# Incidence, risk factors, and management of blindness after orbital surgery

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**Running Head:** Blindness after orbital surgery

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## Abstract

Purpose: Severe vision loss is a risk of orbital surgery which physicians should prepare for and counsel patients about, but the overall risk rate is unknown. This research was conducted to determine the risk of severe vision loss related to surgery within the orbit.

Design: Retrospective review

Subjects: Patients who underwent orbital surgery at either of two academic medical centers between January 1994 and December 2014.

Methods: A billing database search was conducted to identify all patients who had undergone orbital surgery during the study period, cross-checked against diagnostic codes related to vision loss. Charts were screened to determine baseline demographic and medical history, surgical procedure, intra- and perioperative management, and visual acuity. Patients with preoperative visual acuity ≥20/200 that had worsened to ≤20/400 after orbital surgery were included for detailed review. Statistical analysis was conducted to identify factors posing particular risk or benefit to the visual outcome in these cases.

Main Outcome Measures: Visual acuity after orbital surgery

Results: A total of 1665 patients underwent orbital surgery during the inclusion period, with 14 patients sustaining severe vision loss ranging from counting fingers at 1 foot to no light perception (overall risk=0.84%). Etiology of vision loss included retrobulbar hemorrhage, malpositioned implant, optic nerve ischemia, or direct optic nerve insult. When stratified by surgical approach, the risk of a blinding surgical complication was significantly higher for patients undergoing orbital floor repair in the setting of multiple facial fractures (subgroup risk=6.45%), bony decompression of the optic canal (subgroup risk=15.6%), or intracranial approach to the orbital roof (subgroup risk=18.2%). Seven of 8 patients with a potentially-reversible etiology of postoperative vision loss were returned to the operating room urgently, and 2 regained substantial vision (20/20 and 20/25). Administration of intravenous corticosteroids had no significant effect on visual acuity outcome.

Conclusions: The overall risk of severe vision loss after orbital surgery is 0.84%. The subgroup risk is higher in patients undergoing facial polytrauma repair, optic canal decompression, or orbital apex surgery from an intracranial approach. Close postoperative monitoring and urgent assessment and management of acute vision loss may improve visual outcome in some cases.

Blindness is a devastating complication following orbital surgery, and one that is discussed with patients in the preoperative informed consent process. The overall incidence of blindness due to orbital surgery, however, has not been thoroughly studied. Published case reports describe vision loss occurring after fracture repairs, partial or total removal of orbital mass lesions, and orbital decompression surgeries.[1-5] A few larger case series have addressed postoperative vision loss rates for specific subpopulations or surgical case types.[6-10] The reported incidence of postoperative blindness varies from 0-24% in the literature, likely due in part to the different risk profiles of the cases addressed by each series.[7-8] The variability in the available literature makes patient selection, operative timing, and preoperative risk counseling difficult.

The goal of this study was to assess the incidence rate and risk factors for postoperative vision loss occurring as a complication of orbital surgery, using data accumulated over an extended period of time from a tertiary academic medical center (AMC). As a surgical setting, several institutional features of the AMC may uniquely influence outcomes: multiple specialties operate within the orbit (Ophthalmology, Otolaryngology, Oral Maxillofacial Surgery, Plastic Surgery, Neurosurgery), multiple surgeons within each specialty, and surgeons-in-training (medical students, residents, fellows) are involved in the peri- and intraoperative management. Given the frequent referral of severe traumas and complex cases to AMCs for surgical intervention in addition to the routine cases, a better understanding of the risk of blindness after orbital surgery in this setting would be of value.

## Methods

Institutional Review Board approval was obtained, and all research was carried out in compliance with HIPAA regulations and in accordance with the Declaration of Helsinki. This was a retrospective chart review study conducted at the University of Washington's two major urban hospitals, one of which is the only Level I trauma center for a 5-state catchment area encompassing Washington, Wyoming, Alaska, Montana and Idaho. Patients seen between January 1994 and December 2014 were eligible for inclusion. The medical center's billing database was searched for Current Procedural Terminology codes for orbitotomy (CPT: 21077, 21172, 21175, 21179, 21180, 21182-21184, 21260-21263, 21267, 21268, 21385-21397, 21390, 21395, 21400, 31075, 31225, 31292, 31293, 61580, 61581, 67400, 67405, 67412-67414, 67420, 67430, 67440, 67445, 67450) and ICD-9 procedure codes (16.09, 16.23, 16.92) to identify patients who had undergone orbital surgery for any indication. These records were subsequently cross-checked with a search of International Classification of Disease-9 codes for vision loss, visual field disturbance, blindness (369.xx), or optic neuropathy (377.xx).

The resulting records were screened with chart review to identify patients who had experienced significant visual loss (defined as final visual acuity of ≤ 20/400 in the affected eye) during the acute or subacute postoperative period (≤ 14 days) after orbitotomy. Patients were excluded if preoperative vision was already ≤ 20/200 (threshold for legal blindness), or if preoperative vision could not be determined. When a patient's preoperative visual acuity had not been documented in the hospital chart, effort was made to verify baseline visual acuity by other historic means such as records from their private eyecare clinician.

Chart review data collected for each patient included demographics, past medical and ocular history, surgical indication, and preoperative ophthalmic exam findings. Intraoperative details of the procedure were extracted, including surgical findings, implants, relevant medication infusions (corticosteroids, antibiotics, osmotics), anticoagulation status, and any surgical or anesthesia complications. Postoperative data included medications, postoperative eye exam (visual acuity, pupils, extraocular motility), and postoperative imaging results. Details of each patient's vision loss were also collected, including time of onset, signs or symptoms leading to diagnosis, and how it was treated medically and/or surgically. The total postoperative follow-up duration and final visual acuity were noted.

Descriptive statistics were calculated for demographics, incidence rates, and follow-up duration. Statistical analysis for comparison of subgroup incidence rates was performed with MedCalc for Windows, version 15.1 (MedCalc Software, Ostend, Belgium). Postoperative visual acuity outcomes in steroid versus non-steroid groups were compared with a two-sample T-test.

## Results

During the period from January 1994 through December 2014, a total of 1665 patients underwent orbitotomy at the University of Washington Medical Center hospitals. Of these, 198 patients had been assigned ICD-9 diagnostic codes for optic nerve and/or visual dysfunction. These charts were thoroughly reviewed to identify 17 patients with visual acuity which had declined to ≤20/400 after surgery. Two of these patients were excluded due to lack of documented or verifiable preoperative visual acuity: one was a trauma patient who could not undergo subjective preoperative ophthalmologic exam due to intubation at the scene, while the other was an ambulatory surgery patient with no previous record of eye care in any setting. Finally, 1 patient was excluded because fulminant spread of a pre-existing orbital infection was the proximal cause of blindness, rather than the orbital surgery itself. As such, 14 patients were included in the full review, representing an overall incidence rate of 0.84%.

