VISUAL LOSS AFTER ANESTHESIA DIFFERENT CAUSES: DIFFERENT SOLUTIONS

- A Review -

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Historical Introduction

It has long been recognized that visual damage may follow the administration of anesthesia. In 1937, Guedel noted "We still see too much conjunctivitis and traumatic keratitis following anesthesia"[1]. The injuries at that time were attributed to open drop ether through a gauze covered mask in hard to handle subjects who half opened their eyes during light anesthesia (the patient was deemed in part responsible). A towel was customarily placed over the eyes and often became saturated quickly. Given that about 90% of anesthetics were produced by ether, mostly by an open or simple mask system throughout the first half of the 20th century, it is not surprising that serious corneal injuries were common but considered a small price to pay for a pain free surgical experience.

Administration of gas through an oral tube was considered and rejected by John Snow in 1858 because he claimed that the tube fell out as the patient lost consciousness[2]. Also, he considered his inhalation device via a mask with pliable sheet lead edges superior. The first use of tracheal tubes, a flexible gum catheter, was for resuscitation[3]. The Scottish neurosurgeon, Sir William Macewen, described several cases in 1880 in which he admitted patients several days preoperatively to practice placement of an endotracheal tube before induction of chloroform anesthesia[4]. But endotracheal anesthesia did not become general practice until after the development of the laryngoscope by Magill and others and the detachable blade by MacIntosh in 1941[5,6]. The incidence of eye injuries decreased dramatically. Over the past two decades not only have short acting drugs been developed but the need to monitor oxygenation has been emphasized. Once more corneal injuries are prone to occur as patients, still partially anesthetized rub their eyes with fingers attached to a pulse oximeter.

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Recent Causes of Postoperative Visual Loss

Over the past 2 decades, cases of total blindness or severe visual impairment have been increasingly described after anesthesia.

Review of records of 60,965 patients in 1996 revealed an incidence of postoperative visual difficulties 0.056% (34 patients)[7]. Corneal abrasion occurred in 21 cases, a figure which many practicing anesthesiologists today might consider lower than reality. Other injuries included conjunctivitis (n = 7), chemical injury (n = 1), direct trauma (eyelid hematoma, n = 1) blurred vision persisting longer than 3 days (n = 3) and blindness (n = 1). A specific cause for the injury was determined in only 21% of cases. But other older studies, many in the nonanesthesiology literature have documented incidences of postoperative visual impairment ranging from 0.1% to 1%[8-10]. Several of these cases concerned open heart surgery and the visual loss may have been related to embolic causes. One review of 37 cases which also included a survey of the Scoliosis Research Society concluded that the incidence of significant visual loss after spine surgery is on the order of one case per 100 spine surgeons per year[11] Yet other retrospective reviews indicated one case of blindness in 56,000 surgical procedures at one university hospital, 4 cases out of 14,000 and 6 cases over a 10- year period at another[12,13,14]. Studies of patients with significant deterioration of sight have pointed to ischemic optic neuropathy, usually of the posterior part of the nerve.

Has the Incidence of Postoperative Visual Loss Increased Recently?

Although visual loss due to ischemic optic neuropathy has been recognized as a complication of spinal surgery since the 1950's, it was considered a rare event and received little attention[15]. However, during the1990's several cases were reported1[1,16-19]. Even more cases were reported over the next decade[20-23]. Many reasons have been suggested for the increase. It may be that with the improvement in safety of anesthesia, rare problems assume greater visibility. Or it may be that with more aggressive surgery, procedures are longer and more complex, often combining hypotensive techniques and performed on older patients with more co morbidities who are at greater risk for postoperative problems. Certainly there has been a remarkable expansion of available instrumentation offering alternative therapies for patients with chronic back pain who have not responded to other treatments. With the risk, small as it is, of disease transmission with blood transfusion, patients and their doctors are often hesitant to use banked blood, replacing lost blood with crystalloids. In the past, ophthalmologists have opined that the occurrence of visual defects after anesthesia is common, related to pressure and thus unremarkable. Also anesthesiologists have been unwilling to come forward with descriptions of adverse outcomes for fear of litigation. Data collection to define risk factors has been slow and difficult although recent reviews have identified common elements and defined better strategies.

