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Review Article

Postoperative Vision Loss Following Prone Position Spinal Surgery: A Review of Published Cases

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Abstract

Background: Postoperative visual loss (POVL) is a rare but devastating complication of prone-position spinal surgery, most often caused by ischemic optic neuropathy (ION).

Objective: To synthesize published epidemiology, pathophysiology, and case-level evidence on POVL after prone spinal surgery.

Methods: Narrative review of case reports, case series, registries, administrative-database studies, case-control analyses, and practice advisories.

Findings: Incidence ranges roughly from 1 to 6 per 10,000 spinal fusions, declining over 1998-2012 (1.63 to 0.60 per 10,000). Independent risk factors include male sex, obesity, Wilson frame use, longer anesthetic duration, greater blood loss, and lower colloid proportion. Published cases document posterior ION, central retinal artery occlusion, and cortical blindness, frequently bilateral and often permanent.

Conclusions: No treatment reliably reverses established POVL; management centers on risk stratification, staged surgery, hemodynamic and hematocrit optimization, and positioning vigilance.

Keywords: Postoperative vision loss; Ischemic optic neuropathy; Prone position; Spinal surgery; Central retinal artery occlusion

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Introduction

Postoperative visual loss (POVL) after spinal surgery is a rare but catastrophic complication first drawing wide clinical attention through case series in the 1990s [1,2] and subsequent registry and administrative-database studies. Most cases occur after prolonged procedures performed in the prone position for complex spinal fusion, and the dominant mechanism is ischemic optic neuropathy (ION) rather than direct ocular injury [3,4]. Because POVL is unpredictable, frequently bilateral, and has no established effective treatment once established, the literature has focused heavily on identifying risk factors and prevention strategies rather than rescue therapy [5,6]. This review synthesizes the epidemiologic, mechanistic, and

case-level literature on POVL following prone spinal surgery, drawing on registry data, large administrative cohorts, case-control studies, and individual published cases of ION, central retinal artery occlusion (CRAO), and cortical blindness. The topic is clinically important beyond its rarity: complex, multilevel instrumented fusions for deformity correction and degenerative disease have become more common, cases are frequently performed on older and heavier patients with more cardiovascular comorbidity, and any resulting blindness is typically permanent, generating substantial morbidity, litigation risk, and patient distress disproportionate to the small number of affected individuals each year [7,8]. Consequently, professional societies have issued and periodically updated formal practice advisories addressing preoperative counseling, intraoperative positioning, and hemodynamic management specifically for this complication [9,5], while parallel efforts have used large administrative datasets to track incidence trends over time and identify modifiable risk factors [10,6].

Epidemiology

Estimates of POVL incidence after spinal fusion vary by an order of magnitude depending on ascertainment method. Using the US Nationwide Inpatient Sample (NIS), Rubin et al. found an ION incidence of 1.02 per 10,000 posterior thoracic, lumbar, and sacral fusions (257 cases among 2,511,073 discharges, 1998-2012), with a significant decline from 1.63 per 10,000 in 1998-2000 to 0.60 per 10,000 in 2010-2012 (incidence rate ratio 0.72 per three-year interval, 95% CI 0.58-0.88) [10]. A separate NIS-based risk-prediction analysis reported a comparable overall rate with sex- and comorbidity-stratified risk as high as 844.5 per million procedures in men aged 40-64 with obstructive sleep apnea [7]. A longitudinal medical-claims database instead found a substantially higher rate of 6 per 10,000 fusions (65 of 106,936 procedures), illustrating the sensitivity of estimates to data source and coding practice [11]. Earlier

single-institution and multicenter series reported visual complication rates from roughly 0.09% to 0.2%, including a case-control analysis drawn from 126,666 consecutive procedures at one institution [12,1,13]. For contrast, ION incidence after cardiac surgery (1.43 per 10,000) showed no significant change over the same period, arguing against a pure coding artifact explaining the spine-fusion decline [14]. The ASA Postoperative Visual Loss Registry, a voluntary case series rather than a denominator-based study, recorded 93 spine surgery cases, of which 83 (89%) were ION and 10 (11%) were CRAO [3]. Scoping and narrative reviews collating this heterogeneous literature echo the same wide range, roughly 0.013% to 1% [15,16,17,8].