The surgical approaches for 1665 patients who underwent orbitotomy during the 20-year review period are summarized in **Table 1**, along with the postoperative vision loss incidence rate for each approach, which ranged from 0 – 18.2%. Postoperative vision loss occurred in 5 of the 32 orbitotomy cases in which the bony optic canal was surgically opened and/or decompressed (15.6%), with no statistical difference between intracranial approach (n=4 of 22, 18.2%) versus endoscopic trans-sphenoidal approach to the optic canal (n=1 of 10, 10%), ($p$=0.587, 95% CI - 0.21 to 0.38). Vision loss was significantly more likely after combined-approach orbital fracture repairs (6.45%, $p$=0.002), or after craniotomy with orbitotomy (18.2%, $p$ <0.0001), compared to the cohort's overall incidence rate. Lateral orbitotomy did not pose a significantly different risk of vision loss compared to anterior orbitotomy ($p$=0.428, 95% CI 0.13 to 24.8).

Among the 14 patients with significant vision loss after orbitotomy, there were 8 females (57%) and 6 males (43%), average age was 47.8 years (range: 30-69 years), with the right eye involved in 9 cases and the left in 5 cases. A summary of each patient's surgical and postoperative course is given in **Table 2**. Postoperative vision checks were conducted by a physician from the surgical service that performed the procedure, in the post-anesthesia care unit when the patient awakened (range: 30 minutes to 2 hours postoperatively). Vision checks were continued by nursing staff every 6-8 hours thereafter if the patient was admitted to the hospital. Patient #10 experienced profound periocular edema related to a periscapular microvascular free flap that was placed on his cheek, which impeded routine vision checks for several days. Postoperative pupil exams were not consistently documented to have occurred in any patient. All of the fracture repairs with low vision outcomes were performed within 4 days of injury (average 1.8 days, range 8 hours to 3.6 days). Average total follow-up after a vision loss event was 20.4 months (range: 1-71 months).

The etiology of vision loss was concluded to be retrobulbar hemorrhage (RBH) in 4 cases (29%), malpositioned implant in 2 (14%), ischemic event in 2 (14%), optic nerve compression due to expansion of a hemostatic packing agent in 1 (7%), seroma in 1 (7%), direct optic nerve injury in 1 (7%), and uncertain in 3 (21%). While there were no reported adverse anesthesia events among the 14 patients with vision loss, review of anesthesia records found 7 patients experienced at least one episode of systolic blood pressure <80mmHg during surgery, 8 patients experienced at least one episode of mean arterial pressure decrease >30% from baseline, and 5 patients experienced both events (#1, 5, 8, 11, 14). The vast majority of such hypotensive episodes lasted 5 minutes or less. No patient experienced systolic blood pressure <80mmHg for greater than 10 minutes. Two patients experienced mean arterial pressure decrease >30% from baseline for 30 minutes, one of whom suffered ischemic vision loss (#14) while the other patient's mechanism of vision loss could not be definitively identified (#11).

Retrobulbar hemorrhage occurred as early as upon awakening or as late as 2 days after surgery. Review of anesthesia records found all RBH patients had been extubated with positive airway pressure, with no supplemental pressure applied to the orbit during awakening. Bucking was documented in one RBH patient (#3) who desaturated due to secretions on the initial extubation attempt and was therefore extubated 3-4 hours later in the intensive care unit without complications. All 4 of the vision-compromising RBH events occurred in patients who had undergone orbital fracture repair with implants. Three of the 4 had required screw-fixated hardware placement. One patient with RBH (#5) had been on daily 81mg aspirin leading up to surgery and appears to have continued it uninterrupted perioperatively, while the others received no anticoagulants before or after surgery.

In 8 patients, the etiology of vision loss involved some form of postoperative compression of the optic nerve (oxycel expansion, RBH, seroma accumulation, malpositioned implants), which could potentially be surgically addressed. Seven of these patients were urgently returned to the operating room, 2 of whom regained substantial vision (patient #1 to 20/20, and #4 to 20/25), and 1 of whom regained enough vision to perceive hand motions (patient #6). The patient who was nonoperatively managed (patient #3) was noted to have low vision at a postoperative bedside check 6 hours after surgery. CT scan showed a modest-sized extraconal orbital hemorrhage along a well-positioned plate. The patient's visual acuity was attributed to sleepiness and thick ophthalmic ointment. He was discharged without intervention, and had no light perception (NLP) at all subsequent outpatient postoperative follow-up exams. Spontaneous improvement was seen in 4 of 6 non-operatively managed cases (patients #7, 11, 12, and 14), but the improvement was limited to small gains to the level of light perception or counting fingers.

Intravenous steroids were given to 9 of the 14 patients, with the first dose administered a mean of 2.5 hours after initial diagnosis of vision loss (range 0.5 hours to 6 hours after diagnosis; 0.5 hours to 6 days postoperatively). There was no significant difference in the visual acuity outcomes for patients who received steroids (median NLP, range NLP to 20/20) versus those who did not (median LP, range NLP to 20/25), $p$=0.765.

## Discussion

Vision loss is the most severe complication directly associated with orbital surgery. As such, careful preoperative counseling is needed regarding that risk.[11] The literature, however, contains limited data on the incidence rate of vision loss after orbital surgery beyond case reports or case series dedicated to specific orbital pathology subtypes. In those reports, incidence rates vary widely. For example, a multi-surgeon series of orbital tumor excisions (n=137) reported zero cases of postoperative vision loss.[7] In contrast, two articles solely addressing orbital cavernous hemangiomas reported vision loss rates of 24% (with 4% NLP) in one series and 7% in the other.[8-9] Another multi-surgeon series which focused exclusively on complications of orbital floor fracture repair found the rate of postoperative vision loss was 2 out of 189 patients (1.06%).[10] Data from a review of 410,189 patients who underwent surgery with general or central neuraxis regional anesthesia found 405 cases of postoperative vision loss (0.1%), but these were not limited to orbitotomy cases and no denominator was provided to calculate an orbitotomy-specific incidence rate.[12] A single-surgeon series of 1593 orbitotomy cases for all indications reported the incidence of blindness to be 0.44% but the study only included patients with preoperative visual acuity of >20/40 and postoperative NLP vision in the analysis, which may underestimate the true risk by excluding patients with baseline visual deficits or those whose vision loss was subtotal.[6]

In our series of 1665 orbital surgeries performed at two academic tertiary referral centers, 14 patients (0.84%) experienced postoperative vision loss beyond the level of 20/400 acuity. The risk of a blinding surgical complication was significantly higher for the subset of patients undergoing surgery for orbital floor fracture repair in the setting of multiple facial fractures (6.45%), bony decompression of the optic canal (15.6%), or an intracranial approach to the orbital roof (18.2%).

