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TOPIC HIGHLIGHT

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Perioperative visual loss after spine surgery

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Abstract

Perioperative visual loss (POVL) is an uncommon, but devastating complication that remains primarily associated with spine and cardiac surgery. The incidence and mechanisms of visual loss after surgery remain difficult to determine. According to the American Society of Anesthesiologists Postoperative Visual Loss Registry, the most common causes of POVL in spine procedures are the two different forms of ischemic optic neuropathy: anterior ischemic optic neuropathy and posterior ischemic optic neuropathy, accounting for 89% of the cases. Retinal ischemia, cortical blindness, and posterior reversible encephalopathy are also observed, but in a small minority of cases. A recent multicenter case control study has identified risk factors associated with ischemic optic neuropathy for patients undergoing prone spinal fusion surgery. These include obesity, male sex, Wilson frame use, longer anesthetic duration, greater estimated blood loss, and decreased percent colloid administration. These risk factors are thought to contribute to the elevation of venous pressure and interstitial edema, resulting in damage to the optic nerve by compression of the vessels that feed the optic nerve, venous infarction or direct mechanical compression. This review will expand on these findings as well as the recently updated American Society of Anesthesiologists practice advisory on POVL. There are no effective treatment options for POVL and the diagnosis is often irreversible, so efforts must focus on prevention and risk factor modification. The role of crystalloids versus colloids and the use of α-2 agonists to decrease intraocular pressure during prone spine surgery will also be discussed as a potential preventative strategy.

Key words: Perioperative visual loss; Ischemic optic neuropathy; Central retinal artery occlusion; Cortical blindness; Posterior reversible encephalopathy; Spine surgery; Prone positioning

Core tip: Perioperative visual loss (POVL) is an uncommon, but devastating complication that remains primarily associated with spine and cardiac surgery. The incidence and mechanisms of visual loss after surgery remain difficult to determine. Ischemic optic neuropathy accounts for the vast majority of these cases, with retinal ischemia, cortical blindness, and posterior reversible encephalopathy observed with low incidence. Recently identified risk factors include obesity, male sex, Wilson frame use, longer anesthetic duration, greater estimated blood loss, and decreased percent colloid administration. POVL is often permanent and untreatable, so prevention is key to limiting its impact.

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INTRODUCTION

Perioperative visual loss (POVL) associated with spine surgery is a rare and disastrous complication that is generally irreversible and without definitive etiology.

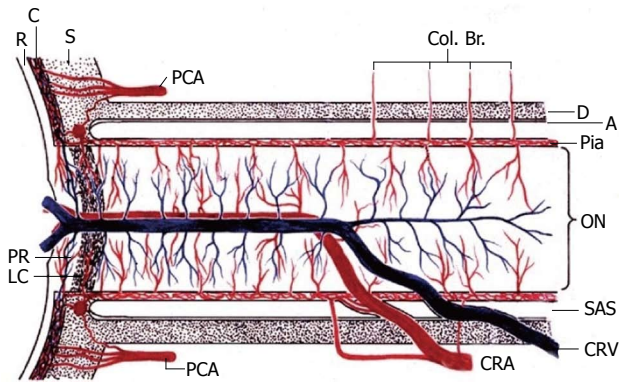

Figure 1 Schematic representation of blood supply of the optic nerve. Reproduced from Hayreh *et al*[20]. A: Arachnoid; C: Choroid; CRA: Central retinal artery; Col. Br.: Collateral branches; CRV: Central retinal vein; D: Dura; LC: Lamina cribrosa; ON: Optic nerve; P: Pia; PCA: Posterior ciliary artery; PR: Prelaminar region; R: Retina, S: Sclera; SAS: Subarachnoid space.

First described by Hollenhorst *et al*[1] in 1954, there have been numerous reports since establishing a clear link between spine surgery in the prone position and vision loss. Unfortunately, however, the research on this topic is limited due to its rare occurrence and consists largely of individual case reports and series[2-5]. This article reviews the different types of postoperative visual loss complications after spine surgery. The theoretical pathogenesis, risk factors, and prevention strategies including the use of colloids versus crystalloids and α-agonists to decrease intraocular pressure (IOP) are also discussed.

