Brian Gill James E. Heavner

Postoperative visual loss associated with spine surgery

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B. Gill (⊠) Orthopaedic Surgery, Texas Tech University Health Sciences Center, Lubbock, TX, USA E-mail: brian.gill@ttuhsc.edu Tel.: +1-806-7432465 Fax: +1-806-7431305

J. E. Heavner Anesthesiology, Texas Tech University Health Sciences Center, Lubbock, TX, USA

Abstract Postoperative visual loss associated with spine surgery is a rare complication with no established definitive etiology. Multiple case reports have been published in the literature, and an overview of the case reports of the various visual disturbances following spine surgery is presented. Our objective was to review the current literature and determine if there were any risk factors that suggest what kind of patients have a higher likelihood of developing postoperative visual loss. Furthermore, analysis of factors common to the cases may offer a better understanding of possible etiologies leading to prevention strategies of postoperative visual loss. We used PubMed to perform a search of literature with spine surgery cases that are associated with visual disturbances. A total of 7 studies representing 102 cases were reviewed and evaluated in regard to age, sex, comorbidities, diagnosis, operative time, blood loss, systolic blood pressure, lowest hematocrit, and visual deficits and improvement. Ischemic optic neuropathy, especially posterior ischemic optic neu-

ropathy, was the most common diagnosis found in the studies. The average age of the patients ranged from 46.5 years to 53.3 years with the majority having at least one comorbidity. Operative time ranged on average from 385 min to 410 min with a median in one case series of 480 min, average blood loss ranged from 3.5 l to 4.3 l and no visual improvement was seen in the majority of the cases. The etiology of postoperative visual loss is probably multifactorial, however, patients with a large amount of blood loss producing hypotension and anemia along with prolonged operative times may be causing a greater risk in developing visual disturbances. An acute anemic state may have an additive or synergistic effect with other factors (medical comorbidities) leading to visual disturbances. Although our study failed to provide definitive causative factors of postoperative visual loss, suggestions are made that warrant further studies.

Keywords Spine surgery · Blindness · Visual loss/disturbance · Ischemic optic neuropathy

Introduction

Hollenhorst first described visual loss following spine surgery in 1954 [22] and more reports of visual disturbances following spine surgery have been published since then. Postoperative visual disturbances are morbid events that occur infrequently, but are debilitating to the patient when they present. The incidence is unknown due to the rareness of the events, as well as, the reported events being only case reports. Roth et al. [41] estimated perioperative visual loss to be 1 per 61,000 in their study of nonopthalmological surgeries. A more recent and defined article in terms of spine surgery by Warner et al. [51] reported that they had no incidences of perioperative visual loss in approximately 12,000 spinal procedures. Reports of visual disturbances in the literature, especially following spine surgery, is of growing concern to the surgeon as well as to the anesthesiologist. In 1999, the American Society of Anesthesiologists created the Postoperative Visual Loss Registry (ASA POVL) to determine if there is a common denominator or risk factors that make an individual more susceptible to postoperative visual loss. This registry excludes ophthalmologic surgeries.

In the most recent report released by the ASA POVL [33], 79 cases have been submitted with 67% of cases being associated with spine surgery. Ten percent of the cases occurred with cardiac bypass procedures and 25% with miscellaneous procedures (liver transplant, tho-racoabdominal aneurysm resections, peripheral vascular procedures, head and neck operations, prostectomies and other cases). The ophthalmologic diagnosis associated with the spine surgery cases (N=53) included ischemic optic neuropathy (ION) (n=43), central retinal artery occlusion (CRAO) (n=7) and unknown diagnosis (n=3). This report was abbreviated; therefore, an

extensive review is unavailable. Their results are presented in Table 1 as a comparison with the other published reports found in the literature.

The purpose of this article is to (1) review the cases in the literature regarding visual disturbances in association with spine surgery, (2) describe the visual disturbances associated with spine surgery, and (3) discuss the various pathophysiological mechanisms thought to produce the disturbances.