Reported risk factors for postoperative vision loss include the surgical approach to orbitotomy. Purgason et al. reported a higher risk of complication from lateral orbitotomy (35%) compared to anterior orbitotomy (3%) in a series of 137 orbital tumor excisions, with complications including diplopia and unfavorable scarring, but there were 0 cases of postoperative vision loss in either subgroup.[7] Our series found a statistically equivalent rate of vision loss after lateral and anterior orbitotomies. Instead, as may logically be expected, the highest-risk approaches were those closest to the orbital apex and optic canal. In a 7-year review of spheno-orbital meningiomas involving the optic canal, Cannon et al. reported major vision loss after surgery in 4 out of 12 (one case with 20/400 acuity, and three NLP).[13] In contrast, there were no instances of postoperative vision loss in a surgical series of 23 patients with meningiomas of the tuberculum sellae near the optic chiasm.[14] An optic chiasm meningioma in our series (patient #11) suffered vision loss to NLP, then subsequently recovered hand motions vision. Aside from direct insult to the optic nerve during surgery (as with patient #7), the likely mechanisms of insult during optic canal surgery are ischemic events due to vessel traction, compression, or surgical sacrifice of vascular structures; thermal injury secondary to cautery to maintain hemostasis; vibratory insults from drills, rongeurs, or chisels during bone removal; and/or elevated postoperative intraorbital pressure due to bleeding or edema.[2,13] Hypotensive anesthesia has also been reported to play a role, and may have been a contributing factor in 2 cases in our series.[15-16]

Vision loss in 4 of 14 patients in our series was due to RBH, all of which occurred in fracture repair cases. One report found that 48% of blindness after orbital fracture repair was attributable to intraorbital hemorrhage.[17] The material used for fracture repair implant does not appear to influence the acute complication rate in several published studies, but it has been suggested that fenestrated implants may reduce the risk of RBH-related compartment syndrome by allowing the hemorrhage a route of egress from the orbit into the adjacent sinuses.[18-20] The timing of reported RBH occurrence ranges from the immediate postoperative period during emergence from anesthesia out to 7 days after surgery, with the majority of events occurring in the first 10 hours.[10,17,21] In our series, RBH timing ranged from 6 hours to 2 days. This suggests that retaining high-risk patients for inpatient observation after surgery may be beneficial, as emergent release of orbital pressure can improve outcomes in cases of orbital compartment syndrome.[21-23]Features that make a patient high risk for RBH include orbitotomy due to orbital fracture, presence of comorbid facial fracture(s), and anticoagulant usage. One of 4 patients who lost vision due to RBH in our series had continued aspirin perioperatively. It is important to advise patients to stop any non-crucial anticoagulants prior to orbital surgery.

In a series of 189 orbital floor fractures, Gosau et al. reported intraorbital hematoma occurred at an incidence rate of 3.2%, and was more likely in heavily traumatized patients with comminuted fractures.[10] Similarly, patients in our series with pan-facial fractures were more likely to suffer visual complications than those with isolated orbital fractures, suggesting trauma severity plays a role in the risk of postoperative blindness. Several factors may contribute to the higher incidence of blindness in heavily-traumatized orbits. First, systematic literature analyses have found that zygomatic fracture—including zygomaticomaxillary complex, LeFort II, and isolated fracture of the zygoma—is strongly correlated with vision loss due to an underlying mechanism of traumatic optic neuropathy or optic nerve compression.[23-25] Perhaps having sustained an injury from a force with sufficient intensity and trajectory to cause zygomatic fracture renders the optic nerve more vulnerable to further surgical insult (e.g. intraoperative retraction, postoperative compression due to edema or RBH), and/or visual decline due to delayed effects of the acute injury.[23] Second, greater trauma leads to more soft tissue edema. In our series, all of the fracture repairs with low vision outcomes were performed within 4 days of injury (average 1.8 days). This raises our concern that traumatic edema compounded by surgical edema may put intraorbital pressures closer to a tipping point at which optic nerve perfusion is compromised.[26-27] Many surgeons advocate repair of facial fractures as soon as possible after injury to optimize aesthetic outcomes, despite the issues that evolving edema may pose.[28] Somewhat reassuringly, Dal Canto reported similar complication rates for orbital fracture repairs performed within 14 days versus 15-29 days after injury.[29] However, the series was fairly small (n=58), had no vision-loss events, and the “early” repair group averaged 9 days after trauma, so the study’s findings may not be able to adequately reflect the role that acute edema plays in postoperative risk.[29] Further research is needed.

Management of new-onset vision loss after orbital surgery involves prompt detection of declining vision, assessment of its etiology by the surgical team and possibly ophthalmology consult, and urgently addressing any potentially-reversible factors.[6,11,19,30] A strategy for prompt detection should include consistent intraoperative pupil monitoring, as well as vision and pupil exams in the post-anesthesia care unit upon awakening. To facilitate intraoperative pupil monitoring for efferent and afferent pupillary defects,~~,~~ both eyes should be included within the sterile field~~,~~ and care should be taken to avoid administering medications that may cause prolonged pupillary dilation. Patients who undergo orbital procedures with a higher risk of vision loss should have serial monitoring at least every 4-6 hours for the first 24-48 hours after surgery. Every surgeon involved in orbital procedures should be trained to reliably and consistently perform the postoperative vision and pupil exam, regardless of subspecialty.[30] For patients who are to be discharged, the patient or family members can be trained to check vision at home and instructed to contact the surgical team with any new concerns. Patient and family education may have expedited detection of the complication in patient #10, who demonstrated intact vision on postoperative exams at 4 and 9 days postoperatively, then noted vision changes at home 21 days after surgery but did not report concerns until his next outpatient appointment on day 27. We also recommend that all patients undergo a comprehensive baseline ophthalmologic evaluation prior to orbital surgery to assess for and document any preexisting ocular pathology requiring specific operative precautions, and to allow comparison with postoperative findings.

In addition to intraoperative and postoperative exams, some surgeons opt to routinely obtain orbital imaging at the conclusion of every case, while others obtain imaging urgently if concerns arise. Of our 14 cases, postoperative imaging identified a potentially-reversible cause of visual decline in half, resulting in 6 patients being returned to the operating room. Another 2 patients were urgently returned to the operating room without imaging, to avoid delay. Visual improvement was seen in 3 of 8 patients after reoperation with dramatic improvement in 2 cases, whereas spontaneous improvement in nonoperatively managed cases was limited to very small gains. Multiple reports in the literature support acute orbital decompression in the setting of postoperative vision loss, either with canthotomy & cantholysis, surgical evacuation of RBH, or urgent decompression of the orbital apex, tailoring the intervention to the situation.[4-6,10,21-23] Patients can regain vision when a reversible insult is promptly addressed. As seen in other case series, the addition of systemic corticosteroids did not significantly impact the final visual acuity outcomes in our cohort. However, the small sample size, broad range of vision loss etiologies, and variable elapsed time between surgery and steroid administration limit the ability to critically analyze the benefit of steroids in this study.