EPIDEMIOLOGY

Vision loss occurring with spine surgery may result from: anterior ischemic optic neuropathy (AION) or posterior ischemic optic neuropathy (PION); central retinal artery occlusion (CRAO); cortical blindness; and posterior reversible encephalopathy (PRES). Two large retrospective studies determined that the incidence of POVL is approximately 1/60000 to 1/125000 of all general anesthetics[6,7]. However, the risk of POVL is believed to be significantly greater following cardiac and spine surgeries. A recent review by Shen and colleagues of 5.6 million patients from the National Inpatient Sample (NIS) found that the incidence of POVL to be 3.09/10000 (0.03%) after spinal fusion and 8.64/10000 (0.09%) after cardiac surgery[5]. Other large-scale series suggest that the rate of POVL may be even higher after spine surgery, with incidence rates ranging from 0.094%[4] to 0.2%[8]. Visual loss was more common after spinal fusion for scoliosis and posterior lumbar fusion than anterior lumbar fusion or cervical fusion[4]. It was also noted to be significantly increased in hip and femur operations (1.86/10000, or 0.19%)[5]. These procedures share several features including large blood loss, hemodynamic perturbations, high embolic loads, and significant inflammation.

According to the American Society of Anesthesiologists (ASA) Postoperative Visual Loss Registry, the most common causes of POVL in spine procedures are the two different forms of ischemic optic neuropathy (ION): AION and PION, accounting for 89% of the cases[9]. PION was diagnosed in 60% of these cases[9]. In this database, CRAO only accounted for 11% of the cases.

According to the most recent NIS review, gender plays an important role with men displaying a higher risk of POVL after spinal fusion relative to women (OR = 1.75), which is consistent with the ASA POVL Registry[9], previous case series[10], and a recent multicenter case-control study[11]. Age appears to be a factor as well with those aged 50-64 years displaying an increased risk (OR = 1.75). Also notable and unexplained was the finding that children < 18 years old had the highest overall risk for POVL (OR = 6.91), which was primarily attributed to cortical blindness rather than AION/PION and may represent a different etiology[5].

ANATOMY

Blood supply to the optic nerve

In order to understand POVL, especially ION, it is important to have a basic understanding of the blood supply to the optic nerve (Figure 1). For an exhaustive review, please see Hayreh[12], 2001. The ophthalmic artery, originating from the internal carotid artery, and its various branches is the principal blood supply to the retina, globe, and optic nerve. The central retinal artery, a branch of the ophthalmic artery, supplies the inner retina.

The anterior portion of the optic nerve (optic nerve head) has a rich arterial supply principally from the posterior ciliary artery (PCA) circulation, except for the surface nerve fiber layer, which is supplied by the retinal circulation. The blood supply in the optic nerve head has a sectorial distribution, which may explain the segmental vision loss seen in ischemic disorders[13].

The posterior portion of the optic nerve is supplied by the pial vascular plexus, which is supplied by multiple pial branches originating from the peripapillary choroid, circle of Haller and Zinn, central retinal artery, ophthalmic artery, and other orbital arteries[13].

In contrast to the densely supplied anterior and posterior portions of the nerve, the central portion within the optic canal is supplied only by the pial vascular plexus derived from arterial extensions of the anterior and posterior blood supplies and intraneural branches of the central retinal artery. This comparatively sparse vascular supply to the mid portion of the optic nerve renders it more susceptible to ischemia and it is this portion of the nerve that is though to be related to PION[14]. However, it is important to note that there is significant interindividual variability in the complex blood supply to the optic nerve, especially in terms of the location and pattern of watershed zones[12].

