; 67:1384–7
20. Larsen FS, Olsen KS, Hansen BA, Paulson OB, Knudsen GM: Transcranial Doppler is valid for determination of the lower limit of cerebral blood flow autoregulation. Stroke 1994; 25:1985–8
21. Drummond JC: The lower limit of autoregulation: time to revise our thinking? ANESTHESIOLOGY 1997; 86:1431–3
22. Pillunat LE, Anderson DR, Knighton RW, Joos KM, Feuer WJ: Autoregulation of human optic nerve head circulation in response to increased intraocular pressure. Exp Eye Res 1997; 64:737–44
23. Nitta A, Kimura Y, Takayma H: The blood flow in post-laminar optic nerve in monkeys was dependent on intraocular pressure level. Nippon Ganka Gakkai Zasshi 1989; 93:206–14
24. Weinstein JM, Duckrow RB, Beard D, Brennan RW: Regional optic nerve blood flow and its autoregulation. Invest Ophthalmol Vis Sci 1983; 24:1559–65

25. Lam AK, Douthwaite WA: Does the change of anterior chamber depth or/and episcleral venous pressure cause intraocular pressure change in postural variation? Optom Vis Sci 1997; 74:664–7

26. Cheng MA, Todorov A, Tempelhoff R, McHugh T, Crowder CM, Lauryssen C: The effect of prone positioning on intraocular pressure in anesthetized patients. ANESTHESIOLOGY 2001; 95:1351–5

27. Ozcan MS, Praetel C, Bhatti MT, Gravenstein N, Mahla ME, Seubert CN: The effect of body inclination during prone positioning on intraocular pressure in awake volunteers: A comparison of two operating tables. Anesth Analg 2004; 99:1152–8

28. Hayreh SS: The blood supply of the optic nerve head and the evaluation of it: Myth and reality. Prog Retin Eye Res 2001; 20:563–93

29. Pazos GA, Leonard DW, Blice J, Thompson DH: Blindness after bilateral neck dissection: Case report and review. Am J Otolaryngol 1999; 20:340–5

30. Weiss KL, Wax MK, Haydon RC 3rd, Kaufman HH, Hurst MK: Intracranial pressure changes during bilateral radical neck dissections. Head Neck 1993; 15:546–52

31. Johnson MW, Kincaid MC, Trobe JD: Bilateral retrobulbar optic nerve infarctions after blood loss and hypotension: A clinicopathologic case study. Ophthalmology 1987; 94:1577–84

32. Marks SC, Jaques DA, Hirata RM, Saunders JR Jr: Blindness following bilateral radical neck dissection. Head Neck 1990; 12:342–5

33. Nawa Y, Jaques JD, Miller NR, Palermo RA, Green WR: Bilateral posterior optic neuropathy after bilateral radical neck dissection and hypotension. Graefes Arch Clin Exp Ophthalmol 1992; 230:301–8

Anesthesiology 2007; 106:1249

What Happened to the Old Visual Evoked Potential Monitoring?

To the Editor:—I read with interest the results of the American Society of Anesthesiologists Postoperative Visual Loss Registry and the analysis of the 93 spine surgery cases with postoperative visual loss.[1] Striking to me was that the majority of the complications happened in settings that were thought safe in the past. It has long been taught that prevention of direct ocular pressure, severe hypoxia, anemia, and hypotension prevent blindness in the majority of patients undergoing prone spine surgery. This report and analysis of data showed that direct ocular pressure contributed to only a small percentage of the documented cases, and that blindness occurred over a wide range of systolic pressure, homodynamics, and hemoglobin concentrations. That led me to conclude that while prevention is the best cure for this problem, best prevention is not currently understood; it raised in my mind the question of intraoperative visual system monitoring. Today, we routinely use pulse oximetry, capnography, and even processed electroencephalographic monitoring to identify and promptly correct hypoxemia, ventilatory inadequacy, and awareness. Isn't it logical that in high-risk cases where blindness is possible that we should be monitoring the patient intraoperatively to identify early retinal changes that could correlate with this tragic event and try to prevent that outcome?

