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# Perioperative Risk Factors for Posterior Ischemic Optic Neuropathy

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**BACKGROUND:** Infarction of the optic nerve posterior to the lamina cribrosa, called posterior ischemic optic neuropathy (PION), is a condition that can result in profound bilateral blindness. Cases of PION treated at this institution and those described in the literature were analyzed to identify clinical features that profile those individuals at risk of PION in an attempt to identify major contributing factors that could be addressed prophylactically to enable effective prevention.

**STUDY DESIGN:** Salient clinical features in seven cases of PION diagnosed at the Doheny Eye Institute between 1989 and 1998 are compared with 46 cases of PION reported in the literature.

**RESULTS:** In the Doheny series there were six men and one woman aged 12 to 66 years (mean, 47 years). Five patients were status-post spine surgery, one was status-post knee surgery, and one had a bleeding stomach ulcer. Vision loss was simultaneously bilateral in six of seven patients (85.7%) and was apparent immediately after surgery. There were no abnormal retinal or choroidal findings including diabetic retinopathy, in any of the patients. Notable contributing factors were blood loss in all seven patients, ranging from 2,000 to 16,000 mL, with a drop in hematocrit of 9.5% to 19% (mean, 14%), and intraoperative systemic hypotension in all patients. Facial edema was a factor in three of six spine surgery patients (50%). Patients reported in the literature had a mean age of 50 years and were also predominantly men (34 of 46, 74%) who underwent spine surgery (30 of 46, 65.2%).

**CONCLUSIONS:** Middle-aged men undergoing spine surgery with prolonged intraoperative hypotension and postoperative anemia and facial swelling are at risk of developing PION from hypovolemic hypotension. Avoiding or immediately correcting these contributory factors can reduce the incidence of PION. (J Am Coll Surg 2002;194:705–710. © 2002 by the American College of Surgeons)

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The human optic nerve contains 1.2 million myelinated nerve fibers that arise from cell bodies in the ganglion cell layer of the retina and first synapse at the lateral geniculate nucleus. Ischemia can profoundly damage these fibers by inducing axonal swelling from impaired axoplasmic transport.<sup>1</sup> Posterior ischemic optic neuropathy (PION) is a rare event that follows infarction of the posterior portion of the optic nerve and manifests with a variety of clinical characteristics. Patients of all ages can be affected and the condition is often of sudden onset with bilateral involvement. Visual impairment is most often very severe because the optic nerve swells as a con-

sequence of ischemia. Posterior extension of swelling along the nerve involves the portion of the optic nerve within the sphenoid optic canal, where the nerve is encased in bony, nonelastic structures. Anterior extension causes swelling in the rigid confines of the scleral canal. In each circumstance, axonal compression causes profound optic nerve damage.

A better understanding of the risk factors that predispose to this devastating condition can help prevent its occurrence and stimulate development of new avenues of therapy. To identify risk factors associated with PION, we analyzed the clinical characteristics of 7 patients with PION in our institution and compared the findings with 46 cases of PION reported in the literature.

## METHODS

### Case reports

Patient 1: A 66-year-old man with diabetes and hypertension underwent partial L3 and total L4-5 laminectomy.

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tomy, bilateral L3-4 and L5, S1 medial facetectomy, bilateral foraminectomy and decompression of the L4-5 and S1 roots, and segmental fixation of the lumbar spine through transverse process fusion. The surgical procedure lasted 5 hours, during which time the blood pressure was kept between 140/70 mmHg and 120/50 mmHg. The estimated blood loss was 2,400 mL and the hematocrit dropped from 41.0% preoperatively to 31.5% postoperatively. A Wilson frame was used during the operation, and the patient had postoperative generalized facial swelling.

During the first two to three postoperative days, the patient complained of progressive, binocular visual loss. The initial fundus examination was unremarkable to the attending internist. Three months later there was inferior sectoral atrophy of the disk in the right eye and complete optic atrophy in the left eye; intraocular pressure and retina were unremarkable. Visual acuity deteriorated to 20/50 in the right eye and no light perception in the left eye.

Patient 2: A 43-year old obese man underwent bilateral L3-4 and L4-5 laminectomy, partial facetectomy of L3-5 for nerve root decompression with bilateral pedicle screw implementation, and bilateral transverse process fusion from L3 through L5. The procedure lasted approximately 8.5 hours, and the estimated blood loss was 2,000 mL. The blood pressure was kept at 100/40-60 mmHg for 6.5 hours. During the operation, a Wilson frame was used, and severe generalized facial swelling was noted after the operation. Postoperatively, the hematocrit decreased from a preoperative value of 43.2% to 31.0%.