Venous drainage occurs mostly via the central retinal vein that is drained by the internal jugular vein. In the pre-laminar region of the eye, there are retinociliary collaterals to the peripapillary choroidal veins and drainage through these collaterals can become significant in case

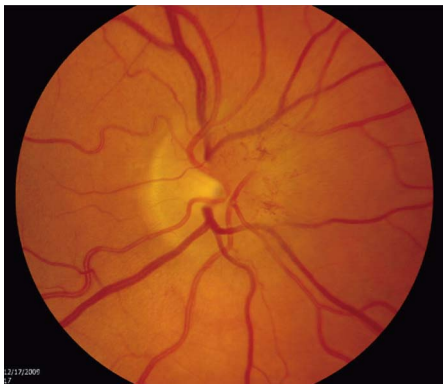

Figure 2 Fundoscopic exam of acute anterior ischemic optic neuropathy demonstrating blurring of the optic disk margin from edema. Lee LA, Mudumbai R. Postoperative visual loss. In: Ehab Farag, editor. Anesthesia for Spine Surgery. Cambridge University Press, 2012.

of central retinal vein thrombosis[14].

VISION LOSS AFTER SPINE SURGERY

Ischemic optic neuropathy

Postoperative ION is a devastating complication that can occur after a variety of surgical procedures, most often following cardiothoracic surgery[15], instrumented spinal fusion[16,17], and head and neck surgery[18]. ION can be categorized as either anterior or posterior, depending on whether the insult occurs in the anterior or posterior portion of the optic nerve. The type of ION observed varies depending on the type of surgery performed, with AION occurring most frequently after cardiac surgery and PION occurring most frequently after spine surgery in the prone position or radical neck dissection[9].

Anterior ischemic optic neuropathy

AION is likely caused by occlusion or hypoperfusion of the anterior optic nerve head by the PCAs and typically presents with sudden onset painless vision loss and a visual field defect. It is distinguished on fundoscopy by diffuse or segmental disc edema with ensuing atrophy and sometimes splinter hemorrhages around the optic disc (Figure 2)[19,20]. AION can be further classified as either arteritic or nonarteritic. Arteritic AION is rarely found perioperatively. It is caused by temporal arteritis and often presents in the elderly with an elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), markers that are entirely non-specific in the postoperative period[21].

Nonarteritic AION occurs both spontaneously in the community and in the perioperative setting, often in patients with pre-existing vascular disease[22]. Additional risk factors include diabetes mellitus, arterial hypotension, arterial hypertension, blood loss, prone positioning during surgery, prolonged surgery, atherosclerosis, sleep apnea, and migraine[13]; however, it can occur in patients that are otherwise healthy. The pathology is likely a combination of these factors, perhaps together with abnormal autoregulation and other patient specific characteristics that predispose to ischemic injury[23]. Perioperative nonarteritic AION is most often associated with cardiac surgery, especially CABG, and generally presents immediately upon awakening from surgery. On rare occasion, AION may occur abruptly after a "delay" or period of normal vision lasting hours to days[24].

Posterior ischemic optic neuropathy

Posterior ION results from infarction of the optic nerve posterior to the lamina cribrosa and also manifests as sudden onset painless visual loss and visual field deficiencies. In contrast to AION, the fundoscopic examination initially reveals a completely normal appearing fundus, with optic nerve pallor and atrophy occurring only after approximately 4-6 wk[24]. It tends to cause significant bilateral visual loss or complete blindness and is usually discovered on waking from the surgical procedure[13]. In the ASA Registry of spine-related ION, 46% of the patients reported had no light perception[9], which is usually permanent. Like AION, PION may also be classified as either arteritic or nonarteritic. The arteritic form is attributable to temporal arteritis and the nonarteritic form is seen most commonly following spine surgery.

A host of hemodynamic derangements could contribute to the development of postoperative PION including: hypotension, anemia, increased venous pressure, prone positioning during surgery, increased cerebrospinal fluid, and direct ocular compression[25]. Anemia and hypotension are almost always observed in patients that develop postoperative PION[26]. The pial vessels that supply the posterior optic nerve lack an autoregulatory mechanism, rendering them susceptible to ischemia during periods of hypotension and when the blood oxygen carrying capacity is decreased[27]. However, studies comparing patients with POVL after spine surgery with those of controls demonstrated no difference in perioperative hematocrit and blood pressure, suggesting a multifactorial cause[10,28].