Materials and methods

A literature search was performed using PubMed reviewing spine surgery cases associated with visual disturbances. The search included all peer reviewed English journals without any other limitations. Key words used in the search included "spine surgery," "blindness," "visual loss/disturbance," and "ischemic optic neuropathy." All forms of spine surgery were included, although the majority of cases were lumbar decompressions and fusions. All identified literature-reported cases in English have been included in this review. A total of 7 studies representing 102 cases were reviewed and evaluated in regard to age, sex, comorbidities, diagnosis, operative time, blood loss, systolic

 Table 1
 Visual Disturbances following Spine Surgery: Comparison of Cases

| Number of cases | 15 cases | 37 cases | 43 cases | 7 cases |
|---|---------------------|----------------|------------------------|---|
| References | [1, 12, 24, 34, 46] | [36] | [33] | [33] |
| Age (year) avg; range | 53.3; (12–79) | 46.5; (12–68) | 49^a ;(19–73) | 49^a ;(35-71) |
| Sex (%) | | | | |
| Male | 60 | NA^b | NA | NA |
| Female | 40 | NA | NA | NA |
| Diagnosis (#) | | | | |
| PION ^c | 10 | 14 | 43^{d} | NA |
| AION ^e | 1 | 8 | 0 | 0 |
| CRAO/CRVO ^f | 1 | 9 | 0 | 7 |
| Cortical infarct | 3 | 3 | 0 | 0 |
| Other/unspecified | 0 | 3 | 0 | 0 |
| Comorbidities (%) | 80 | 58 | NA | NA |
| Operative time avg; range (min) | 385; (180-510) | 410; (120-750) | 480^a ; (180–1440) | 330^a ; (204–540) |
| Systolic Blood Pressure avg; range (mmHg) | NA | 77; (0–98) | NA | NA |
| Blood loss avg; range (1) | 4.3; (0.8–16) | 3.5; (0.4–18) | 2.3^{a} ; (0.2–20.0) | 0.7^a ; (0.5–1.3) |
| Lowest recorded Hct avg: range (%) | (18.5–39) | 28: (18–36) | 25.5^{a} : (19–40) | 33^a : (29–38) |
| Visual Deficits (%) | (, | -, () | | , |
| Unilateral | 53 | 70 | 42 | 100 |
| Bilateral | 47 | 30 | 58 | 0 |
| Follow up (%) | | | | |
| Visual Improvement | 40 | 36^h | 0 | 0 |
| No change | 60 | 68^h | 56^g | 100^{g} |
| Not reported | 0 | 0 | 44 | 0 |
| | | | | |

Median

^bNot available

^cPosterior ischemic optic neuropathy

^dIschemic optic neuropathy (unspecified PION or AION)

^eAnterior ischemic optic neuropathy

^fCentral retinal artery occlusion/central retinal venous occlusion

^gNo vision recovery

^h As reported in article

blood pressure, lowest hematocrit, and visual deficits and improvement. Statistical analysis was performed by one of the authors (B.G.) to obtain the data reported in Results section and in Table 1. We lacked a control group, which precluded us from doing a more detailed analysis.

Results

In the largest review of visual loss following spine surgery to date, Myers et al. [36] presented 37 cases. The average age of the patients was 46.5 years with a range of 12-68 years. Risk factors noted in the patients included hypertension, smoking, diabetes, and vascular disease. Almost all of the patients were placed in the prone position throughout the operations, which averaged 410 min. Systolic blood pressure and hematocrit dropped from an average of 130 mmHg and 40% preoperatively to 77 mmHg and 28% intraoperatively, respectively. In a matched group comparison of patients with no visual disturbances outlined in their study, only operative time and intraoperative blood loss were found to be significantly greater in patients with visual impairment. The most common symptoms that the patients experienced were visual field/acuity loss or complete blindness. Symptoms were noted in 81% of the cases by the second postoperative day. ION accounted for the majority of the diagnosis with central retinal artery occlusion and cortical ischemia making up the remainder of the diagnoses. Only 36% reported improvement and 68% reported no improvement [36].