Pathophysiology

Even from the earliest reports it was clear that postoperative visual loss (POVL) was not a single entity. In a few cases, cause could be identified, as, for example, when foreign bodies entered the eyes causing corneal abrasions or pressure directly on the orbits had been long and excessive[24]. Also, some cases resolved, and others did not. Although the final denominator is ischemia of some part of the optic nerve, why blindness should result in some patients and not others under apparently similar conditions requires identification and dissection of many variables. Possible explanations for these differences may be explained by the different blood supply to the various parts of the optic nerve[25]. The anterior portion of the optic nerve including the optic disc and the part of the nerve within the scleral canal is supplied mainly by the short posterior ciliary arteries by way of the choriocapillaris around the disc and by branches that form an anastomotic ring around the nerve. Additional supply from collateral vessels of the ophthalmic artery and the central retinal artery is also common. Near the lamina cribosa, the optic nerve head is supplied by a rich capillary network derived from the circle of Zinn-Halle. The posterior part of the optic nerve has a peripheral vascular blood supply only from pial vessels from branches of the ophthalmic artery. Although the central retinal artery may supply branches to the central fibers, the blood supply to this part of the nerve is significantly less than to the

anterior part. Blood supply to the intracanalicular part of the nerve is from the pial network of branches of the internal carotid and anterior cerebral arteries. Finally, the optic chiasm is supplied by branches from the internal carotid and anterior cerebral arteries. The posterior and middle cerebral arteries provide blood supply to the retrogeniculate optic radiation and the occipital cortex.

The blood supply to the retina is from terminal branches of the central retinal artery to the inner layers and the choriocapillaris to the outer layers. Both circulations must be intact to maintain retinal activity. The fovea is supplied solely by the choriocapillaris. The short posterior ciliary arteries and central retinal artery are end-arteries. Each short posterior ciliary artery supplies a distinct area and watershed zones may form at the boundaries, creating precarious areas of blood supply.

Types of ischemic visual loss

Injury due to an ischemic event in the visual pathway that results in postoperative visual loss may be due to several causes of decreased oxygen delivery.

Classification is based on the site of injury.

1. Ischemic injury to the optic nerve is divided to anterior ischemic optic neuropathy (AION) and posterior ischemic optic neuropathy (PION). In AION, optic disc swelling may be seen and in PION, fundal examination is normal initially but disc pallor becomes apparent.
2. Cortical blindness is caused by visual loss associated with the optic radiation and occipital cortex. It results from emboli, shock, or cardiac arrest and is caused by damage to the occipital cortex. Blindness, normal fundal examination and retention of light response are seen
3. Central retinal arterial occlusion occurs after embolic or thrombotic events or is associated with excessive extraocular pressure. A characteristic "cherry red" spot is seen on the retina. Central retinal venous occlusion is diagnosed by finding retinal hemorrhages in all 4 quadrants, cotton wool spots and dilated tortuous retinal veins