The prone position, a key element to spine surgery, is also the setting in which the majority of postoperative PION is observed. Prone positioning, especially when in the Trendelenburg position, leads to increased orbital venous pressure through an increase in abdominal venous pressure, thus increasing resistance to local blood flow[29]. Direct orbital pressure, often seen with face pillows/cushions or other positioning devices, has also been implicated in the pathogenesis of PION. However, with the resultant decreased perfusion pressure to the optic nerve head and central retinal artery, AION or CRAO would be more likely observed[26]. Avoidance of the prone position and direct ocular pressure is insufficient, however, to prevent postoperative PION, as cases have been documented following surgery in the supine position and with the use of head pins[3,9,30].

Risk factors associated with ischemic optic neuropathy and spine surgery

Recently in 2012, the Postoperative Visual Loss Study

Group published a multicenter case-control study that explored the risk factors for ION after spinal fusion surgery in the prone position[11]. Prior studies of ION after spine surgery were limited by small numbers without appropriately matched controls or by lack of associated intraoperative data (estimated blood loss, fluids administered, type of surgical frame, case duration, *etc.*)[4,5,10]. This study comparing 80 cases from the ASA Postoperative Visual Loss Registry to 315 controls from 17 institutions throughout the United States addressed these shortcomings. Obesity, male sex, Wilson frame use, longer anesthetic duration, greater estimated blood loss, and decreased percent colloid administration were significantly and independently associated with ION after spinal fusion surgery[11].

Theoretical mechanisms for ischemic optic neuropathy after prone spine surgery

The most popular pathophysiologic explanations used today for ischemic optic neuropathy during prone position are the elevation of venous pressure and development of interstitial edema[11]. Theoretically, these two processes can cause damage to the optic nerve by compression of the vessels that feed the optic nerve, venous infarction or direct mechanical compression. A rise in central venous pressure can occur in obese patients when their abdomen is compressed during prone position. Venous pressure can also elevate when the head position is lower than the heart, a given when patients are placed in the Wilson frame. Lower oncotic pressure leading to a growing interstitial edema can occur when there is significant inflammation and capillary leak such as in situations of major blood loss and/or prolonged cases. The same can occur when less colloid is used overall. Thus far, these explanations are simply theories that require further investigation. Why the male sex appears to be a risk factor for ischemic optic neuropathy during prone position is still a puzzle, but it has been suggested that estrogen may serve a protective role[31].

Retinal ischemia: Branch and central retinal artery occlusion

Central retinal artery occlusion (CRAO) decreases blood supply to the entire retina, whereas occlusion of a retinal branch (BRAO) affects only a portion of the retina. Both are ophthalmic emergencies and analogous to an acute stroke of the eye. Retinal ischemia has been documented in both adults and children following ocular trauma[32], and also embolic[33] and vasospastic episodes[34].

With respect to spine surgery, these conditions are mostly commonly seen during the perioperative period from improper patient positioning and external compression on the eye[35]. Of the 93 cases submitted to the ASA Visual Loss Registry, there were 10 cases of CRAO[9], representing a much smaller percentage than ION. Perioperative trauma was noted in 70% of the cases, as evidenced by corneal abrasion, ipsilateral decreased supraorbital sensation, ophthalmoplegia, ptosis, or unilateral erythema[9].

Theoretical mechanisms that have been used to explain CRAO include thromboembolism, direct pressure to the globe, and increased intraocular pressure. Decreased oxygen carrying capacity and blood flow to optic nerve such as from hypovolemia, anemia, large blood loss, and peripheral vascular disease, have also been suggested etiologic factors for CRAO. The use of horseshoe-shaped headrest has been associated with this complication. Hollenhurst *et al*[1] described CRAO in eight patients after prone spine surgery on horseshoe headrest. In fact CRAO in spine surgery was subsequently referred to as "headrest syndrome[22]". Increased risk is also observed in patients with altered facial anatomy, osteogenesis imperfecta, and exophthalmos, all of which can increase effects of external compression[36].