This study has several limitations. Most significantly, the search method based on billing codes is limited by the accuracy of coding. Billing codes also lack more granular data regarding whether treatment occurred in the anterior or posterior orbit, and whether lesions were in the intraconal or extraconal space. The retrospective design led to exclusion of patients whose preoperative visual acuity had not been tested, which may lead to underestimation of the incidence rate of postoperative vision loss. We excluded 2 patients due to unknown preoperative visual acuity. If these 2 patients were assumed to have initial acuity $\geq$20/200 and were thus included in the series, our overall incidence rate of postoperative vision loss would be 0.96%

rather than 0.84%. Furthermore, patients who did not have postoperative vision checked or documented would have been excluded by this retrospective analysis technique. Finally, the study focused on visual acuity outcomes, and not formal visual field data, ocular alignment, or other measures of ophthalmologic dysfunction. Thus, the incidence rate in this study does not encompass all of the vision-related risks of orbital surgery, since pathologies including center-sparing scotomas, large peripheral field defects, cranial neuropathies, diplopia, pupillary abnormalities, and color vision deficiencies, can compromise a patient's function and quality of life without affecting their acuity on eye chart testing.

In conclusion, the incidence of severe loss of visual acuity after orbital surgery at a tertiary referral academic medical center in this 20-year retrospective series is 0.84%. This incidence rate may underestimate the overall risk of visual morbidity, given the inherent limitations detailed above. Patients undergoing craniotomy with orbitotomy, optic canal decompression, or orbital fracture repair in the setting of multiple facial fractures have a substantially higher risk of vision loss. Careful patient selection and preoperative counseling about the risk of postoperative blindness are important aspects of patient preparation for surgery. Close postoperative monitoring and urgent management of potentially-reversible compressive causes of vision loss can improve outcomes.

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## Précis

This large series finds the overall risk of vision loss after orbital surgery is 0.84%, with substantially higher incidence in transcranial orbitotomy (18.2%), optic canal decompression (15.6%), and facial polytrauma orbital fracture repairs (6.45%).

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Purpose: Severe vision loss is a risk of orbital surgery which physicians should prepare for and counsel patients about, but the overall risk rate is unknown. This research was conducted to determine the risk of severe vision loss related to surgery within the orbit.

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Results: A total of 1665 patients underwent orbital surgery during the inclusion period, with 14 patients sustaining severe vision loss ranging from counting fingers at 1 foot to no light perception (overall risk=0.84%). Etiology of vision loss included retrobulbar hemorrhage, malpositioned implant, optic nerve ischemia, or direct optic nerve insult. When stratified by surgical approach, the risk of a blinding surgical complication was significantly higher for patients undergoing orbital floor repair in the setting of multiple facial fractures (subgroup risk=6.45%), bony decompression of the optic canal (subgroup risk=15.6%), or intracranial approach to the orbital roof (subgroup risk=18.2%). Seven of 8 patients with a potentially-reversible etiology of postoperative vision loss were returned to the operating room urgently, and 2 regained substantial vision (20/20 and 20/25). Administration of intravenous corticosteroids had no significant effect on visual acuity outcome.

Conclusions: The overall risk of severe vision loss after orbital surgery is 0.84%. The subgroup risk is higher in patients undergoing facial polytrauma repair, optic canal decompression, or orbital apex surgery from an intracranial approach. Close postoperative monitoring and urgent assessment and management of acute vision loss may improve visual outcome in some cases.

Blindness is a devastating complication following orbital surgery, and one that is discussed with patients in the preoperative informed consent process. The overall incidence of blindness due to orbital surgery, however, has not been thoroughly studied. Published case reports describe vision loss occurring after fracture repairs, partial or total removal of orbital mass lesions, and orbital decompression surgeries.[1-5] A few larger case series have addressed postoperative vision loss rates for specific subpopulations or surgical case types.[6-10] The reported incidence of postoperative blindness varies from 0-24% in the literature, likely due in part to the different risk profiles of the cases addressed by each series.[7-8] The variability in the available literature makes patient selection, operative timing, and preoperative risk counseling difficult.

The goal of this study was to assess the incidence rate and risk factors for postoperative vision loss occurring as a complication of orbital surgery, using data accumulated over an extended period of time from a tertiary academic medical center (AMC). As a surgical setting, several institutional features of the AMC may uniquely influence outcomes: multiple specialties operate within the orbit (Ophthalmology, Otolaryngology, Oral Maxillofacial Surgery, Plastic Surgery, Neurosurgery), multiple surgeons within each specialty, and surgeons-in-training (medical students, residents, fellows) are involved in the peri- and intraoperative management. Given the frequent referral of severe traumas and complex cases to AMCs for surgical intervention in addition to the routine cases, a better understanding of the risk of blindness after orbital surgery in this setting would be of value.

## Methods

Institutional Review Board approval was obtained, and all research was carried out in compliance with HIPAA regulations and in accordance with the Declaration of Helsinki. This was a retrospective chart review study conducted at the University of Washington's two major urban hospitals, one of which is the only Level I trauma center for a 5-state catchment area encompassing Washington, Wyoming, Alaska, Montana and Idaho. Patients seen between January 1994 and December 2014 were eligible for inclusion. The medical center's billing database was searched for Current Procedural Terminology codes for orbitotomy (CPT: 21077, 21172, 21175, 21179, 21180, 21182-21184, 21260-21263, 21267, 21268, 21385-21397, 21390, 21395, 21400, 31075, 31225, 31292, 31293, 61580, 61581, 67400, 67405, 67412-67414, 67420, 67430, 67440, 67445, 67450) and ICD-9 procedure codes (16.09, 16.23, 16.92) to identify patients who had undergone orbital surgery for any indication. These records were subsequently cross-checked with a search of International Classification of Disease-9 codes for vision loss, visual field disturbance, blindness (369.xx), or optic neuropathy (377.xx).

The resulting records were screened with chart review to identify patients who had experienced significant visual loss (defined as final visual acuity of $\leq$ 20/400 in the affected eye) during the acute or subacute postoperative period ($\leq$ 21 days) after orbitotomy. Patients were excluded if preoperative vision was already $\leq$ 20/200 (threshold for legal blindness), or if preoperative vision could not be determined. When a patient's preoperative visual acuity had not been documented in the hospital chart, effort was made to verify baseline visual acuity by other historic means such as records from their private eyecare clinician.

Chart review data collected for each patient included demographics, past medical and ocular history, surgical indication, and preoperative ophthalmic exam findings. Intraoperative details of the procedure were extracted, including surgical findings, implants, relevant medication infusions (corticosteroids, antibiotics, osmotics), anticoagulation status, and any surgical or anesthesia complications. Postoperative data included medications, postoperative eye exam (visual acuity, pupils, extraocular motility), and postoperative imaging results. Details of each patient's vision loss were also collected, including time of onset, signs or symptoms leading to diagnosis, and how it was treated medically and/or surgically. The total postoperative follow-up duration and final visual acuity were noted.