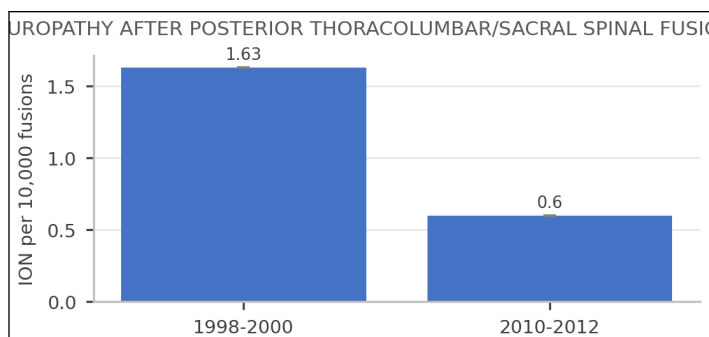


Figure 1: Incidence of ischemic optic neuropathy after posterior thoracolumbar/sacral spinal fusion declined from 1.63 to 0.60 per 10,000 fusion discharges between 1998-2000 and 2010-2012 (incidence rate ratio 0.72 per 3-year interval, 95% CI 0.58-0.88, P=0.002) [10].

Pathophysiology

ION is thought to result from a combination of reduced optic nerve perfusion and elevated venous/orbital pressure during prolonged prone positioning, most often affecting the retrobulbar (posterior) portion of the nerve where watershed vascular supply is most vulnerable [18,4]. Prone positioning itself measurably raises intraocular pressure over the duration of surgery, providing a plausible link between prolonged prone time and impaired optic nerve head perfusion [19]. The largest case-control analysis, spanning 80 ION cases and 315 matched controls across 17 institutions, identified independent predictors: Wilson frame use (adjusted OR

4.30, 95% CI 2.13-8.75), obesity (OR 2.83), male sex (OR 2.53), anesthetic duration per hour (OR 1.39), estimated blood loss per liter (OR 1.34), and a protective effect of higher colloid proportion in non-blood fluid replacement (OR 0.67 per 5% increment) [6]. Administrative-database analyses corroborate several of these associations, adding blood transfusion (IRR 2.72) and age per decade (IRR 1.24) as independent risk factors, while finding female sex protective [10]. CRAO, by contrast, is generally attributed to direct external compression of the globe from malpositioned headrests, horseshoe supports, or periocular pressure, producing a distinct unilateral compressive mechanism rather than a perfusion-deficit one [20,21,22]. Cortical blindness, the rarest mechanism, arises from bilateral occipital lobe ischemia and can occur even with short operative time and minimal blood loss, implicating positional venous congestion or embolic phenomena rather than the hypotension/blood-loss axis dominant in ION [23,24].

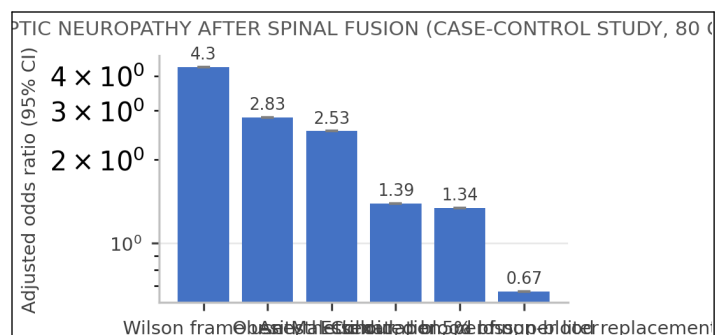


Figure 2: Independent predictors of ischemic optic neuropathy after spinal fusion: Wilson frame use (OR 4.30), obesity (OR 2.83), male sex (OR 2.53), anesthetic duration per hour (OR 1.39), estimated blood loss per liter (OR 1.34), and colloid proportion (protective, OR 0.67 per 5% increment) [6].