Immediately on recovery from general anesthesia, the patient complained of bilateral visual loss. Intraocular pressure and fundus examination were initially unremarkable, but 8 months later showed temporal pallor in the right and diffuse pallor in the left optic disk. Visual acuity at that time was 20/60 in the right eye and no light perception in the left eye.

Patient 3: A 12-year-old boy with Duchennes muscular pseudohypertrophy underwent posterior fusion of the spine from T3 through the sacrum using the Luque-Galveston technique. The duration of the procedure was approximately 6 hours, and the estimated blood loss was 2,500 mL. Blood pressure was kept at 90/55 mmHg for 10 minutes and at 80/50 mmHg for 25 minutes. During operation, a Toronto frame was used. The hemato-

crit decreased to 26.0% at the end of the operation from a preoperative value of 43%.

On recovery from general anesthesia, the patient immediately complained of bilateral visual loss. Initial fundus examination was unremarkable, as were intraocular pressures, but after 12 months the right eye showed optic atrophy more severe than in the left eye. Visual acuity was hand-motion in the right eye and 16/200 in the left eye.

Patient 4: A 57-year-old obese African-American man with hypertension and diabetes underwent complete bilateral hemilaminectomy from L3 to the sacrum (L3-4, L4-5, L5-S1). He had bilateral steel plates placed pedicularly with screw implementation. During the prolonged surgical procedure (more than 8 hours) there was estimated blood loss of 16,000 mL. The depleted volume was replaced by plasma volume expanders, red cell concentrates, and fresh plasma. Blood pressure was kept at 100/60 mmHg for 2 hours and brought as low as 90/60 mmHg for 45 minutes. During the procedure, a Wilson frame was used, and periorbital facial swelling was present after the operation. The hematocrit went from 41% preoperatively to 24% postoperatively.

Immediately after the procedure, the patient complained of bilateral visual loss. Examination revealed a visual acuity of 20/300 in the right eye and hand-motion in the left eye with normal fundus appearances and intraocular pressures. When examined 3 years later, the optic disks demonstrated temporal pallor in the right eye and diffuse optic disk pallor in the left eye.

Patient 5: A 46-year-old hypertensive man presented with a bleeding ulcer and bronchitis. He was treated with antibiotics and ulcer medications, and underwent gastroscopy. Hemoglobin was 7.3 g/dL and hematocrit was 32.7%. One day later, the hemoglobin and hematocrit were 7.3 g/dL and 22.6%, respectively, and the blood pressure was 100/50 mmHg. The anemia was not treated with blood or fluids.

Two days after initial presentation, the patient complained of complete bilateral visual loss. Ocular examination showed bare light perception bilaterally, normal intraocular pressures and retinas, and bilateral optic nerve swelling. Eight months later, he had complete bilateral optic nerve atrophy.

Patient 6: A 65-year-old woman with hypertension and diabetes underwent right knee surgery that lasted 3.5 hours. During the operation the patient lost three units of blood. Two units were replaced with her own

**Table 1.** Clinical Characteristics of the Doheny Series of Posterior Ischemic Optic Neuropathy

Patient No.	Age (y)	Gender	Systemic conditions	Procedure	Intraoperative hypotension time (h)/diastolic BP (mmHg)	Change in hematocrit (%)	Blood loss (mL)	Final visual acuity	
								Right eye	Left eye
1	66	M	Hypertension, diabetes	Spine surgery	5.0/50–70	9.5	2,400	20/50	NLP
2	43	M	Obese	Spine surgery	6.5/40–60	11.8	2,000	20/60	NLP
3	12	M	None	Spine surgery	0.5/50–55	17	2,500	HM	16/200
4	57	M	Hypertension, diabetes, obese	Spine surgery	2.75/60	17	16,000	20/300	HM
5	46	M	Obese	Gastrointestinal bleed	24.0/50	9.9	N/A	LP	LP
6	65	F	Hypertension, diabetes	Knee surgery	4.0/40	19	3,000	20/40	LP
7	44	M	Hypertension, diabetes	Spine surgery	8.5/40–50	11	1,200	20/20	NLP

HM, hand motions; LP, light perception; NLP, no light perception.

blood through a cell-saver. During the operation, her blood pressure was never lower than 110/75 mmHg. But her average preoperative hematocrit was 44% and her average postoperative hematocrit was 23%.