Intraoperative retinal monitoring through visual evoked potentials, with the aim of preserving visual fields, has been used successfully in many cases such as intracranial surgeries,[2] occipital corticectomy for epilepsy,[3] functional endoscopic sinus surgery,[4] optic nerve function surgery, and other surgeries involving the visual pathway. Currently, the use of the visual evoked potential is limited and not routinely practiced in spine surgery performed in prone positioning. It seems obvious that this modality should be used more frequently and even routinely in all prone spine surgeries.

I am aware of conflicting reports about the usefulness of this monitoring modality, but I believe that our reading and correlation of retinal evoked potentials will improve as the monitoring becomes routine. I hope that the future will focus on improving monitoring of the visual evoked potential, perhaps in a form as simple as bispectral monitoring (such as the Bispectral Index®; Aspect Medical Systems Inc., Norwood, MA). Such monitoring may allow us to accurately detect early, reversible damage to the visual pathway and enable us to prevent permanent problems. This would be in the best tradition of anesthesiology.

Adballah I. Kabbara M.D., Louis Stokes Cleveland Department of Veterans Affairs Medical Center, Cleveland, Ohio. draikabbara@yahoo.com

References

1. Lee LA, Roth S, Posner KL, Cheney FW, Caplan RA, Newman NJ, Domino KB: The American Society of Anesthesiologists Postoperative Visual Loss Registry: Analysis of 93 spine surgery cases with postoperative visual loss. ANESTHESIOLOGY 2006; 105:652–9

2. Hidajat RR, McLay JL, Goode DH, Hidayat JR: The value of VEP in the diagnosis and post-operative monitoring of meningioma. Doc Ophthalmol 2006; 113:165–9

3. Curatolo JM, Macdonell RA, Berkovic SF, Fabinyi GC: Intraoperative monitoring to preserve central visual fields during occipital corticectomy for epilepsy. J Clin Neurosci 2000; 7:234–7

4. Herzon GD, Zealear DL: Intraoperative monitoring of the visual evoked potential during endoscopic sinus surgery. Otolaryngol Head Neck Surg 1994; 111:575–9

(Accepted for publication February 8, 2007.)

Anesthesiology 2007; 106:1249

Excessive Crystalloid Infusion May Contribute to Ischemic Optic Neuropathy

To the Editor:—It is unfortunate that Drs. Lee *et al.*[1] and Warner[2] feel compelled to conclude that blindness may be an inevitable consequence of prolonged spine surgery in the prone position, and that patients should be warned of that possibility. While perhaps correct, my experience in supervising many hundreds of such cases without this complication leads me to believe that it is preventable. Although briefly considered by Dr. Lee *et al.* in the Discussion section, sufficient attention was not focused on the large average volume of crystalloid solution (9.7 ± 4.7 l) infused in the 83 patients who developed ischemic optic neuropathy. This volume of infusion is far in excess of what is necessary for maintenance of either blood pressure or urine output. In addition, it has a serious negative impact on the hematocrit, as well as promoting edema of the orbs and optic nerves. Although the etiology of blindness may be multifactorial, as anesthesiologists we must critically assess those aspects of care over which we have control. Limiting crystalloid administration, avoiding severe anemia (hematocrit < 26), and limiting the duration of controlled hypotension, if used, to the dissection period only (not the instrumentation period) are all controllable. I would urge anesthesiologists to limit crystalloid volume in prone spine surgical cases to no more than 40 ml/kg (approximately 3 l in adults) for the entire operative procedure regardless of duration. If additional fluid is deemed necessary, it should be hetastarch (not to exceed 20 ml/kg), albumin, or blood. If necessary, a low-dose dopamine infusion can be used to support circulation and improve urine output. Finally, urine output should not be the benchmark for fluid requirements in these patients. Urine output is commonly diminished while patients are in the prone position for reasons that have not been documented. Diminished urine output in this setting does not lead to renal insufficiency postoperatively.