Descriptive statistics were calculated for demographics, incidence rates, and follow-up duration. Statistical analysis for comparison of subgroup incidence rates was performed with MedCalc for Windows, version 15.1 (MedCalc Software, Ostend, Belgium). Postoperative visual acuity outcomes in steroid versus non-steroid groups were compared with a two-sample T-test.

## Results

During the period from January 1994 through December 2014, a total of 1665 patients underwent orbitotomy at the University of Washington Medical Center hospitals. Of these, 198 patients had been assigned ICD-9 diagnostic codes for optic nerve and/or visual dysfunction. These charts were thoroughly reviewed to identify 17 patients with visual acuity which had declined to ≤20/400 after surgery. Two of these patients were excluded due to lack of documented or verifiable preoperative visual acuity: one was a trauma patient who could not undergo subjective preoperative ophthalmologic exam due to intubation at the scene, while the other was an ambulatory surgery patient with no previous record of eye care in any setting. Finally, 1 patient was excluded because fulminant spread of a pre-existing orbital infection was the proximal cause of blindness, rather than the orbital surgery itself. As such, 14 patients were included in the full review, representing an overall incidence rate of 0.84%.

The surgical approaches for 1665 patients who underwent orbitotomy during the 20-year review period are summarized in **Table 1**, along with the postoperative vision loss incidence rate for each approach, which ranged from 0 – 18.2%. Postoperative vision loss occurred in 5 of the 32 orbitotomy cases in which the bony optic canal was surgically opened and/or decompressed (15.6%), with no statistical difference between intracranial approach (n=4 of 22, 18.2%) versus endoscopic trans-sphenoidal approach to the optic canal (n=1 of 10, 10%), ($p$=0.587, 95% CI -0.21 to 0.38). Vision loss was significantly more likely after combined-approach orbital fracture repairs (6.45%, $p$=0.002), or after craniotomy with orbitotomy (18.2%, $p$ <0.0001), compared to the cohort's overall incidence rate. Lateral orbitotomy did not pose a significantly different risk of vision loss compared to anterior orbitotomy ($p$=0.428, 95% CI 0.13 to 24.8).

Among the 14 patients with significant vision loss after orbitotomy, there were 8 females (57%) and 6 males (43%), average age was 47.8 years (range: 30-69 years), with the right eye involved in 9 cases and the left in 5 cases. A summary of each patient's surgical and postoperative course is given in **Table 2**. Postoperative vision checks were conducted in the post-anesthesia care unit when the patient awakened (range: 30 minutes to 2 hours postoperatively) by a physician from the surgical service that performed the procedure. Vision checks were continued by nursing staff every 6-8 hours thereafter if the patient was admitted to the hospital. Postoperative pupil exams were not consistently documented to have occurred in any patient. Patient #10 experienced profound periocular edema related to a periscapular microvascular free flap that was placed on his cheek, which impeded routine vision checks for several days. All of the fracture repairs with low vision outcomes were performed within 4 days of injury (average 1.8 days, range 8 hours to 3.6 days). Average total follow-up after a vision loss event was 20.4 months (range: 1-71 months).

The etiology of vision loss was concluded to be retrobulbar hemorrhage (RBH) in 4 cases (29%), malpositioned implant in 2 (14%), ischemic event in 2 (14%), optic nerve compression due to expansion of a hemostatic packing agent in 1 (7%), seroma in 1 (7%), direct optic nerve injury in 1 (7%), and uncertain in 3 (21%). While there were no reported adverse anesthesia events among the 14 patients with vision loss, review of anesthesia records found 7 patients experienced at least one episode of systolic blood pressure <80mmHg during surgery, 8 patients experienced at least one episode of mean arterial pressure decrease >30% from baseline, and 5 patients experienced both events (#1, 5, 8, 11, 14). The vast majority of such hypotensive episodes lasted 5 minutes or less. No patient experienced systolic blood pressure <80mmHg for greater than 10 minutes. Two patients experienced mean arterial pressure decrease >30% from baseline for 30 minutes, one of whom suffered ischemic vision loss (#14) while the other patient's mechanism of vision loss could not be definitively identified (#11).

Retrobulbar hemorrhage occurred as early as upon awakening or as late as 2 days after surgery. Review of anesthesia records found all RBH patients had been extubated with positive airway pressure, with no supplemental pressure applied to the orbit during awakening. Bucking was documented in one RBH patient (#3) who desaturated due to secretions on the initial extubation attempt and was therefore extubated 3-4 hours later in the intensive care unit without complications. All 4 of the vision-compromising RBH events occurred in patients who had undergone orbital fracture repair with implants. Three of the 4 had required screw-fixated hardware placement. One patient with RBH (#5) had been on daily 81mg aspirin leading up to surgery and appears to have continued it uninterrupted perioperatively, while the others received no anticoagulants before or after surgery.

In 8 patients, the etiology of vision loss involved some form of postoperative compression of the optic nerve (oxycel expansion, RBH, seroma accumulation, malpositioned implants), which could potentially be surgically addressed. Seven of these patients were urgently returned to the operating room, 2 of whom regained substantial vision (patient #1 to 20/20, and #4 to 20/25), and 1 of whom regained enough vision to perceive hand motions (patient #6). The patient who was nonoperatively managed (patient #3) was noted to have low vision at a postoperative bedside check 6 hours after surgery. CT scan showed a modest-sized extraconal orbital hemorrhage along a well-positioned plate. The patient's visual acuity was attributed to sleepiness and thick ophthalmic ointment. He was discharged without intervention, and had no light perception (NLP) at all subsequent outpatient postoperative follow-up exams. Spontaneous improvement was seen in 4 of 6 non-operatively managed cases (patients #7, 11, 12, and 14), but the improvement was limited to small gains to the level of light perception or counting fingers.

Intravenous steroids were given to 9 of the 14 patients, with the first dose administered a mean of 2.5 hours after initial diagnosis of vision loss (range 0.5 hours to 6 hours after diagnosis; 0.5 hours to 6 days postoperatively). There was no significant difference in the visual acuity outcomes for patients who received steroids (median NLP, range NLP to 20/20) versus those who did not (median LP, range NLP to 20/25), *p*=0.765.