The cases reported in Myers et al. [36] included ten cases that were previously reported in the literature as stated in their article and by cross-referencing their bibliography. These ten cases were excluded from our literature review. Therefore, our search yielded 15 cases found in the literature. The age of the 15 patients who were found by our search ranged from 12 to 79 years with the average being at 53.3 years. Comorbidities included diabetes, hypertension, smoking, heart disease, vascular disease, and obesity. The patients were almost exclusively placed in a prone position with an average operation time of 385 min, average blood loss of 4.3 l, and the lowest reported hematocrit for each patient ranged from 18.5% to 39.0%. The most common diagnosis was ION with the remaining split between central retinal artery/venous occlusion and occipital infarction. Visual improvement occurred in 40% of the cases while 60% reported no change in vision upon final follow-up [1, 12, 24, 34, 46].

Reviewing the ASA POVL registry data reveals that 53 cases associated with spine surgery have been submitted. Of the 53 cases, 43 were diagnosed with ischemic optic neuropathy, 7 were diagnosed with central retinal artery occlusion, and the remaining had an unknown diagnosis. The majority of patients with ION had bilateral involvement, experienced a median blood loss of 2.3 l and had no vision improvement. The patients with CRAO had unilateral visual deficits, experienced a median blood loss of 0.7 l, and none experienced vision improvement. Due to the ASA POVL registry being a closed claims project, previous published data might be included in this registry. We contacted the directors of the ASA POVL registry, but they were unable to supply any additional information or clarification on which cases have been submitted to the closed claims project. Therefore, overlap may exist between the cases found in our literature search with the cases reported in the ASA POVL Registry.

Discussion

Lesions associated with spine surgery

A) Ischemic optic neuropathy

The presence of an afferent pupillary defect indicating a unilateral optic nerve dysfunction, visual field defect, and an absence of other causes of decreased vision is diagnostic of ION. In contrast to ION of other etiologies that usually produce unilateral lesions, over half of the cases reported in the ASA POVL registry had bilateral disease. ION is classified into two categories based upon where the insult occurs in the optic nerve, anterior or posterior. A key difference in determining between anterior and posterior ION is whether optic nerve swelling is present by direct ophthalmoscopy indicating an anterior involvement [46]. Presumably, anterior ischemic optic neuropathy (AION) and posterior ischemic optic neuropathy (PION) can be present simultaneously. This may explain why some of the patients failed to report improvement in their vision because of the possibility that both portions of the optic nerve were involved resulting in a greater degree of injury. Selective anterior or posterior optic nerve neuropathy occurs possibly due at least in part to differences in blood supply to the two different areas of the nerve. Perhaps if only one section of the nerve is involved, then the prognosis may be better. From the presented data and the case reports, it appears that PION may be associated with poorer prognosis for vision improvement as noted by Dunker et al. [12].

Anterior ischemic optic neuropathy: AION is usually painless and irreversible, and is associated with two characteristics: (a) presence of visual field deficits and/or changes in visual acuity, and (b) optic disc edema with subsequent atrophy [17, 55]. The optic disc swelling is perhaps the earliest sign of AION [19, 42]. Splinter hemorrhages may also be present around the optic disc [16]. Most commonly, the visual field defect occurs in the inferior half of the visual field [6, 37, 38]. Visual loss from AION results from infarction of the short posterior ciliary arteries in the choriocapillaris in the watershed areas [37, 52]. However, variations in the blood supply of the optic nerve determine the severity of visual loss as "differing degrees of ischemia produce differing effects" [18, 19, 54].