C. Philip Larson, Jr., M.D., David Geffen School of Medicine at UCLA, Los Angeles, California. plarson@ucla.edu

References

1. Lee LA, Roth S, Postner KL, Cheney FW, Caplan RA, Newman NJ, Domino KB: The American Society of Anesthesiologists Postoperative Visual Loss Registry: Analysis of 93 spine surgery cases with postoperative visual loss. ANESTHESIOLOGY 2006; 105:652–9

2. Warner MA: Postoperative visual loss: Experts, data, and practice. ANESTHESIOLOGY 2006; 105:641–2

(Accepted for publication February 8, 2007.)

Anesthesiology 2007; 106:1250–1

Visual Loss after Spinal Surgery

To the Editor:—The American Society of Anesthesiologists (ASA) Closed Claims Project has provided valuable information regarding risks and potential etiologies of untoward events related to the practice of anesthesia.[1] The ASA Registry for Postoperative Visual Loss arose from some of the same concerns as did the Closed Claims Project: an attempt to understand problems that have become medical–legal issues and to provide better care for our patients.[2] Lee *et al.*[3] have provided a valuable service in documenting data associated with this rare and devastating adverse event. Their report follows closely the recent ASA "Practice Advisory for Perioperative Visual Loss Associated with Spine Surgery."[4] Inasmuch as randomized prospective clinical trials to discern etiology and efficacy of the suggested therapies of increasing blood pressure and hemoglobin concentration would not be feasible, owing to the low incidence, estimated to be approximately 0.03–0.1% for ischemic optic neuropathy (ION)[5,6] (a reduction of 25% would require a study of approximately 200,000–750,000 patients per group), other methodologies are needed to assess possible etiologies and therapies. As pointed out by Lee *et al.*, unfortunately, information regarding the total number of surgical procedures represented by the reports in their database is not available. The registry could be improved by asking those who provide case reports to also indicate the number of similar operations performed during a several-year period (a short period would produce an artificially estimated high incidence). Even this, however, would overstate the incidence, because this complication has never been encountered by most spine surgeons,[7] and likely most institutions. Of greater concern is the recommendation contained in the report and the absence of other recommendations.

We question the recommendation regarding routine preoperative discussion of the possibility of postoperative visual loss, given the exceedingly low incidence. Complications of such low incidence[5,6] (*e.g.*, masseter muscle rigidity/malignant hyperthermia[8,9]) are not routinely discussed, and the rarity of ION makes it unlikely that discussion would be a relevant consideration in whether the patient elected to proceed. In addition, once mentioned, little can be said regarding prevention or therapy, inasmuch as the etiologies of anterior ION and posterior ION are uncertain, and prophylactic and therapeutic maneuvers are of unproven value.

Of interest are the surprising data that the patients' eyes were documented as having been checked in only 51% of cases of ION (frequency not given) and in just 6 of 10 cases of central retinal artery occlusion (frequency of between every 30 min and only once during the entire procedure), which is widely regarded as being caused by direct trauma or pressure applied to the eye. Our spine anesthesia team was established in 1991, and our routine care includes checking the eyes every 15 min of every patient in the prone position. We previously reported 7 cases of visual disturbances after 3,450 spinal surgeries, including four IONs, one central retinal venous thrombosis, and no central retinal arterial thromboses.[5] We are surprised that the registry report contained no recommendation regarding the advisability of frequent checks for absence of direct pressure on the patient's eyes: something that is easily performed, is of no cost, and makes sense physiologically, although of unproven efficacy in preventing central retinal artery thrombosis. In addition, we recommend a simple, quick test of crude visual function and visual fields (*e.g.*, tell how many fingers, and when they can be seen as they are moved from the periphery to a central position) as soon as possible in the immediate postoperative period. The ASA practice advisory[4] and Myers *et al.*[7] in their evaluation of a series of 37 cases of visual loss after spinal surgery also recommend an early postoperative assessment of visual function. This allows for rapid consultation, documentation of the timing of the event, and institution of any recommended, although unproven, therapy.