## Discussion

Vision loss is the most severe complication directly associated with orbital surgery. As such, careful preoperative counseling is needed regarding that risk.[11] The literature, however, contains limited data on the incidence rate of vision loss after orbital surgery beyond case reports or case series dedicated to specific orbital pathology subtypes. In those reports, incidence rates vary widely. For example, a multi-surgeon series of orbital tumor excisions (n=137) reported zero cases of postoperative vision loss.[7] In contrast, two articles solely addressing orbital cavernous hemangiomas reported vision loss rates of 24% (with 4% NLP) in one series and 7% in the other.[8-9] Another multi-surgeon series which focused exclusively on complications of orbital floor fracture repair found the rate of postoperative vision loss was 2 out of 189 patients (1.06%).[10] Data from a review of 410,189 patients who underwent surgery with general or central neuraxis regional anesthesia found 405 cases of postoperative vision loss (0.1%), but these were not limited to orbitotomy cases and no denominator was provided to calculate an orbitotomy-specific incidence rate.[12] A single-surgeon series of 1593 orbitotomy cases for all indications reported the incidence of blindness to be 0.44% but the study only included patients with preoperative visual acuity of >20/40 and postoperative NLP vision in the analysis, which may underestimate the true risk by excluding patients with baseline visual deficits or those whose vision loss was subtotal.[6]

In our series of 1665 orbital surgeries performed at two academic tertiary referral centers, 14 patients (0.84%) experienced postoperative vision loss beyond the level of 20/400 acuity. The risk of a blinding surgical complication was significantly higher for the subset of patients undergoing surgery for orbital floor fracture repair in the setting of multiple facial fractures (6.45%), bony decompression of the optic canal (15.6%), or an intracranial approach to the orbital roof (18.2%).

Reported risk factors for postoperative vision loss include the surgical approach to orbitotomy. Purgason et al. reported a higher risk of complication from lateral orbitotomy (35%) compared to anterior orbitotomy (3%) in a series of 137 orbital tumor excisions, with complications including diplopia and unfavorable scarring, but there were 0 cases of postoperative vision loss in either subgroup.[7] Our series found a statistically equivalent rate of vision loss after lateral and anterior orbitotomies. Instead, as may logically be expected, the highest-risk approaches were those closest to the orbital apex and optic canal. In a 7-year review of spheno-orbital meningiomas involving the optic canal, Cannon et al. reported major vision loss after surgery in 4 out of 12 (one case with 20/400 acuity, and three NLP).[13] In contrast, there were no instances of postoperative vision loss in a surgical series of 23 patients with meningiomas of the tuberculum sellae near the optic chiasm.[14] An optic chiasm meningioma in our series (patient #11) suffered vision loss to NLP, then subsequently recovered hand motions vision. Aside from direct insult to the optic nerve during surgery (as with patient #7), the likely mechanisms of insult during optic canal surgery are ischemic events due to vessel traction, compression, or surgical sacrifice of vascular structures; thermal injury secondary to cautery to maintain hemostasis; vibratory insults from drills, rongeurs, or chisels during bone removal; and/or elevated postoperative intraorbital pressure due to bleeding or edema.[2,13] Hypotensive anesthesia has also been reported to play a role, and may have been a contributing factor in 2 cases in our series.[15-16]

Vision loss in 4 of 14 patients in our series was due to RBH, all of which occurred in fracture repair cases. One report found that 48% of blindness after orbital fracture repair was attributable to intraorbital hemorrhage.[17] The material used for the fracture repair implant does not appear to influence the acute complication rate in several published studies, but it has been suggested that fenestrated implants may reduce the risk of RBH-related compartment syndrome by allowing the hemorrhage a route of egress from the orbit into the adjacent sinuses.[18-20] The timing of reported RBH occurrence ranges from the immediate postoperative period during emergence from anesthesia out to 7 days after surgery, with the majority of events occurring in the first 10 hours.[10,17,21] In our series, RBH timing ranged from 6 hours to 2 days. This suggests that retaining high-risk patients for inpatient observation after surgery may be beneficial, as emergent release of orbital pressure can improve outcomes in cases of orbital compartment syndrome.[21-23] Features that make a patient high risk for RBH include orbitotomy due to orbital fracture, presence of comorbid facial fracture(s), and anticoagulant usage. One of 4 patients who lost vision due to RBH in our series had continued aspirin perioperatively. It is important to advise patients to stop any non-crucial anticoagulants prior to orbital surgery.

In a series of 189 orbital floor fractures, Gosau et al. reported intraorbital hematoma occurred at an incidence rate of 3.2%, and was more likely in heavily traumatized patients with comminuted fractures.[10] Similarly, patients in our series with pan-facial fractures were more likely to suffer visual complications than those with isolated orbital fractures, suggesting trauma severity plays a role in the risk of postoperative blindness. Several factors may contribute to the higher incidence of blindness in heavily-traumatized orbits. First, systematic literature analyses have found that zygomatic fractures—including zygomaticomaxillary complex, LeFort II, and isolated fracture of the zygoma—are strongly correlated with vision loss due to an underlying mechanism of traumatic optic neuropathy or optic nerve compression.[23-25] Perhaps having sustained an injury from a force with sufficient intensity and trajectory to cause zygomatic fracture renders the optic nerve more vulnerable to further surgical insult (e.g. intraoperative retraction, postoperative compression due to edema or RBH), and/or visual decline due to delayed effects of the acute injury.[23] Second, greater trauma leads to more soft tissue edema. In our series, all of the fracture repairs with low vision outcomes were performed within 4 days of injury (average 1.8 days). This raises our concern that traumatic edema compounded by surgical edema may put intraorbital pressures closer to a tipping point at which optic nerve perfusion is compromised.[26-27] Many surgeons advocate repair of facial fractures as soon as possible after injury to optimize aesthetic outcomes, despite the issues that evolving edema may pose.[28] Somewhat reassuringly, Dal Canto reported similar complication rates for orbital fracture repairs performed within 14 days versus 15-29 days after injury.[29] However, the series was fairly small (n=58), had no vision-loss events, and the "early" repair group averaged 9 days after trauma, so the study's findings may not be able to adequately reflect the role that acute edema plays in postoperative risk.[29] Further research is needed.

Management of new-onset vision loss after orbital surgery involves prompt detection of declining vision, assessment of its etiology by the surgical team and possibly an ophthalmology consult, and urgently addressing any potentially-reversible factors.[6,11,19,30] A strategy forprompt detection should include consistent intraoperative pupil monitoring, as well as vision and pupil exams in the post-anesthesia care unit upon awakening. To facilitate intraoperative pupil monitoring for efferent and afferent pupillary defects, both eyes should be included within the sterile field and care should be taken to avoid administering medications that may cause prolonged pupillary dilation. Patients who undergo orbital procedures with a higher risk of vision loss should have serial monitoring at least every 4-6 hours for the first 24-48 hours after surgery. Every surgeon involved in orbital procedures should be trained to reliably and consistently perform the postoperative vision and pupil exam, regardless of subspecialty.[30] For patients who are to be discharged, the patient or family members can be trained to check vision at home and instructed to contact the surgical team with any new concerns. Patient and family education may have expedited detection of the complication in patient #10, who demonstrated intact vision on postoperative exams at 4 and 9 days postoperatively, then noted vision changes at home 21 days after surgery but did not report concerns until his next outpatient appointment on day 27. We also recommend that all patients undergo a comprehensive baseline ophthalmologic evaluation prior to orbital surgery to assess for and document any preexisting ocular pathology requiring specific operative precautions, and to allow comparison with postoperative findings.