Anterior ischemic optic neuropathy is due to infarction at watershed areas between the zones of distribution of the short posterior ciliary arteries. Asymptomatic optic disc swelling may be an early sign and may resolve spontaneously or result in irreversible blindness. It has been reported after various causes of systemic hypotension, including cardiac arrest[26], hemodialysis[27], and even during sleep[28]. However, intraoperative hypotension appears to be rarely associated with AION[25]. Risk factors associated with AION include older age, peripheral vascular disease, hypertension, diabetes, anemia and congenitally small discs[29,30]. Direct orbital pressure may or may not be a risk factor. It is believed that the combination of a morphologically abnormal optic head and one or more vascular risk factors is sufficient to induce AION[31]. Retinal examination shows optic disc edema, often accompanied by splinter hemorrhages at the disc margins. The degree of disc edema may not correlate with the amount of visual loss. Visual evoked potentials are decreased. The disc edema usually resolves in about 2 months and is replaced with disc atrophy. Partial and complete recovery of vision may occur although the prognosis is usually not good, especially if it is a progressive form associated with vascular disease. Therapy has included retrobulbar steroid injections, antiplatelet therapy, anticoagulation, norepinephrine infusion, carbonic anhydrase inhibition and blood replacement[25]. No consistent successes have been reported with any treatment regimens.

Posterior ischemic optic neuropathy presents as acute loss of vision and is thought to be due to decreased oxygen delivery to the posterior part of the optic nerve. Although AION and PION are due to ischemia of the optic nerve, they represent two subtly different pathophysiologic entities, a difference related mainly to different blood supplies. The blood supply to the posterior part of the nerve is from small vessels that may easily be compressed by fluid buildup in that area. Also, structural abnormalities of the optic nerve head have not been identified as a risk factor for PION. Rather than the large series that are described in the literature for AION, papers about PION are more likely to be anecdotal and describe case reports, for example after a specific surgical procedure such as spinal and prostatic robotic surgery and radical neck dissection, migraine, and after multiple trauma with large volume

fluid resuscitation[30-34]. Bilateral PION has also been reported anecdotally after the use of sildenafil which influences the nitric oxide-cyclic guanosine monophosphate pathways which are involved in the pathogenesis of several neurologic disorders[36,37].

Cortical blindness may follow intracranial surgery or be related to stroke. Central retinal artery inclusion as a cause of blindness may be related to direct pressure on the eyes and anemia and hypotension. PION is not considered to be related to orbital pressure.

American Society of Anesthesiologists: Registry

After identification of postoperative blindness as a major concern by practicing anesthesiologists, the American Society of Anesthesiologists (ASA) Committee on Professional Liability established the ASA Postoperative Visual Loss (POVL) Registry to collect detailed information on these cases in 1999[38]. The goal was to identify intraoperative risk factors and patient characteristics by analyses of cases. The Committee posted a detailed case report form on the ASA web site with instructions for anonymous case submission (www.asaclosedclaims.org). Completion of the reports required access to preoperative, intraoperative (including all anesthesia charts) and postoperative (PACU and ophthalmologic examination) records. Over 100 cases were collected (which was the initial goal) and reported on in 2006[39]. Additionally in 2005, the ASA appointed a task force of 12 members to develop a practice advisory for perioperative visual loss associated with spine surgery[40].

Identification of Risk Factors

Early on it was clear that POVL was not a single entity[41]. In a few cases, cause could be identified, as, for example, when foreign bodies entered the eyes causing corneal abrasions or pressure directly on the orbits had resulted in central retinal arterial or venous thrombosis. Also, some cases resolved completely, and others did not, probably due to the area of the nerve damaged and its blood supply.