The report provides a good discussion regarding possible etiologies of ION, including increased venous pressure and trapping of the optic nerve owing to increased interstitial fluid accumulation and thus pressure in an enclosed bony canal. It is possible that the latter issue may also decrease arterial blood flow. As discussed in the report, placing a patient prone in a position with the head slightly elevated decreases intraocular venous pressure. We practice and recommend this, as does the ASA practice advisory for "high-risk patients."[4] In addition, we also limit the volume of crystalloid solution to reduce the possibility of increased interstitial fluid and pressure, although, admittedly, neither this nor the slightly head-up tilted position is a proven efficacious prophylactic therapy.

We were surprised that the report did not consider patients' fraction of inspired oxygen or arterial oxygen tension. We have shown that anemia-induced neurologic deficits in healthy people can be reversed by increasing arterial oxygen concentration.[10] We are further concerned that both Lee *et al.* and the ASA Task Force suggest that protracted surgery and amount of blood loss are risk factors for the development of postoperative visual loss. Neither is physiologically grounded. A more sensible assessment, in the absence of a validated monitor for visual function during anesthesia, would focus on blood loss replacement and maintenance of normovolemia, rather than the volume of loss itself, and the duration of factors that might influence inadequate perfusion of the ophthalmic vasculature, rather than the duration of the surgery. The latter may be a poorly correlating surrogate for hypovolemia/hypoperfusion and may appear erroneously as a univariate factor in a database of a limited number of events. These might also be surrogates for the intravenous infusion of substantial amounts of salt solutions, with the potential adverse action noted above. Anesthesiologists and surgeons should work together to minimize potential contributing factors to this devastating complication; however, in the absence of definitive data, the Task Force's suggestion to alter accepted surgical practice[11,12] is questionable.

Richard B. Weiskopf, M.D.,* John Feiner, M.D., Jeremy Lieberman, M.D., Serena S. Hu, M.D. *University of California, San Francisco, California. rwes@novonordisk.com

At the time of the writing and acceptance of this letter, Dr. Weiskopf was an employee of Novo Nordisk A/S, Bagsvaerd, Denmark. All authors contributed to the writing of this letter.

References

1. Cheney FW: The American Society of Anesthesiologists Closed Claims Project: What have we learned, how has it affected practice, and how will it affect practice in the future? Anesthesiology 1999; 91:552–6

2. Warner MA: Postoperative visual loss: Experts, data, and practice. Anesthesiology 2006; 105:641–2

3. Lee LA, Roth S, Posner KL, Cheney FW, Caplan RA, Newman NJ, Domino KB: The American Society of Anesthesiologists Postoperative Visual Loss Registry: Analysis of 93 spine surgery cases with postoperative visual loss. Anesthesiology 2006; 105:652–9

4. Practice advisory for perioperative visual loss associated with spine surgery: A report by the American Society of Anesthesiologists Task Force on Perioperative Blindness. Anesthesiology 2006;104: 1319–28.

5. Stevens WR, Glazer PA, Kelley SD, Lietman TM, Bradford DS: Ophthalmic complications after spinal surgery. Spine 1997; 22:1319–24

6. Chang SH, Miller NR: The incidence of vision loss due to perioperative ischemic optic neuropathy associated with spine surgery: The Johns Hopkins Hospital Experience. Spine 2005; 30:1299–302

7. Myers MA, Hamilton SR, Bogosian AJ, Smith CH, Wagner TA: Visual loss as a complication of spine surgery: A review of 37 cases. Spine 1997; 22:1325–9

8. Monnier N, Krivosic-Horber R, Payen JF, Kozak-Ribbens G, Nivoche Y,

Adnet P, Reyford H, Lunardi J: Presence of two different genetic traits in malignant hyperthermia families: Implication for genetic analysis, diagnosis, and incidence of malignant hyperthermia susceptibility. ANESTHESIOLOGY 2002; 97: 1067–74

9. Ording H: Incidence of malignant hyperthermia in Denmark. Anesth Analg 1985; 64:700–4

10. Weiskopf RB, Feiner J, Hopf HW, Viele MK, Watson J, Kramer JH, Ho R, Toy P: Oxygen reverses deficits of cognitive function and memory and increased heart rate induced by acute severe isovolemic anemia. ANESTHESIOLOGY 2002; 96:871–7

11. Bradford DS, Boachie-Adjei O: One-stage anterior and posterior hemivertebral resection and arthrodesis for congenital scoliosis. J Bone Joint Surg Am 1990; 72:536–40

12. Dick J, Boachie-Adjei O, Wilson M: One-stage *versus* two-stage anterior and posterior spinal reconstruction in adults. Comparison of outcomes including nutritional status, complications rates, hospital costs, and other factors. Spine 1992; 17:S310–6

(Accepted for publication February 8, 2007.)