Clinical Presentation

POVL typically becomes apparent in the immediate postoperative period, often noted on emergence or within the first 24-48 hours, and is characteristically painless [3,25]. Posterior ION frequently presents with bilateral, severe loss ranging from counting fingers to no light perception, without accompanying pupillary or

fundoscopic abnormality early in the course, since the retrobulbar segment of the nerve is affected [18,26]. CRAO instead presents with acute unilateral loss and, when examined promptly, a pale retina with a cherry-red macular spot [22]. Cortical blindness presents with bilateral loss but preserved pupillary light reflexes, reflecting an intact anterior visual pathway with cortical rather than ocular pathology [23,24]. Associated periorbital findings such as proptosis, chemosis, or supraorbital edema, when present, suggest an orbital compressive component rather than pure ischemic mechanism [27]. Unilateral, rather than bilateral, loss can also occur with posterior ION, as illustrated by an early published case of unilateral blindness after prone lumbar spine surgery [28].

Table 1: Published cases of postoperative vision loss following prone position spinal surgery reviewed here [25,26,23,22,20,27,21,24]; N = 8 reports.						
Report	Age/s ex	Surgery / duration	EBL / hy potensio n	Vision loss type	Later ality	Outcom e
Nguyen 2026	80/M	Posterior lumbar fusion L2-L5, prone, Jackson table / 9.5 h	EBL 1.8 L; MAP 55-65 mmHg	Posterior ION	Bilat eral	Light per ception only, POD1
Bhandar i 2019	33/F	Pedicle screw fixation/plating, prone / prolonged, hours not stated	Mild hyp otension ; anemia	Non-arte ritic posterior ION	Right eye	Countin g fingers; no impr ovement at 1 yr
Goni 2012	38/M	Posterior de compression/instrumen tation L2 fracture, prone / 105 min	EBL 420 mL; BP 90-110 mmHg	Cortical blindnes s	Bilat eral	Permane nt blind ness at 3-yr follo w-up
Tang 2026	53/M	Posterior atlantoaxial reduction/fi xation, prone / not stated	Not stated; h emodyn amically stable	CRAO	Right eye	Improve d to 20/25; severe field loss persisted

Report	Age/s ex	Surgery / duration	EBL / hy potensio n	Vision loss type	Later ality	Outcom e
Bae 1996	53/M	Cervical spine surgery, prone, horseshoe headrest / not stated	Hypoten sion noted; EBL not stated	CRAO	Left eye	Field dist urbance on emer gence; final acuity not stated
Ramakri shnan (Puri) 2024	28/M	Cervicodors al spine injury surgery; position not stated	Not stated	Ophthal mic artery oc clusion	Left eye	Light per ception only
Sys 1996	Not s tated	Posterior lumbar fusion, rectangular headrest / not stated	Not stated	CRAO	Not s tated	Not stated
Huber 1998	Not s tated	Lumbar spine surgery, prone / not stated	Not stated	Bilateral cortical blindnes s	Bilat eral	Not stated

Diagnostic Approach

Evaluation begins with a focused examination confirming visual acuity, pupillary responses (including relative afferent pupillary defect), extraocular movements, and dilated funduscopy to distinguish ION (typically a normal-appearing disc initially in posterior ION) from CRAO (retinal whitening, cherry-red macular spot) [22,17]. Neuroimaging (MRI/MRA) is indicated when cortical blindness is suspected, to identify occipital infarction, and can also help exclude an intracranial or orbital compressive lesion in atypical presentations [23]. Ophthalmology and neurology consultation is recommended promptly given the overlapping differential diagnosis and the narrow window for any intervention in CRAO [5]. Because ION is a clinical diagnosis of exclusion without a definitive confirmatory imaging test, careful correlation with intraoperative course (duration, blood loss, hemodynamics, positioning device, lowest recorded blood pressure and hematocrit) is essential to support the

diagnosis and to exclude alternative causes such as embolic stroke, direct globe trauma, or a compressive orbital process [6,3]. A high index of suspicion is warranted whenever new visual complaints, however vague, follow a lengthy prone procedure, since early recognition — although it has not been shown to alter the ultimate visual outcome — allows prompt exclusion of reversible mimics and appropriate counseling of the patient and family [5,8].