On regaining consciousness, the patient had no complaints, but overnight, while monitored in an intermediate-care ward, her blood pressure fell to 81/41 mmHg for a 4-hour period. She was found to have altered mental status while complaining of visual disturbances. Initial CT and MRI examinations showed no abnormality.

When examined 2 years later, visual acuity was 20/40 in the right eye and the optic disk showed mild to moderate atrophy. There was extensive visual-field loss, leaving only a central island. Visual acuity in the left eye was light perception only from the superior field and fundus examination revealed severe optic atrophy with an unremarkable retina.

Patient 7: A 44-year-old man with spondylolisthesis and foraminal stenosis underwent complete L5 laminectomy, partial facetectomy and foraminotomy, and L5-S1 fusion with autograft and screw placement. The operation lasted 8.5 hours. Diastolic blood pressure was 40 to 50 mmHg throughout the procedure, with systolic blood pressure dropping to 80 mmHg during the last 2 hours of operation. Estimated blood loss was 1200 mL without replacement. There was a drop in hematocrit from 37% to 26% postoperatively.

Two hours after the completion of the operation the patient complained of blindness. Evaluation revealed visual acuity of no light perception in each eye. Intraocular pressures were normal; fundus examination also revealed a normal optic disk, macula, retinal vasculature, and periphery in each eye.

One year later the right eye recovered to a visual acuity

of 20/20 but there was still no light perception in the left eye.

### Summary of Doheny case reports

There was a predominance of men in this series (6 of 7; 85.7%), with the majority experiencing PION after spine surgery (5 of 7; 71.4%). A history of systemic hypertension was present in 4 of 7 patients (57.1 %) and 4 of 7 patients (57.1 %) had diabetes. Although there was bilateral involvement in all cases, the left eye was more severely affected than the right eye in 5 of 6 patients (83.3%). Intraocular pressures were normal, and there were no retinal or choroidal abnormalities, including diabetic retinopathy. As demonstrated in Table 1 hypotension with anemia was the most common predisposing factor for PION in these patients, because all patients experienced intraoperative hypotension for considerable periods of time and were found to have considerable reductions in hematocrit postoperatively.

### Literature review

A review of the literature published after 1950 was performed. Patients with vasculitis, tumors, head surgery, anterior ischemic optic neuropathy, and head rest syndrome<sup>2</sup> were excluded because these patients involved a variety of factors other than intraoperative hypotension and low postoperative hematocrit. By these criteria, there were 46 case reports of patients who experienced visual loss after surgery that was from PION.<sup>3–12</sup>

Table 2 presents the clinical characteristics of these patients. Thirty-four men and 12 women between 13 and 81 years of age (mean, 50 years) had blood loss associated with gastrointestinal bleeding (7 of 46, 15.2%), spine surgery (30 of 46, 65.2%), hip surgery, coronary artery bypass surgery, cardiothoracic surgery,

**Table 2.** Literature Review (Since 1950) of Reported Posterior Ischemic Optic Neuropathy Cases

First author	n	Ages (y)	Inciting event(s)
Chisholm <sup>5</sup>	3	31, 52, 57	S/P cholecystectomy (1), S/P CT surgery (2)
Lazaro <sup>6</sup>	1	64	GI bleed
Presencia <sup>7</sup>	1	18	GI bleed
Johnson <sup>8</sup>	1	59	S/P gastroduodenotomy
Hayreh <sup>9</sup>	4	56, 57, 59, 61	GI bleed
Rizzo <sup>10</sup>	2	46, 62	S/P hip surgery (1), S/P CABG (1)
Brown <sup>11</sup>	5	13, 51, 62, 68, 81	S/P spine surgery (1), GI bleed (1), S/P vascular surgery (1), S/P CABG (2)
Katzmann <sup>12</sup>	1	57	S/P spine surgery
Katz <sup>13</sup>	4	41, 49, 60, 65	S/P spine surgery
Cheng <sup>14</sup>	24	47 ± 15	S/P spine surgery

CABG, coronary artery bypass grafting; CT, cardiothoracic; GI, gastrointestinal; S/P, status post.

and abdominal surgery. Both eyes were affected in 22 of 46 patients (47.8%) with only one eye affected in the remaining 24 of 46 patients (52.2%).