CRAO is often unilateral in presentation with severe visual loss in the affected eye. Patients are found to have a cherry-red spot on the macula, a white ground-glass appearance of the retina, attenuated arterioles, and an afferent pupillary defect[37]. Visual loss from CRAO is almost always irreversible and there are no established effective treatment options.

Cortical blindness

Cortical blindness is the result of decreased perfusion to the occipital cortex by the posterior cerebral artery. The cause is either hypoperfusion or embolic phenomenon. Patients with cortical blindness have normal light reflex and fundoscopic examination as the optic tracts and radiations are unaffected. When one side is affected, the patient presents with contralateral homonymous hemianopsia. If both sides suffer ischemic insult, the patient may have peripheral vision loss or complete blindness. Cortical blindness may improve initially after the infarct, but total recovery is rare.

PRES

PRES is a neurologic syndrome that presents as a combination of seizures, visual changes, vomiting, headache, and decreased level of consciousness. It is associated with acute medical illnesses such hypertensive episodes, autoimmune disease, malignancy, chemotherapy, immunosuppressant therapy, infection, renal disease, vasculitis, eclampsia, and preeclampsia[38]. Although more closely identified with obstetric patients, PRES has also been reported after lumbar fusion[39], hysterectomy[40] and video-assisted-thoracoscopic wedge resection[41]. PRES has characteristic MRI findings. There are two leading theoretical explanation for PRES. One is acute increase in blood pressure above the brain's autoregulatory limit thereby causing brain edema. The other pathophysiologic explanation is cytotoxic drugs or diseases causing endothelial injury and edema formation. Management is appropriate use of anti-seizure and anti-hypertensive agents and treatment of causative factor(s). Unlike ION and CRAO, PRES has a favorable recovery pattern.

TREATMENT AND PREVENTION OF POVL

When a patient reports any visual symptoms following

Table 1 American Society of Anesthesiologists perioperative visual loss practice advisory consensus conclusions

There is a subset of patients who undergo spine procedures while they are positioned prone and receiving general anesthesia that has an increased risk for the development of POVL. This "high-risk" subset includes patients who are anticipated preoperatively to undergo procedures that are prolonged, have substantial blood loss, or both
Consider continuous blood pressure and central venous pressure monitoring in high-risk patients
Consider informing high-risk patients that there is a small, unpredictable risk of POVL
The use of deliberate hypotensive techniques during spine surgery has not been shown to be associated with the development of POVL
Colloids should be used along with crystalloids to maintain intravascular volume in patients who have substantial blood loss
At this time, there is no apparent transfusion threshold that would eliminate the risk of POVL related to anemia
High-risk patients should be positioned so that their heads are level with or higher than the heart, when possible. In addition, their heads should be maintained in a neutral forward position (without significant neck flexion, extension, lateral flexion, or rotation) when possible
Consideration should be given to the use of staged spine procedures in high-risk patients

POVL: Perioperative visual loss.

surgery, an urgent ophthalmologic consultation should be obtained to determine its cause. If an apparent ocular injury or central retinal artery occlusion is not obvious, neuroimaging should be obtained, preferably MRI with gadolinium to assess for intracranial pathologies, including occipital stroke or pituitary apoplexy[42]. If imaging is negative, the most likely etiology is ION. Treatment has often involved high dose steroids, mannitol or other agents to decrease intraocular pressure, and anti-platelet agents; however, none of these approaches have been shown to be effective[13,24,43].

Our group recently examined the effect of crystalloid versus colloid and the use of the α-agonist Brimonidine on IOP during prone spine surgery[44]. Of note, the mean rate of IOP rise in the prone position and mean IOP at the end of surgery was significantly greater in patients receiving crystalloid than those receiving colloid. Topical Brimonidine also led to a significant reduction in IOP, both intraoperative and postoperative. Ocular perfusion pressure, however, did not vary significantly between the groups as hypotension was aggressively treated, suggesting that maintenance of blood pressure may be a more important factor in determining perfusion pressure. Much larger studies are needed to determine whether maintaining appropriate ocular perfusion pressure reduces the risk of POVL after spine surgery.