Table 2: Mechanism, laterality, and risk profile of postoperative vision loss after prone spinal surgery [3,6,10,18,4].				
Mechanism	Share of registry POVL cases	Typical laterality	Key risk factors	Prognosis
Ischemic optic neuropathy (posterior)	83/93 (89%)	Frequently bilateral	Wilson frame, male sex, obesity, longer anesthesia, greater blood loss, lower colloid proportion	Poor; 38% any improvement, 85% no improvement if no light perception at onset
Central retinal artery occlusion	10/93 (11%)	Usually unilateral	Direct globe/orbital compression from headrest or periocular pressure	Generally poor; occasional partial recovery reported with early treatment
Cortical blindness	Not separately quantified in registry	Bilateral	Can occur with short surgery and minimal blood loss; mechanism uncertain	Often permanent

Management

Because no proven treatment reliably reverses established POVL, the literature emphasizes prevention. The ASA practice advisories recommend informing high-risk patients (those undergoing lengthy prone spinal fusion) of the risk, considering staged procedures to limit anesthetic duration and blood loss, maintaining blood pressure near baseline rather than inducing deliberate hypotension,

monitoring blood loss and hematocrit, using colloids together with crystalloids for major blood loss, positioning the head level with or higher than the heart when possible, avoiding direct globe pressure, and periodic globe checks during prone positioning [5,9]. Avoidance of the Wilson frame, given its strong association with ION, is a specific positioning recommendation supported by case-control data [6]. For CRAO from external compression, meticulous attention to headrest fit and periodic repositioning checks is the primary preventive measure [21,20]. Once vision loss occurs, management is largely supportive: some reports describe empiric measures such as blood pressure optimization, correction of anemia, and, for CRAO, hyperbaric oxygen therapy, which was associated with partial visual recovery in one recently published case [22]. No pharmacologic or surgical intervention has demonstrated reliable efficacy for established ION [8,4]. Surgical planning strategies proposed to reduce risk in anticipated long, high-blood-loss cases include staging multilevel fusions into two or more shorter sessions, minimizing time in extreme head-down or dependent positioning, using a padded, well-fitted headrest that avoids direct globe contact, and intraoperative confirmation of head and neck position after any repositioning of the operating table [5,17]. Anesthetic management further emphasizes avoiding deliberate hypotensive technique in patients undergoing prolonged prone spinal fusion, replacing blood loss promptly with a combination of crystalloid and colloid, and considering earlier transfusion thresholds in patients with substantial blood loss, consistent with the protective association of colloid administration and the harmful association of transfusion volume identified in the case-control literature [6,10]. Because published evidence for these measures derives largely from observational and case-control data rather than randomized trials, current guidance is presented as a practice advisory rather than a mandate, and continues to be updated as new registry data accrue [9,5].

Outcomes/Prognosis

Visual outcomes after POVl are generally poor. In a mixed-surgery posterior ION series, only 38% of patients showed any improvement, and among those presenting with no light perception, 85% had no recovery [18]. In an early spine-specific series of 37 POVl cases, 68% had no change in vision and 3% worsened [2]. Individual published cases mirror this pattern: permanent bilateral blindness at three-year follow-up after cortical blindness [23], no improvement in posterior ION at one year [26], and only partial recovery of acuity with persistent severe field loss after treated CRAO [22]. CRAO appears to carry somewhat more potential for partial recovery than ION, particularly with early intervention such as hyperbaric oxygen, though evidence is limited to isolated case reports [22,27]. Cortical blindness, though rare, can also be permanent even after apparently uncomplicated surgery with minimal blood loss and stable hemodynamics, indicating that a benign intraoperative course does not exclude a poor visual outcome [23]. Bilaterality itself appears to portend a worse prognosis; registry-derived estimates place bilateral involvement in more than half of ION cases, though the exact proportion is reported inconsistently across secondary sources [3,4]. Overall, most published cases describe irreversible or only marginally improved vision, underscoring that prevention remains the primary clinically actionable strategy and that patient counseling before high-risk surgery should set realistic expectations about the limits of rescue therapy [17,8].

Conclusion

POVl after prone spinal surgery, while rare, is a serious complication with a large body of registry, administrative, and case-level literature converging on a consistent risk profile: prolonged prone positioning, substantial blood loss, hypotension, obesity, male sex, and use of a Wilson frame. ION accounts for the majority of cases and carries the poorest prognosis, while CRAO and cortical blindness

are less common but mechanistically distinct. Because no reliable treatment exists once vision loss occurs, anesthetic and surgical teams should focus on preoperative risk discussion, intraoperative hemodynamic and positioning vigilance, and staged surgery for anticipated high-risk cases.

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