Identification of risk factors for POVL has been the subject of several reviews. Prone and lateral position and long surgery are readily associated.[39,42]. Time honored speculation, mainly by surgeons and ophthalmologists has identified pressure on the eyes and, intraoperative hypotensive hypovolemia, combined with perioperative anemia as obvious risk factors. Review of cases from the ASA registry indicates that direct eye pressure is not a factor for PION but even mild hypotension sustained for hours may be partially causative, especially in a patient previously documented as hypertensive[39]. To decrease the need for replacement with banked blood, crystalloid infusions are often given and the Hct decreased, resulting in hemodilution. Postoperative facial swelling is usually marked. A recent study indicates that central hypervolemia with hemodilution impairs cerebral autoregulation in humans, making blood flow pressure dependent. Increased ocular venous pressure may also be a factor[43]. A porcine animal model showed that while compensatory mechanisms for cerebral blood flow was maintained during specified conditions of hypotension and anemia, optic nerve compensatory mechanisms failed and were unable to preserve oxygen delivery[44]. Autoregulation may be further impaired by inhalation anesthetics. Avoidance of Trendelenberg position and minimization of intravenous fluids are appropriate steps to avoid facial edema and tissue pressure increase. Intraocular pressure is increased in the prone position and thus ocular perfusion pressure decreases[45]. Urine output is often markedly decreased or even absent. A table frame such as the Jackson frame allows better accommodation of an increased girth and may improve renal flow by maintaining renal perfusion pressure. Also, if intraabdominal pressure is maintained close to normal, pressure is not increased in epidural veins and thus bleeding is lessened. Anatomical variation in the blood supply to the optic nerve is undetectable to the anesthesiologist but might explain why only a few patients sustain POVL. Or it may be that the optic head variation, combined with major shifts in fluid balance and in blood pressure may be responsible. As such, POVL would be confined to the anterior part of the optic nerve, rather than the posterior part.

The POVL Registry identified some common findings[39].

1. The cause of postoperative blindness appeared to be ischemic optic neuropathy in about 90% of cases. In only 6% (n = 3) was central retinal

artery occlusion diagnosed (i.e. POVL is rarely due to pressure on the eyes).

2. The prone position places patients at risk. The incidence dramatically increases for prone times between 5 and 9 hours. But as the Registry does not contain denominator data of all cases in the prone position, definitive conclusions regarding risk and duration of the prone position cannot be made.
3. Younger age does not appear to be protective as many patients are under 60 years. One case report diagnosed PION, initially thought to be functional visual loss, in a 33 year old 2 months after spine surgery[46] The occurrence in younger, healthier individuals suggests that intraoperative physiologic variables such as edema formation and venous congestion in the prone position as well as "normal" physiologic variation in ocular hemodynamics may be important etiologic factors.
4. Measurement of intraocular pressure (IOP) over time in the prone position indicates about 100% increase over 6 hours and uniform increases from baseline of 20+/- 7 mmHg to 29 +/- 9 mmHg in the initial prone position to 41 +/- 10 mmHg at the end of surgery. Given this increase in IOP, decreased mean arterial blood pressure could markedly reduce ocular perfusion pressure. Other authors have cited high blood sugar levels postoperatively and suggest that tight control during the perioperative period is essential to increase neuronal survival.
5. In all cases there was considerable blood loss and replacement with large volumes of crystalloid solutions.

However, several factors are not considered by the Registry, namely rate of blood loss and time to replacement, types and amount of fluid used, urine output, and levels of glucose and blood pressure control, especially in patients identified as hypertensive but now well controlled by combinations of beta and calcium channel blockers and angiotensin converting enzyme inhibitors. Hypertension is a disease state and recovery to blood pressure levels within a normal range does not equate to cure of all attributed pathologies. The same may be said of diabetes. Development of a compartment syndrome within the eye has been described associated with facial edema and PION and confirmed by magnetic resonance imaging (MRI)[47,48]. Dilated superior ophthalmic veins have also been seen on MRI, suggesting that an increase in orbital venous pressure during surgery contributes to the development of PION[49]. The concept of perioperative fluid replacement in general has been challenged[50]. Concern has been raised that fluid resuscitation may be over generous and even contribute to complications such as pulmonary edema, myocardial dysfunction, bacterial translocation and development of sepsis and multiorgan failure[51,52]. Patients who developed postoperative blindness after lumbar surgery also had very large positive fluid balance.