## DISCUSSION

Although infarction of the intraorbital portion of the optic nerve from systemic blood loss has been known since Hippocrates, it is often difficult to distinguish between infarction of the intraocular portion of the optic nerve (anterior ischemic optic neuropathy, AION) and PION. This is especially true in reports published before 1950.<sup>13</sup> In our review of the literature since 1950, we found reports of AION to be more frequent than reports of PION. Of note is that the reports of AION are not associated with prolonged episodes of blood loss and systemic hypotension. Both AION and PION can be triggered by hypotension; but in AION, age, long-standing diabetes, and hypertension with an anatomic predisposition (“disk at risk”) are more important factors. In contradistinction, in an animal model of PION, hypotension was found to be the most important factor.<sup>14</sup> To further differentiate the two entities, AION is almost always unilateral, generally demonstrates sectoral optic nerve edema, and manifests peripapillary hemorrhages with the edema.<sup>7,15</sup> This was not seen in the present study.

The anemia associated with PION has also been linked to anemia-related retinopathies, the chief of which is leukemic retinopathy, a retinal manifestation of anemia and thrombocytopenia typically in a leukemic patient with concomitant hyperviscosity. The findings in these entities include a normal-appearing optic nerve and retinal changes consisting of cotton-wool spots and diffuse retinal hemorrhages stemming from a proposed

mechanism of relatively acute, ischemia-induced, microvasculature breakdown.<sup>16</sup> Additionally, another condition, termed hypotensive retinopathy, conceptually needs to be differentiated from PION. Hypotensive retinopathy is also known as venous stasis retinopathy or ocular ischemic syndrome, and the pathophysiology behind this condition involves poor arterial perfusion pressure with chronic tissue ischemia, often from carotid occlusive disease.<sup>17</sup> The clinical fundus findings, limited to the retina, including venous tortuosity, microaneurysms, and retinal hemorrhages were not found in our patients. In contrast to these two diseases, PION does not involve the retina.

Table 3 compares the findings in the patients presented above with the findings in patients with PION described in the modern literature. There was a predominance of men (85.7% in this series compared with 74% in the literature), with bilateral involvement in 85.7% in this series (compared with 47.8% in the literature). In the present series of patients, PION was commonly associated with blood loss and hypotension. Five of seven patients underwent spine surgery, an area with an abundant vasculature that is not easily accessible during surgery. In these cases there was extensive blood loss, as evidenced by intraoperative hypotension and low postoperative hematocrit levels. The use of a Wilson frame was another common feature. Because three of the five spinal surgery patients were obese, they might have exceeded the Wilson frame’s design to reduce abdominal venous pressure. Although none of these cases were thought to be a “head rest syndrome,” defined as direct ocular compression by malpositioning of the head rest leading to ischemia, the dependent positioning of the head appears to have led to generalized cephalic edema

**Table 3.** Comparison of Doheny Series with Cases in the Literature

Characteristic	Doheny series (n)	Literature review (n)
Gender		
Men	6/7 (85.7%)	34/46 (74%)
Women	1/7 (14.3%)	12/46 (26%)
Age (y)	12–66 (mean, 47)	13–81 (mean, 50)
Prevalence of precipitating event		
Spine surgery	5/7 (71.4%)	30/46 (65.2%)
Gastrointestinal bleed	1/7 (14.3%)	7/46 (15.2%)
Knee surgery	1/7 (14.3%)	0
Abdominal surgery	0	2/46 (4.3%)
Hip surgery	0	1/46 (2.2%)
Coronary artery bypass grafting	0	3/46 (6.5%)
Cardiothoracic surgery	0	2/46 (4.3%)
Vascular surgery	0	1/46 (2.2%)

in four of five patients (80%) in this series. This specific circumstance probably further compromised the already low perfusion pressure of circulation to the optic nerve, because of the aforementioned systemic hypovolemia. The reason(s) that in unilateral cases the left eye was more severely affected than the right eye is open to speculation.

The profile of patients with PION is middle-aged men with sudden, bilateral vision loss, most often and more profoundly in the left eye, after gastrointestinal bleeding or spine surgery, complicated by major intraoperative hypovolemic hypotension, postoperative facial edema, and considerable reduction in hematocrit, as compared with preoperative values. The presence of hypertension and diabetes in more than half of the patients in the Doheny series suggests that these systemic disorders can play a role in the pathogenesis of PION in these patients. It has been demonstrated that these systemic disorders result in the loss of normal vasculature autoregulation from vascular endothelial dysfunction.<sup>18</sup> For example, in the setting of fluctuating blood pressures, diabetic patients fail to demonstrate normal compensatory vasoconstriction and vasodilation.<sup>19</sup> The same probably precludes autoregulatory compensation in hypertensive and diabetic patients in whom hypotension occurs during surgery.