Anesthesiology 2007; 106:1251

In Reply:—In response to Dr. Larson's rather dogmatic conclusions on how to avoid perioperative ischemic optic neuropathy, I am pleased that he has never personally experienced this complication in one of his patients. His observation drives home the primary point of the report by Dr. Lee *et al.*[1] and my editorial[2]: There are too few of these complications at this time to scientifically deduce causative risk factors. Quite simply, it is not logistically or financially possible at this time to prospectively search for causative risk factors of this devastating complication as it occurs in patients undergoing spine surgery while positioned prone.

Therefore, it is difficult to understand what data Dr. Larson uses as a basis for his recommendations. There are no data to suggest that limiting crystalloid administration to less than 40 ml/kg regardless of duration of the surgical procedure impacts ischemic optic neuropathy (negatively or positively). The same can be said for his suppositions about hematocrit levels of less than 26 and limiting durations of controlled hypotension to only the dissection period of spine surgery. Data from multiple studies document that many patients who have Dr. Larson's "risk factors" do not develop ischemic optic neuropathy—and many who develop ischemic optic neuropathy receive crystalloid volumes of less than 40 ml/kg, have hematocrits intraoperatively well above 26, and are provided care without the use of controlled hypotension. In short, there is no scientific reasoning to justify Dr. Larson's strongly worded, unsupportable recommendations.

Dr. Weiskopf raises two points to which I would like to respond. First, he speculates that periodic intraoperative checks of the eyes for absence of direct pressure on patients' eyes may be useful in preventing central retinal artery thrombosis. His spine team evidently established periodic intraoperative eye checks for all prone-positioned spine surgery patients and found that none of their 3,450 patients developed this complication.[3] However, as he notes, the frequency of this event is very low. It is, therefore, impossible to draw any conclusion or even inference that his team's eye checks had anything to do with the outcomes that their patients experienced. Regarding the use of eye checks, it is disappointing to find that 6 of the 10 patients with central retinal artery occlusion in the America Society of Anesthesiologists Visual Loss Registry had at least one eye check during their procedures.[1] In those 6 patients, eye checks apparently did not prevent this problem from occurring. Therefore, should eye checks be done? Intuitively, yes—they are certainly cheap and easy. However, there are no data showing that they are effective in reducing the frequency or severity of central retinal artery occlusion.

Dr. Weiskopf also indirectly suggests that increased inspired oxygen concentrations, resulting in elevated arterial oxygen tension, may be helpful in decreasing the frequency of perioperative vision loss. He cites an excellent study that he and colleagues performed in volunteers who were made acutely anemic, finding that increased inspired oxygen concentrations reversed the negative cognitive effects of the anemia.[4] Ironically, hyperoxia has an autoregulation-related vasoconstrictive effect on the end-retinal arterioles of the central retinal artery, reducing both the diameter of the arterioles and also their blood flow and velocity.[5] It is not clear what impact this finding has on blood flow to the various regions of optic nerves, but increasing inspired oxygen concentrations may not be as helpful as he seems to suggest.

Mark A. Warner, M.D., Mayo Clinic, Rochester, Minnesota. warner.mark@mayo.edu

References

1. Lee LA, Roth S, Posner KL, Cheney FW, Caplan RA, Newman NJ, Domino KB: The American Society of Anesthesiologists Postoperative Visual Loss Registry: Analysis of 93 spine surgery cases with postoperative visual loss. ANESTHESIOLOGY 2006; 105:652–9

2. Warner MA: Postoperative visual loss: Experts, data, and practice. ANESTHESIOLOGY 2006; 105:641–2

3. Stevens WR, Glazer PA, Kelly SD, Lietman TM, Bradford DS: Ophthalmic complications after spinal surgery. Spine 1997; 22:1319–24

4. Weiskopf RB, Feiner J, Hopf HW, Viele MK, Watson J, Kramer JH, Ho R, Toy P. Oxygen reverses deficits of cognitive function and memory and increased heart rate induced by acute severe isovolemic anemia. ANESTHESIOLOGY 2002; 96:871–7

5. Rose PA, Hudson C: Comparison of retinal arteriolar and venular variability in healthy subjects. Microvasc Res 2007; 73:35–8

(Accepted for publication February 8, 2007.)