Other factors contributing to a decrease in perfusion pressure include blood viscosity, as AION has been reported in patients with an increased viscosity. Sickle cell disease and polycythemia have led to this above normal viscosity [42, 44]. Decreased oxygen transport associated with iron-deficiency anemia and hemorrhages are also reported in the literature as causes of AION [2, 30, 39, 47, 48]. Anatomical differences, especially in small optic disks, are associated with a higher frequency of AION [26, 35]. Multiple treatment modalities have had little success in the treatment of AION. Retrobulbar steroid injections, antiplatelet therapy, anticoagulants, phenytoin, norepinephrine and blood replacement have been reported in the literature as attempted modalities [3, 17, 21, 29, 43].

Posterior ischemic optic neuropathy: PION is like AION presenting as acute loss of vision with visual field defects. PION is not usually associated with occlusive vascular disease. Severe anemia or hypotension is the more likely cause of PION because of the more tenuous blood supply to the retrobulbar optic nerve [4, 54]. Due to this poor blood supply, a watershed area exists within the posterior portion of the optic nerve. Williams et al. [54] suggests that the etiology of PION is multifactorial when "severe hypotension and anemia are combined with at least one other factor (e.g., congenital absence of the central artery, infection or venous obstruction)". Surgery, trauma or GI bleeding in which severe anemia and hypotension occurs have also been associated with PION [20, 25, 27, 36, 46].

B) Central retinal artery occlusion

According to Lee, CRAO is thought to have three possible etiologies, direct pressure on the globe from face masks and cushions in the prone position, by emboli, or by low-perfusion pressure in the retina. The findings of low-estimated blood loss, lack of anemia, shorter duration of prone position (versus patients with ION), unilateral disease, and no vision recovery were consistent with the proposed etiologies [33]. The site and duration of the vascular insufficiency presumably determines the visual defect caused and its reversibility. For instance, most retinal artery occlusions due to emboli during open-heart surgery resolve within 30 min of discontinuation of the artery results in a limited

field defect or blurred vision. Ophthalmologic examination in patients with CRAO reveals a pale, edematous retina, cherry-red spot on the fovea, and platelet, fibrin or cholesterol emboli in the narrowed retinal arteries. Optic atrophy, the usual outcome in ION, occurs in 50% of eyes with CRAO [54].

Proposed theories of visual disturbances following spine surgery

Although a direct causal relationship has yet to be determined, there are numerous suggestions on how intraoperative events may lead to blindness following spine surgery. The above-mentioned case reports suggest several modifiable contributing factors including anemia, hypotension, and prolonged pressure on the eye as predisposing factors. Risk factors, especially atherosclerotic, have been proposed that may predispose a patient to an increased risk of visual loss, which include chronic hypertension, smoking, vascular disease, diabetes, increased blood viscosity, and anatomic anomalies [5, 6, 8, 13, 27, 54].

Anemia is a proposed contributing factor in visual loss associated with spine surgery as well as other surgeries [8, 27, 28, 32]. Myers et al. [36] found that only operative time and intraoperative blood loss were statistically significant in their patients experiencing vision loss. There was no difference between the blindness group and the control in hematocrit and systolic blood pressure, although both were considered to be below normal.

The notion that hypotension is a contributing factor that has been reported, but induced intraoperative hypotension and its relative few complications in the majority of patients suggests that hypotension in the absence of other risk factors is unlikely the direct causative agent [36]. The amount of time that the patient remains hypotensive may be more important than the degree of hypotension [18]. Infarction of the optic nerve head where the blood supply is most susceptible to compression from edema has been shown to occur in anemic patients who experience hypotension [25].

Direct pressure upon the eye, increasing intraocular pressure (IOP), has been documented as a possible factor resulting in visual loss [7, 14, 15, 22, 23, 45, 46, 53, 56]. Venous congestion and arterial occlusion are the proposed reasons why ischemia occurs in the retina due to the prone positioning [50]. When IOP exceeds intravenous pressure, venous collapse occurs resulting in decreased blood flow.