Given the poor prognosis and lack of validated treatment options, it is essential to take prophylactic measures during surgery to prevent the development of POVL. The ASA Task Force on Perioperative Blindness, consisting of anesthesiologists, neuro-ophthalmologists, and spine surgeons was formed in 2005 to evaluate the literature and develop a practice advisory to help deal with this issue. In 2006, a "practice advisory" was published and the consensus conclusions are listed in Table 1[42]. Other guidelines found in this advisory as well as the update published in 2012[43], suggest periodically checking hemoglobin and hematocrit values, and avoidance of direct pressure on the globe to avoid CRAO injuries. A variety of commercially available devices are available to help limit mechanical ocular compression during prone surgery, but these still require vigilance on the part of the surgeon and anesthesiologist as patient movement and shifting of the device may occur. If POVL is suspected, additional efforts directed towards optimizing hemoglobin/hematocrit values, hemodynamic status, and systemic oxygenation may be appropriate[43].

PERIOPERATIVE VISUAL LOSS IN OTHER SURGERIES

Perioperative visual loss has also been associated with robotic and laparoscopic surgeries. Cases of visual impairment have been reported to occur in minimally invasive proctocolectomy, laparoscopic nephrectomy and robotic prostatectomy[45-48]. During robotic prostatectomy, increased intraocular pressure occurs due to prolonged duration in steep Trendelenburg position combined with CO_2 insufflation of the abdomen. The central venous pressure within the thorax increases with Trendelenburg position, which may reduce drainage of blood flow from the head, thereby leading to elevation in IOP. During CO_2 insufflation, the increase in intra-abdominal pressure will further augment the increase in intrathoracic pressure. Furthermore, insufflation of CO_2 increases the carbon dioxide in the blood, which can lead to cerebral vasodilatation and increased cerebral blood volume. The end result is elevation in venous pressure. It is unknown whether the same risk factors for POVL in spine surgery can be applied to laparoscopic and robotic surgeries, but it appears venous congestion and interstitial edema are commonalities among these surgeries. As robotic surgeries gain popularity, studies to find population at risk are underway. Conservative management, however, with attempts to decrease venous congestion and interstitial edema would seem appropriate.

CONCLUSION

In summary, POVL in spine surgery is extremely rare, but it remains a dreaded complication despite significant efforts to identify risk factors and a pathophysiological mechanism. Potential causes of POVL after spine surgery include anterior ischemic optic neuropathy, posterior ischemic optic neuropathy, cortical blindness, retinal isch-

emia, and posterior reversible encephalopathy syndrome. The vast majority of cases are related to ischemic optic neuropathy. Many reports have attempted to link hypotension, anemia, and blood loss to the development of this disease; however, no single mechanism can entirely explain the varied circumstances in which it occurs. This suggests a multifactorial etiology and perhaps individual susceptibility related to varied optic nerve blood supply and anatomy.

In the largest and most comprehensive study to date, the Postoperative Visual Loss Group, using data from the ASA Post Operative Visual Loss Registry, identified obesity, male sex, Wilson frame use, longer anesthetic duration, greater estimated blood loss, and decreased percent colloid administration as significant independent risk factors for the development of ION. These risk factors, with the possible exception of male sex, are thought to promote a rise in venous pressure and interstitial edema limiting optic nerve perfusion. Further studies will hopefully elucidate whether the use of colloid and/or topical a-agonists to limit the rise in IOP during complex prone spine surgeries is important in maintaining ocular perfusion and reducing the incidence of POVL.

Given the complete lack of effective treatment modalities, prevention is crucial for limiting the incidence and destruction of POVL. Practitioners are encouraged to follow the ASA guidelines listed in Table 1, especially for patients identified as high risk undergoing procedures that are known to result in visual loss.

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