It appears that certain patients are at risk of developing POVL. Although presence of any single factor listed below may not place the patient at increased risk, the combination of several circumstances should be considered as potentially problematic. Patients should be informed that low as the incidence is, there is a risk of PION following complicated back surgery[13,40]. Identified factors are as follows:

1. *Repeat spinal surgery and the prone position*. The patient may be a chronic pain patient who has had many previous surgeries and now presents for a potentially long procedure, which requires extensive instrumentation. Considerable blood loss may be anticipated and the patient may have predonated blood, thus reducing his/her hematocrit preoperatively.
2. *Body habitus and social conditions*. Disc disease is often associated with smoking, obesity and sedentary life style. Obesity was identified in many of the registry patients. Hypoxia and/ or bronchospastic disease may occur during anesthesia.
3. *Hypotension*. Hypertensive patients are often unstable intraoperatively and given the decrease of intraocular perfusion pressure associated with the prone position, ocular perfusion pressure

may be seriously decreased if any period of hypotension occurs or if it is prolonged.

4. *Hyperglycemia.* Diabetes and increased perioperative glucose levels have been associated with poor neurologic outcome as hypoxic or ischemic tissue is unable to metabolize sugar through normal pathways and the size of infracted areas is increased. Patients undergoing spinal surgery are often treated prophylactically with steroids to decrease edema formation, which further increases blood glucose levels. Stress also contributes to hyperglycemia. Recent studies have emphasized the need for tighter perioperative glycemic control 80-155 mg/dl)[53].
5. *Anemia, blood loss and hemodilution.* Hemodilution and predonation therapy may result in anemia. Earlier guidelines for care of the young trauma victim suggested that blood could be replaced with crystalloid in the amount of 1 to 3ml. However, patients for prolonged spinal surgery are usually not healthy. Crystalloids stay in the circulation less than an hour before leaking to other tissues. In the prone position, especially if there is a degree of Trendelenberg tilt, fluid will gravitate to dependent soft tissues in the face and around the eyes causing edema and increasing venous pressure. Excess fluid also fills the intestinal wall, further increasing intraabdominal pressure which decreases renal output (which may in turn be treated by increased fluid boluses) and increases bleeding from epidural venous plexuses. Average blood loss in complicated spine surgery is 4,000 ml. Current guidelines advocate replacement of blood as necessary to maintain adequate oxygen delivery. Excessive crystalloid replacement may contribute to POVL[54] and cause the development of a compartment syndrome within the eye. Preoperative volume loading may not be necessary in most cases. The classic third space probably does not exist[50]. Demand related regimens should be followed to improve patient outcome. Perioperative fluid shifting must be minimized. Fluid balance should be maintained. The tetrastarches, hydroxyethyl starch 130/0.4, recently approved in the United States, have been shown to represent a substantial advance in colloid therapy, offering good volume replacement with a low risk of side effects[55]. Particularly convincing of the superiority of colloids for perioperative fluid replacement is the ability of hydroxyethyl starch to improve tissue oxygen tension (pti02) significantly more than crystalloids indicating improved microperfusion and less endothelial swelling[56]. But given the enormous variability of the patient, his condition, and the perioperative parameters a means to assess what exactly meets appropriate fluid replacement is still lacking. The intravascular space is not static. The esophageal Doppler, supplying continuous real time objective data, may well emerge as the monitor of preload conditions and help us manage cardiac contractility and the effect of afterload impedance on left ventricular performance[57,58].
6. *Long surgery.* Average length of surgery exceeded 5 hours in the registry study.

Treatment

Although several therapies have been tried there does not appear to be a reliable curative therapy. Prompt evaluation, diagnosis and documentation are important. Some improvement may occur with restoration of Hb to preoperative levels, maintenance of blood pressure, head up position and diuresis of excess fluid. Steroids, hyperbaric oxygenation and surgery have not shown consistent results[30,34,47]. A recent issue of the Newsletter of the Anesthesia Patient Safety Foundation (vol 23; 1: 1-20), considers a review of informed consent for spine surgery and attempts made by surgeons to increase awareness of the complication, still noting that there is no cure.

Management

Given that POVL cannot be reversed, then all attempts at avoiding the complication should be made.