Other cases have also been reported of blindness in the prone position [9, 11, 36, 40]. Most patients who undergo spine surgery in the prone position do not experience visual disturbances [34]. There are also published reports of patients experiencing visual loss after general surgeries in the supine position where it is highly unlikely that direct pressure on the eye ever occurred [27, 54]. Therefore, other factors must contribute to these visual changes such as ocular anatomy, coexisting morbidities, and fluid replacement [34]. For example, occlusive eye protection may not allow appropriate expansion of the eyelids, thus placing pressure upon the eye and increasing IOP [55]. PION may occur when patients are placed in the prone position for a long duration, thereby increasing venous congestion especially when receiving large volume fluid replacement [11, 54]. Even though direct compression of the globe can cause perioperative blindness, more likely etiologies remain [34].

A recent article by Cheng et al. [10] examined IOP in spinal surgeries studying the effects of prone positioning under general anesthesia on IOP. They showed that a statistically significant increase in IOP occurs in the prone position when compared to the patient's preoperative baseline under general anesthesia. Lam et al. [31] showed an increase in IOP over baseline in prone awake patients after 8 min. Moreover, a direct correlation was found between the amount of time spent in the prone position and the resulting increase IOP [10]. IOP may also be influenced by prone positioning in that peritoneal pressure increases translating into higher central venous pressure and peak inspiratory pressure. When a patient is in the prone position, the optimal position is a head-neutral or head-up position due to studies showing that Trendelenburg po-Finally, a sition increases IOP [49]. positive intraoperative fluid balance has been shown to increase IOP, and perhaps may have an additive effect on increasing IOP when the patient is placed in the prone position although further studies are needed to investigate this question.

Conclusion

Postoperative visual loss remains an unfortunate and infrequent morbid event occurring in patients undergoing spine surgery. According to the cited incidence rates, postoperative visual loss is rare enough that a practicing spine surgeon would highly unlikely encounter this complication during their career, yet it still may occur at any given time. Although we believe that the etiology is multifactorial, blood loss and operative times are two intriguing characteristics we discovered in our review. All patients may be at the risk of postoperative visual disturbances following spine surgery but there seems to be a greater risk when the surgery is prolonged and the amount of blood loss as previously suggested by Myers et al. [36]. An acute anemic state may have an additive or synergistic effect with other possible causative factors leading to an ischemic event at the optic nerve. Careful attention to perioperative details may influence a patient's risk in developing visual disturbances, for example, smoking cessation before surgery, documentation of visual impairment prior to surgery, avoiding direct pressure on the eye, and judicious use of drugs and anesthetics in patients with glaucoma. Other examples include avoiding prolonged reduction of oxygen delivery to the eye as a result of hypotension or anemia and minimizing the duration a patient is in the prone position. In conclusion, we believe there is ample evidence presented here to suggest that a search for dissimilarities in risk factors and intraoperative events associated with different lesions found in patients with postoperative visual disturbances might advance the understanding of causes and prevention of this unfortunate outcome.

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References

- Alexandrakis G, Lam BL (1999) Bilateral posterior ischemic optic neuropathy after spinal surgery. Am J Ophthamol 127:354–355
- Ballen PH, Fox MJ, Weissman GS (1985) Ischemic optic neuropathy secondary to intestinal hemorrhage. Ann Ophthalmol 17:486–488
- Bastiaensen LAK, Keunen RWM, Tijssen CC, Vandoninck JJ (1986) Anterior ischemic optic neuropathy: sense and nonsense in diagnosis and treatment. Doc Ophthalmol 61:205–210
- Beck RW, Servais GE, Hayreh SS (1987) Anterior ischemic optic neuropathy IK. Cup-to-disc ratio and its pathogenesis. Ophthalmology 94:1503– 1508
- Beri M, Klugman MR, Kohler JA, Hayreh SS (1987) Anterior ischemic optic neuropathy: VII. Incidence of bilaterality and various influencing factors. Ophthalmology 94:1020–1028
- Boghen DR, Glaser JS (1975) Ischaemic optic neuropathy: the clinical profile and history. Brain 98:689–708
- Bohlman HH (1978) Complications in orthopaedic surgery. Lippincott, Philadelphia