Like any retrospective investigation, the present study is limited in not being able to consider all patients who did not present with findings of PION. So, no determination of incidence is possible until a large-scale, prospective study of anesthesia- and surgery-related complications is performed.

The results of this study suggest that the incidence of

PION could be reduced by altering perioperative management of middle-aged men undergoing spine surgery, such as minimizing hypovolemic hypotension intraoperatively, or immediately replacing blood loss, or both. In conjunction with postoperatively avoiding and aggressively treating facial edema, this could maintain sufficient optic nerve perfusion to prevent PION.

## REFERENCES

1. Sadun AA. Optic atrophy and papilledema. In: Jakobiec F, Albert D, eds, Principles and practice of ophthalmology. Philadelphia: WB Saunders; 1993;2529–2538.
2. Vandam LD. Positioning of patients for operations. In: Rogers MC, Tinker JH, Covino BG, Longnecker D, eds, Principles and practice of anesthesiology. St Louis: Mosby Year Book; 1993;703–718.
3. Chisholm LA. Optic neuropathy of recurrent blood loss. *Br J Ophthalmol* 1969;53:289–295.
4. Lazaro EJ, Cinotti AA, Eichler PN, Khawaja AA. Amaurosis due to massive gastrointestinal hemorrhage. *Am J Gastroenterol* 1971;55:50–53.
5. Presencia AC, Hernandez AM, Guia ED. Amaurosis following blood loss. *Ophthalmologica (Basel)* 1985;191:119–121.
6. Johnson MW, Kincaid MC, Trobe JD. Bilateral retrobulbar optic nerve infarction after blood loss and hypotension. *Ophthalmology* 1987;94:1577–1584.
7. Hayreh SS. Anterior ischemic optic neuropathy. VIII. Clinical features and pathogenesis of post-hemorrhagic amaurosis. *Ophthalmology* 1987;94:1488–1502.
8. Rizzo JF, Lessell S: Posterior ischemic optic neuropathy during general surgery. *Am J Ophthalmol* 1987;103:808–811.
9. Brown RH, Schauble JF, Miller NR. Anemia and hypotension as contributors to perioperative loss of vision. *Anesthesiology* 1994;80:222–226.
10. Katzmann SS, Moschonas CG, Dzieba RB. Amaurosis secondary to massive blood loss after lumbar spine surgery. *Spine* 1994;19:468–469.

11. Katz DM, Trobe JD, Cornblath WT, Kline LB. Ischemic optic neuropathy after lumbar spine surgery. *Arch Ophthalmol* 1994;112:925–931.
12. Cheng MA, Sigurdson W, Tempelhoff R, Laurysen C. Visual loss after spine surgery—a survey. *Neurosurgery* 2000;46:625–631.
13. Hollenhorst RW, Wagner HP. Loss of vision after distant hemorrhage. *Am J Med Sci* 1950;219:209–218.
14. Hacıyakupoglu G, Isiguzel I, Zorludemir S, et al. Early ultrastructural findings and superoxide dismutase levels in experimental ischemic optic neuropathy—effect of hypertension and hypotension. *Ophthalmologica* 2001;215:55–60.
15. Arnold AC. Ischemic optic neuropathy, diabetic papillopathy, and papillophlebitis. In: Yanoff M, Duker JS, eds. *Ophthalmology* London: Mosby International Ltd; 1999;Ch 11.7.
16. Schachat AP, Markowitz JA, Guyer DR, et al. Ophthalmic manifestations of leukemia. *Arch Ophthalmol* 1989;107:697–700.
17. Dugan JD, Gree WR. Ophthalmologic manifestations of carotid occlusive disease. *Eye* 1991;5:226–238.
18. Dzau VJ. Tissue angiotensin and pathobiology of vascular disease: a unifying hypothesis. *Hypertension* 2001;37:1047–1052.
19. McAuley DF, McGurk C, Nugent AG, et al. Vasoconstriction to endothelin-1 is blunted in non-insulin-dependent diabetes: a dose-response study. *Clin Sci (Lond)* 2000;99:175–179.

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