Anesthesiology 2007; 106:1251–2

In Reply:—We gratefully acknowledge the interest that Drs. Kabbara, Larson, and Weiskopf *et al.* have shown in our article on spine surgery and postoperative visual loss (POVL).[1] It is only through the continued interest and investment of time and resources by anesthesiologists, ophthalmologists, and surgeons that we will develop preventative strategies and/or treatment for this devastating perioperative complication. These letters provide an opportunity to discuss and expand on topics that space limitations would not allow in the original article.

Dr. Kabbara makes an insightful deduction in noting that our current lack of proven risk factors for ischemic optic neuropathy (ION), and its possible multifactorial etiology, would make an intraoperative monitor of optic nerve function a logical means to prevent ION. Unfortunately, previous studies have demonstrated that anesthetics diminish or ablate visual evoked potentials, making their intraoperative reliability poor.[2,3] Additional technical problems include poor sensitivity of the light-flash

Dr. Roth has received financial compensation for providing expert witness testimony on behalf of patients, hospitals, and physicians in cases of perioperative visual loss.

as opposed to pattern-evoked potentials. Further research and technical advances will be required before the sensitivity and specificity of this monitor for detecting optic nerve dysfunction are acceptable for routine clinical use. Moreover, some patients do not develop clinical visual deficits until several days after surgery, and it is unclear how useful an "intraoperative" optic nerve monitor would be in these situations.

Dr. Larson summarized his personal experience over many years and his personal beliefs about cause-and-effect relations regarding ION. Unfortunately, there is no way to validate the summary statements and beliefs derived from his anecdotal experience. Moreover, our clinical experience makes us concerned that limiting fluids to a specific amount, without regard to urine output or blood loss, may lead to underresuscitation and increase the risk of organ failure.

Although the American Society of Anesthesiologists POVL Registry has provided detailed descriptive characteristics of patients who develop ION after major spine surgery, it cannot be used to determine risk factors because there are no denominator data and no unaffected patients for comparison. Because of the nature of complex spine surgery, it is possible that patients who do not develop ION after major spine surgery have received similar amounts of crystalloid. The American Society of Anesthesiologists recently reviewed the scientific evidence and expert opinion regarding the anesthetic management to reduce the risk of perioperative visual loss in prone spine surgery. Because of the lack of scientific literature, an advisory, not a guideline, resulted. Although the advisory recommended the use of both colloid and crystalloid, specific amounts of these solutions could not be recommended because of the absence of any evidence-based literature.[4]

Dr. Weiskopf *et al.*'s point about frequent eye checks to prevent central retinal artery occlusion from globe compression is appreciated. Because the focus of our article was ION, we did not explicitly state, but do completely agree, that frequent eye checks during major prone spine surgery are of unquestionable value to prevent globe compression. Data on inspired oxygen concentration and arterial oxygen were not collected and therefore could not be examined with respect to anemia. The clinical use of high inspired oxygen concentration in the potential presence of ischemia remains controversial because of theoretical risks of reactive oxygen species tissue damage.[5] Other details regarding clinical care of spine patients at the authors' institution were noted, including limitation of crystalloid infusion, but again, the benefit of this practice with respect to prevention of ION cannot be validated based on the literature.