- Brown RH, Schauble JF, Miller NR (1994) Anemia and hypotension as contributors to perioperative vision loss. Anesthesiology 80:222-226
- Cheng MA, Sigurdson W, Tempelhoff R, Lauryssen C (2000) Visual loss after spine surgery: a survey. Neurosurgery 46:625–630
- Cheng MA, Todorov A, Tempelhoff R et al (2001) The effect of prone positioning on intraocular pressure in anesthetized patients. Anesthesiology 95:1351–1355
- Dilger JA, Tetzlaff JE, Bell GR et al (1998) Ischaemic optic neuropathy after spinal fusion. Can J Anaesth 5:63–66

- Dunker S, Hsu HY, Sebag J, Sadun AA (2002) Perioperative risk factors for posterior ischemic optic neuropathy. J Am Coll Surg 194:705–710
- Feit RH, Tomsak RL, Ellenberger C (1984) Structural factors in the pathogenesis of ischemic optic neuropathy. Am J Ophthamol 98:105–108
- Givner I, Jaffe N (1950) Occlusion of the central retinal artery following anesthesia. Arch Ophthalmol 43:197– 201
- Grossman W, Ward WT (1993) Central retinal artery occlusion after scoliosis surgery with a horseshoe headrest. Spine 18:1226–1228
- Hayreh SS (1974) Anterior ischemic optic neuropathy: II. Fundus on ophthalmoscopy and flourescein angiography. Br J Ophthalmol 58:964–980
- Hayreh SS (1974) Anterior ischaemic optic neuropathy, III: treatment, prophylaxis, and differential diagnosis. Br J Ophthalmol 58:981–989
- Hayreh SS, Kolder HE, Weingeist TA (1980) Central retinal artery occlusion and retinal tolerance time. Ophthalmology 87:75–78
- Hayreh SS (1981) Anterior ischemic optic neuropathy V. Optic disc edema an early sign. Arch Ophthalmol 99:1030–1040
- Hayreh SS (1981) Posterior ischemic optic neuropathy. Ophthamologica 182:29–41
- Hollenhorst RW, Wagener HP (1950) Loss of vision after distant hemorrhage. Am J Med Sci 219:209–218
- 22. Hollenhorst RW, Svien HL, Benoit CF (1954) Unilateral blindness occurring during anesthesia for neurosurgical operations. Arch Ophthalmol 52:819– 830
- Hoski JJ, Eismont FJ, Green BA (1993) Blindness as a complication of intraoperative positioning. J Bone Joint Surg [Am] 75:1231–1232
- 24. Huber JF, Grob D (1998) Bilateral cortical blindness after lumbar spine surgery. Spine 23:1807–1809
- Johnson MW, Kincaid MC, Trobe JD (1987) Bilateral retrobulbar optic nerve infarctions after blood loss and hypotension. Ophthamology 94:1577–1584
- 26. Jonas JB, Gusek GC, Naumann OH (1988) Anterior ischemic optic neuropathy: nonarteritic form in small and giant cell arteritis in normal sized optic discs. Int Ophthalmol 12:119–125