In addition to intraoperative and postoperative exams, some surgeons opt to routinely obtain orbital imaging at the conclusion of every case, while others obtain imaging urgently if concerns arise. Of our 14 cases, postoperative imaging identified a potentially-reversible cause of visual decline in half, resulting in 6 patients being returned to the operating room. Another 2 patients were urgently returned to the operating room without imaging, to avoid delay. Visual improvement was seen in 3 of 8 patients after reoperation with dramatic improvement in 2 cases, whereas spontaneous improvement in nonoperatively managed cases was limited to very small gains. Multiple reports in the literature support acute orbital decompression in the setting of postoperative vision loss, either with canthotomy & cantholysis, surgical evacuation of RBH, or urgent decompression of the orbital apex, tailoring the intervention to the situation.[4-6,10,21-23] Patients can regain vision when a reversible insult is promptly addressed.

As seen in other case series, the addition of systemic corticosteroids did not significantly impact the final visual acuity outcomes in our cohort. However, the small sample size, broad range of vision loss etiologies, and variable elapsed time between surgery and steroid administration limit the ability to critically analyze the benefit of steroids in this study.

This study has several limitations. Most significantly, the search method based on billing codes is limited by the accuracy of coding. Billing codes also lack more granular data regarding whether treatment occurred in the anterior or posterior orbit, and whether lesions were in the intraconal or extraconal space. The retrospective design led to exclusion of patients whose preoperative visual acuity had not been tested, which may lead to underestimation of the incidence rate of postoperative vision loss. We excluded 2 patients due to unknown preoperative visual acuity. If these 2 patients were assumed to have initial acuity $\geq$20/200 and were thus included in the series, our overall incidence rate of postoperative vision loss would be 0.96%

rather than 0.84%. Furthermore, patients who did not have postoperative vision checked or documented would have been excluded by this retrospective analysis technique. Finally, the study focused on visual acuity outcomes, and not formal visual field data, ocular alignment, or other measures of ophthalmologic dysfunction. Thus, the incidence rate in this study does not encompass all of the vision-related risks of orbital surgery, since pathologies including center-sparing scotomas, large peripheral field defects, cranial neuropathies, diplopia, pupillary abnormalities, and color vision deficiencies can compromise a patient's function and quality of life without affecting their acuity on eye chart testing.

In conclusion, the incidence of severe loss of visual acuity after orbital surgery at a tertiary referral academic medical center in this 20-year retrospective series is 0.84%. This incidence rate may underestimate the overall risk of visual morbidity, given the inherent limitations detailed above. Patients undergoing craniotomy with orbitotomy, optic canal decompression, or orbital fracture repair in the setting of multiple facial fractures have a substantially higher risk of vision loss. Careful patient selection and preoperative counseling about the risk of postoperative blindness are important aspects of patient preparation for surgery. Close postoperative monitoring and urgent management of potentially-reversible compressive causes of vision loss can improve outcomes.

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**Table 1.** Surgical approach to orbitotomy, and the associated incidence rate of postoperative vision loss.

| **Surgical Site and Approach** | **CPT and ICD-9 procedural codes** | **Number of patients** | **Vision loss incidence (n, %, *p*-value*)** |
|---|---|---|---|
| Orbital roof, extracranial | 21172, 21175, 21179, 21180 | 15 | 0 |
| Orbital roof and apex, intracranial | 21182, 61580, 61581 | 22 | n=4<br>18.2%<br>(***p*<0.0001**) |
| Medial wall, extracranial | 21260, 21267 | 3 | 0 |
| Medial wall, extra- and intracranial | 21261, 21268 | 28 | 0 |
| Floor fracture, trans-antral | 21385 | 28 | 0 |
| Floor fracture, periorbital | 21390, 21395, 21400, 21386 | 977 | n=5<br>0.51%<br>(*p*=0.27) |
| Floor fracture, combined approach** | 21387 | 31 | n=2<br>6.45%<br>(***p*=0.002**) |
| Medial wall or floor, trans-sinus, non-fracture | 31075, 31225, 31292 | 153 | 0 |
| Orbital apex, endoscopic trans-sinus | 31293 | 10 | n=1<br>10%<br>(***p*=0.001**) |
| Anterior orbitotomy, non-fracture | 16.09, 67400, 67405, 67412, 67413, 67414 | 297 | n=1<br>0.33%<br>(*p*=0.39) |
| Lateral orbitotomy, non-fracture | 16.23, 16.92, 67420, 67430, 67440, 67445, 67450 | 101 | n=1<br>0.99%<br>(*p*=0.62) |

CPT=Current Procedural Terminology, ICD-9=International Classification of Disease Ninth Revision

* *p*-value represents comparison of subgroup incidence rate versus overall whole-group incidence rate. Statistically significant *p*-values are shown in bold.