We agree with Dr. Weiskopf that consenting patients undergoing major spine surgery for the risk of POVL is challenging, but our experience reading closed claims files for POVL has repeatedly revealed that patients believe that they should have been consented for the risk of blindness associated with major spine surgery. The fact that the authors have made four significant intraoperative interventions aimed at preventing POVL demonstrates that it is of great concern to anesthesiologists and surgeons. Rest assured that it is of even more concern for patients. There is no widely accepted threshold of incidence of complications to preclude discussion of risks. Most states use the "reasonable patient" standard for consent as described by O'Leary, in which a physician is required "to disclose information that a reasonable patient under similar circumstances would want to know to make an informed decision."[6] These risks would include common side effects and complications of low severity, and those that are less common, but with significant impact, such as blindness.

The data are clear regarding the types of spine cases in which ION occurs: prolonged operations in the prone position with large blood loss.[1] We, like others, speculate that the physiologic basis for these findings may have more to do with the prone position in which venous pressures are elevated and the time that it takes for optic nerve axons to become dysfunctional. Large blood loss increases the potential for hypovolemia and the occurrence of anemia, and increases fluid administration and transfusion of blood products, all of which may affect oxygen delivery to the tissues. However, any theory of causation for ION remains to be proven. We agree with the authors that maintenance of normovolemia is important and would be useful data to analyze, but this assessment is subject to varied interpretation, particularly in the prone position. This information would have to be collected in a prospective fashion with rigid criteria and uniform monitoring. We would like to clarify that we did not advocate a change in surgical practice, except for consent, without a randomized controlled trial comparing the effects of staged surgery for major spine procedures with single-stage surgery, because this alternative also has the potential for significant morbidity. We agree that surgeons and anesthesiologists must work together to minimize potential contributing factors to the development of POVL for our patients. The data demonstrate that two of these factors are prolonged spine surgery in the prone position and large blood loss.[1]

Finally, we would like to reiterate that the clinical phenomenon of perioperative ION occurs at such a low frequency (highest incidence reported to date 0.1%)[7] that prospective clinical studies randomizing patients to treatment arms would require a multicenter, long-term, costly study. Currently, there is no evidence-based medicine to support any causative (or preventative) statements regarding the development of ION. Because of the low incidence of ION, and the predominance of these cases in spine operations of 6 h or longer and blood loss of 1,000 ml or greater,[1] most anesthesiologists are fortunate enough to have never encountered this complication, regardless of their anesthetic management. However, good fortune should not be equated with best practice when the etiology and prevention of ION remain unproven.

Lorri A. Lee, M.D.,* Steven Roth, M.D., Karen L. Posner, Ph.D., Frederick W. Cheney, M.D., Robert A. Caplan M.D., Nancy J. Newman, M.D., Karen B. Domino, M.D., M.P.H. *University of Washington, Seattle, Washington. lorlee@u.washington.edu

References

1. Lee LA, Roth S, Posner KL, Cheney FW, Caplan RA, Newman NJ, Domino KB. The American Society of Anesthesiologists Postoperative Visual Loss Registry: Analysis of 93 spine surgery cases. ANESTHESIOLOGY 2006; 105:652–9

2. Cedzich C, Schramm J, Mengedoht CF, Fahlbusch R: Factors that limit the use of flash visual evoked potentials for surgical monitoring. Electroencephalogr Clin Neurophysiol 1988; 71:142–5

3. Tenenbein PK, Lam AM, Lee LA: Effects of sevoflurane and propofol on flash visual evoked potentials (abstract). ANESTHESIOLOGY 2006; 105:A196

4. Practice advisory for perioperative visual loss associated with spine surgery: A report by the American Society of Anesthesiologists Task Force on Perioperative Blindness. ANESTHESIOLOGY 2006; 104:1319–28

5. Liu S, Liu W, Doing W, Miyake M, Rosenber GA, Liu KJ. Electron paramagnetic resonance-guided normobaric hyperoxia treatment protects the brain penumbral oxygenation in a rat model of transient focal cerebral ischemia. J Cereb Blood Flow Metabol 2006; 26:1274–84

6. O'Leary CE. Informed consent for anesthesia: Has the time come for a separate written consent document? ASA Newsletter 2006; 70 (6)

7. Stevens WR, Glazer PA, Kelley SD, Lietman TM, Bradford DS: Ophthalmic complications after spinal surgery. Spine 1997; 22:1319–24

(Accepted for publication February 8, 2007.)