Currently the pathogenesis of PION remains unclear. Although one author has stated that preventive and therapeutic measures remain elusive[59], evidence based medicine point to multiple areas where the risks can be reduced significantly. We may never identify the trigger in a specific patient and indeed it probably varies between patients. Given the multifactorial issues involved, it is unlikely that there is a single cause. Current recommendations might include:

1. Preanesthetic assessment should investigate any history of vascular disease or diabetes and ensure that the patient is in optimal condition. A history of previous visual problems should be sought and documented.
2. The patient's body must be well protected. There should be no abdominal compression and the eyes should be padded and observed frequently. One report indicates that use of some goggles may not prevent against excessive pressure and retinal occlusion[60]. Notations must be made of checks at regular intervals on the record. Use of a Jackson frame may be indicated.
3. The head must be positioned at or above the level of the heart. If a Wilson frame is used, flexing the spine frame, allows the legs to be lowered, thus improving gravitational blood flow away from the operative site.
4. Invasive monitoring of blood pressure allows accurate assessment of blood pressure and blood sugar levels. Elevated blood sugar levels (>150 mg/dl) should be treated.
5. Blood pressure should be maintained as close as possible at normal levels for the patient.
6. Fluid balance should be maintained. Measurement of fluid input and output must be maintained (placement of a Foley catheter is necessary). Hemodilution should be minimized.
7. Colloid should be included in fluid replacement.
8. Urinary output should be maintained consistently, using small doses diuretics if necessary rather than resorting to large fluid challenges, especially in otherwise healthy individuals.
9. Blood replacement should be timely. Frequently anesthesiologist delay replacing blood until the end of the case in an otherwise stable patient, reasoning that it is preferable that the patient lose less of the high Hb replacement blood. Especially if the patient has predonated and may be maintained on beta adrenergic blocking drugs, and recognizing that replacement will be necessary, blood should be replaced early. Although base line and periodic Hct levels are customarily measured, intraoperatively they are often inaccurate. Also, red blood cell transfusions should not be dictated by a single hemoglobin "trigger" as these values are often erroneous. Also, shortly after predonation, the Hb level may be abnormally low.
10. Operative time should be kept as short as possible. Staging a procedure is an alternative.
11. Accurate charting and recording of as much intraoperative information as possible is essential. The use of electronic record keeping is advised. Some anesthesiologists have argued that swings in blood pressure are common intraoperatively and although there are usually no postoperative consequences, these aberrations might only provide fodder for a plaintiff's lawyer. A defense expert can more easily persuade a jury that care and attention was given to the patient if much legible information is available. However, should the blood pressure be recorded as severely depressed for hours and no action was taken, then the defending anesthesiologist may well experience difficulties in the face of an adverse outcome.
12. Follow up of the patient through the Postoperative Care Unit with documentation is important. POVL may not be realized for several hours after emergence from anesthesia, especially if the eyes are swollen shut or if the patient does not have access to his glasses or his trachea has remained intubated Attempts to assess vision should be made and recorded

as soon as possible. Also, if facial swelling is apparent, the patient should be placed in reverse Trendelenberg position, diuretics given to increase urinary output and promote fluid shifts from the tissues, blood replaced to restore Hb to preoperative levels, normoglycemia assured and hemodynamic and respiratory stability maintained. Appropriate consults should be obtained.

Conclusion

Over time it is clear that there is no single factor for postoperative visual loss. It fact, more than likely it is an accumulation of events and determined in part by techniques and surgeries that are in fashion. However, if all associated and/or implicated factors are considered and awarded an appropriate value, then techniques can be altered in an overall and sensible fashion to minimize the effects of these perturbations. Although the numbers in the Registry are low, 80-90% occurrence with a p value of <.001 is indicative of a risk or causative factor. We may not know which straw breaks the camel's back and results in POVL but we have a better understanding of the components of that burden and how we may be able to lessen the risks perioperatively.

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