** All involved repair of multiple facial fractures in addition to orbital floor

**Table 2.** Case overview of patients with vision loss after orbital surgery.

| Case | Age, Sex | Pre VA | Diagnosis | Orbitotomy Approach | Intraoperative Findings/Details (surgery duration) | Post VA (elapsed time since surgery) | Pain? | Interventions Upon Noting Vision Loss | Etiology | Final VA (total follow-up time) |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 47,F | 20/20 | Clinoid meningioma | Frontotemporal craniotomy with orbitotomy, + optic canal decompression (NSGY) | Tumor compressing optic nerve. Oxycel placed in superior posterior orbit to aid hemostasis. (300min) | CF at 1 foot (4hrs),<br>NLP (5hrs) | Yes | IV solumedrol, stat MRI, Ophthalmology consult, return to OR (at 6hrs post-op) where oxycel was removed. | Oxycel expansion + small RBH compressing optic nerve | 20/20 (60mos) |
| 2 | 59,M | 20/25 | Intraconal orbital cavernous hemangioma | Trans-cutaneous lateral orbitotomy (OPRS) | Lateral orbital rim removed and lateral rectus muscle disinserted to access mass. (290min) | NLP (upon awakening) | No | IV solumedrol, stat CT, Ophthalmology exam (showed CRAO), ocular massage. | CRAO | NLP (49mos) |
| 3 | 40,M | 20/30 | Facial fractures (bilateral LeFort I-III, bilateral orbital floor & medial wall) | ORIF via trans-conjunctival & trans-caruncular incisions (PRS) | Porous polyethylene implant to reconstruct medial wall and floor defects. (540min) | CF at 1 foot (6hrs) | No | CT (extraconal hematoma along well-positioned implant), Ophthalmology consult. Discharged home without intervention. | RBH (no anti-coagulant) | NLP (2mos) |
| 4 | 30,F | 20/50 | Orbital floor fracture | ORIF via trans-conjunctival incision (PRS) | Subperiosteal dissection to the orbital apex. Screw-fixated porous polyethylene + titanium implant. (110min) | 20/100 (upon awakening),<br>LP (2days) | Yes | Stat CT (hemorrhage along well-positioned implant). Incision opened at bedside to drain hematoma POD#2. Returned to OR to redrain hematoma POD#3. | Large delayed RBH (no anti-coagulant) | 20/25 (6mos) |
| 5 | 48,F | 20/25 | Orbital floor and lateral wall fractures | ORIF via combined approach: buccal-gingival and trans-conjunctival (PRS) | Screw-fixated porous polyethylene + titanium implant. (150min) | “Intact” (upon awakening),<br>LP (2days) | Yes | IV dexamethasone, stat CT (large RBH), lateral canthotomy/ cantholysis, return to OR (at 2 days post-op) where 10mm hematoma was evacuated and implant was removed. | Large delayed RBH (patient on aspirin) | NLP (1mos) |
| 6 | 41,M | 20/25 | Facial fractures (bilateral LeFort I-II, Left zygoma, cribriform plate, orbital floor and medial wall) | ORIF via trans-conjunctival incision (PRS) | Screw-fixated porous polyethylene + titanium implant. (160min) | NLP (upon awakening) | Yes | IV hydrocortisone, stat CT (hemorrhage along malpositioned implant with entrapped medial rectus), return to OR (at 0.5hrs post-op) where hematoma was drained and implant was repositioned. | Malposition implant + small RBH | HM (3mos) |
| 7 | 59,M | 20/20 | Orbital apex Schwannoma | Frontotemporal craniotomy with orbitotomy, + optic canal (NSGY + OPRS) | Traction suture passed through optic nerve sheath to displace optic nerve and allow access to schwannoma. (330min) | LP (upon awakening),<br>CF (3hrs) | No | IV dexamethasone, MRI | Intra-operative trauma to optic nerve | CF at 3 feet (9mos) |
| 8 | 49,M | 20/25 | Facial fractures (Left ZMC, and inferior orbital rim and floor) | ORIF via combined approach: buccal-gingival and trans-cutaneous eyelid (PRS) | Porous polyethylene to orbital floor, and screw-fixated titanium plates to zygoma and orbital rim. (230min) | NLP (7hrs) | Yes | IV Solumedrol and mannitol, Ophthalmology consult, lateral canthotomy/cantholysis, return to OR (at 10hrs post-op) where incision was opened, implant was removed, and large hematoma was drained. | Large RBH (no anti-coagulant) | NLP (1mos) |

| Case | Age, Sex | PreVA | Diagnosis | Orbitotomy Approach | Intraoperative Findings/Details (surgery duration) | Post VA (elapsed time since surgery) | Pain? | Interventions Upon Noting Vision Loss | Etiology | Final VA (total follow-up time) |
|---|---|---|---|---|---|---|---|---|---|---|
| 9 | 47,F | 20/30 | Facial fractures (bilateral ZMC, NOE, orbital floors and medial walls) | ORIF via trans-cutaneous anterior orbitotomy (PRS) | Porous polyethylene and calvarial bone grafts. (510min) | CF at 1 foot (upon awakening), NLP (10hrs) | No | IV Solumedrol, incision opened at bedside, Ophthalmology consult, urgent CT scan (showed well-positioned implants and no RBH). Return to OR (at 11hrs post-op) where implant was removed. | Uncertain. TON from intraoperative bony manipulation, ischemia from globe retraction? | NLP (48mos) |
| 10 | 46,M | 20/20 | Parry Romberg with enophthalmos | Trans-cutaneous anterior orbitotomy (PRS) | Removal of previously-placed silastic impants, placement of screw-fixated porous polyethylene implant to orbital floor, and periscapular free flap to cheek. | "Normal" (upon awakening, inpatient POD4, and outpatient POD9). Pt noted vision loss POD21, but did not report until POD27. | No | Ophthalmology consult (POD27), CT scan (showed fluid collection along well-positioned orbital floor implant), and return to OR (POD28) where fluid collection was drained and implant was removed. | Serous fluid accumulation | NLP (6mos) |
| 11 | 50,F | 20/30 | Clinoid and suprasellar meningioma with optic chiasm compression | Frontotemporal craniotomy with orbitotomy, + optic canal decompression (NSGY) | Tumor was noted to compress the optic nerve, so canal was opened. Hemostasis was particularly challenging. (440min) | NLP (2days) | No | None | Uncertain. Hypotension, TON from drill, cautery, or surgical manipulation of nerve? | HM (4mos) |
| 12 | 42,F | 20/125 | Thyroid-associated orbitopathy | Trans-cutaneous lateral decompression (OPRS) + Endoscopic medial wall and optic canal decompression (ENT) | Routine procedure with typical anatomy. (240min) | NLP (upon awakening) | No | IV solumedrol, hyperbaric oxygen, CT scan (showed optic nerve thickening and mild intraconal fat stranding). | Uncertain. TON from drill, ischemia from globe retraction? | LP (24mos) |
| 13 | 42,F | 20/30 | Facial fractures (Bilateral zygoma and lateral orbital walls) | ORIF via combined approach: buccal-gingival and trans-cutaneous eyelid (PRS) | Porous polyethylene and bone grafts to orbital floor, and screw-fixated plates to zygoma. (480min) | NLP (6days; when able to report) | No | IV solumedrol, Ophthalmology consult, return to OR (at 6 days post-op) where bone grafts near apex were repositioned more anteriorly. | Posterior bone grafts compressing optic nerve | NLP (71mos) |
| 14 | 69,F | 20/30 | Intracavernous carotid aneurysm extending to ophthalmic art. | Frontotemporal craniotomy with orbitotomy, + optic canal decompression (NSGY) | Intracavernous aneurysm clipped, ophthalmic artery aneurysm clipped, optic canal unroofed. (575min) | NLP (8days) | No | None | Ophthalmic artery ischemia | LP (2mos) |

CF=Able to Count Fingers, CRAO=Central Retinal Artery Occlusion, ENT=Otolaryngology, FinalVA=Visual acuity at time of final follow-up exam, HM=Perception of Hand Motion, LP=Light Perception, NLP=No Light Perception, NOE=Naso-orbital-ethmoidal fracture, NSGY=Neurosurgery, OPRS=Oculoplastic Surgery, ORIF=Open Reduction Internal Fixation fracture repair, POD=Postoperative Day, PreVA=Preoperative Visual Acuity, PostVA=Postoperative Visual Acuity, PRS=Plastic/Reconstructive Surgery, RBH=Retrobulbar hemorrhage, TON=Traumatic optic neuropathy, ZMC=Zygomaticomaxillary Complex fracture