- 27. Katz DM, Trobe JD, Cornblath WT, Kline LB (1994) Ischemic optic neuropathy after lumbar spine surgery. Arch Ophthalmol 112:25–31
- Katzman SS, Moschnas CG, Dzioba RB (1994) Amaurosis secondary to massive blood loss after lumbar spine surgery. Spine 19:468–469
- 29. Kollarits CR, McCarthy RW, Corrie WS, Swann ER (1981) Norepinephrine therapy of ischemic optic neuropathy. J Clin Neuro Ophthalmol 1:283–288
- Laibovitz, RA (1982) Reversible ischemic optic neuropathy in severe anemia. Tex Med 78:56–58
- 31. Lam AK, Douthwaite WA (1997) Does the change of anterior chamber depth or/and episcleral venous pressure cause intraocular pressure change in postural variation?. Optom Vis Sci 74:664–667
- Lee AG (1995) Ischemic optic neuropathy following lumbar spine surgery. J Neurosurg 83:348–349
- 33. Lee LA (2003) ASA postoperative visual loss registry: preliminary analysis of factors associated with spine operations. ASA Newslett 67:7–8
- Lee LA, Lam AM (2001) Unilateral blindness after prone lumbar spine surgery. Anesthesiology 95:793–795
- 35. Mansour AM, Shoch D, Logani S (1988) Optic disk size in ischemic optic neuropathy. Am J Ophthalmol 106:587–589
- 36. Myers M, Hamilton S, Bogosian A et al (1997) Visual loss as a complication of spine surgery: a review of 37 cases. Spine 22:1325–1329
- Quigley HA, Miller NR, Green WR (1985) The pattern of optic nerve fiber loss in anterior optic neuropathy. Am J Ophthalmol 100:769–776
- Repka MX, Savino PJ, Schatz NJ (1983) Clinical profile and long term implications of anterior ischemic optic neuropathy. Am J Ophthamol 96:478– 483
- Rootman J, Butler D (1952) Ischemic optic neuropathy—a combined mechanism. Br J Ophthalmol 64:826–831
- 40. Roth S, Gillesberg I (1999) Anesthesia and perioperative complications. Mosby, St. Louis
- Roth S, Thisted RA, Erickson JP et al (1996) Eye injuries after nonocular surgery: a study of 60,965 anesthetics from 1988 to 1992. Anesthesiology 85:1020– 1027
- Russell RW, Bharucha N (1978) The recognition and prevention of border zone cerebral ischemia during cardiac surgery. Q J Med 187:303–323

- 43. Sadun AA (1983) The efficacy of optic nerve sheath decompression for anterior ischemic optic neuropathy and other optic neuropathies. Am J Ophthalmol 115:384–389
- 44. Slavin ML, Barondes MJ (1988) Ischemic optic neuropathy in sickle cell disease. Am J Ophthalmol 105:212–213
- 45. Slocum HC, O'Neal KC, Allen CR (1948) Neurovascular complications from malposition on the operating table. Surg Gynecol Obstet 86:729–734
- Stevens WR, Glazer PA, Kelley SD et al (1997) Ophthalmic complications after spinal surgery. Spine 22:1319–1324
- 47. Sweeney PJ, Breuer AC, Selhorst JB et al (1982) Ischemic optic neuropathy: a complication of cardiopulmonary bypass surgery. Neurology 32:560–562
- Tice DA (1987) Ischemic optic neuropathy and cardiac surgery. Ann Thorac Surg 44:677
- 49. Tsamparlakis J, Casey TA, Howell W, et al (1980) Dependance of intraocular pressure on induced hypotension and posture during surgical anesthesia. Trans Ophthalmol Soc UK 100:521–526
- Walkup HE, Murphy JD (1952) Retinal ischemia with unilateral blindness—a complication occurring during pulmonary resection in the prone position. Report of two cases. J Thorac Surg 23:174–175
- Warner ME, Warner MA, Garrity JA et al (2001) The frequency of perioperative vision loss. Anesth Analg 93:1417– 1421
- Weinstein JM, Duckrow RB, Beard D, Brennan RW (1983) Regional optic nerve blood flow and its autoregulation. Invest Ophthalmol Vis Sci 24:1559– 1565
- West J, Askin G, Clarke M, Vernon SA (1990) Loss of vision in one eye following scoliosis surgery. Br J Ophthalmol 74:243–244
- Williams E, Hart W, Tempelhoff R (1995) Postoperative ischemic optic neuropathy. Anesth Analog 80:1018– 1029
- 55. Wilson JF, Freeman SB, Breene DP (1991) Anterior ischemic optic neuropathy causing blindness in the head and neck surgery patient. Arch Otolaryngol Head Neck Surg 117:1304–1306
- 56. Wolfe SW, Lospinuso MF, Burke SW (1992) Unilateral blindness as a complication of patient positioning for spinal surgery. Spine 17:600–605