Intubation
- Trismus resolves with muscle relaxant.
- If cardiac involvement, avoid agents that depress cardiac contractility.

Maintenance
- Choose drugs based on hemodynamic status.
- If hepatosplenomegaly or liver disease is present, avoid muscle relaxants dependent on hepatic metabolism.
- Regional anesthetics have been used and may be beneficial in minimizing airway manipulation and aiding in postoperative pain control. Bleeding dyscrasias must be ruled out prior to having a regional anesthetic.

Emergence/Extubation
- Fully awake and supine pts are ideal.
- Check for cuff leak if airway felt "tight" on intubation.
- Potential for prolonged ventilation, especially chronic aspirators.
- Rapid emergence agents (propofol, remifentanil, desflurane) will help facilitate process especially if neurologic component.
- Avoid hypoxia and hypercarbia.

Regional Anesthesia
- Thrombocytopenia and/or factor deficiencies may preclude regional anesthesia.

Adjuvants
- Dexmedetomidine 0.5 mcg/kg can be administered for postop delirium or agitation, especially in children.
- IV acetaminophen 1 g (adults) for pain control if no hepatic involvement; children may receive up to 15 mg/kg.
- H_2 blocker or PPI IV if symptomatic GERD preop.

Anticipated Problems/Concerns
- Airway difficulty during induction and intubation, especially if Hx of dysphagia or upper airway obstruction.
- If present preop, aspiration risk may still continue postop; consider leaving an NG or OG tube.
- Postop airway obstruction due to swelling of lymphoid tissue in head and neck area.
- Postop respiratory failure.
- Postop muscle spasms.
- Postop bleeding, especially if known bleeding dyscrasia preop.

Glaucoma, Closed-Angle

Steven Gayer

Risk
- According to the WHO, glaucoma is the second most common cause of blindness worldwide.
- Risk factors include hyperopia (far-sightedness), age >60, female gender, and family Hx.

Perioperative Risks
- Postop vision loss
- Inducing acute ACG

Worry About
- Causing sustained, marked elevations in IOP
- A chronic, narrowed angle becoming acutely closed periop

Overview
- There are a number of variants of glaucoma, and terminology can be confusing. These include acquired versus congenital, high IOP versus normal pressure, acute versus chronic, and open angle versus closed angle.
- ACG is categorized as either acute or chronic. Acute ACG is an urgent condition; chronic ACG is far more common and often asymptomatic.

Etiology
- ACG occurs when the distance at the outer periphery of the globe between the iris and cornea diminishes. Some individuals are born with narrowed angles and as they age and the lens thickens, further compromising the space.
- In predisposed individuals, chronic narrow-angle glaucoma may acutely progress to full-angle closure. Acute-angle closure occurs when the iris moves into direct contact with the cornea, physically blocking the natural egress of aqueous fluid.

Usual Treatment
- Acute ACG:
  - Administer topical beta-blocker, α2-agonist, pilocarpine 2% or 4% (pilocarpine is effective in inducing miosis only when iris ischemia is relieved, i.e., when IOP falls to <50 mm Hg).
  - Administer IV/oral acetazolamide 5–10 mg/kg (alternatives include hyperosmotic agents, e.g., IV 20% mannitol 1–2 g/kg, oral 50% glycerol 1–1.5 g/kg [contraindicated in diabetics], oral isosorbide 1.5–2.0 g/kg).
  - Topical steroids.
  - Place pts in the supine position (to allow lens-iris diaphragm to move posteriorly).
  - Analgesia and antiemetics.
  - Laser iridotomy.
- Chronic ACG:
  - Reduce IOP with prostaglandins (latanoprost, bimatoprost, travoprost).
  - Other surgical options include trabeculectomy, gonioplasty, or lens extraction.
  - Trabeculectomy, the gold standard procedure, involves creating a small hole in the sclera to allow freer drainage of aqueous humor.
  - Drainage device implants involve insertion of a tube shunt into the anterior chamber.
  - Cataract surgery may relieve a narrowed drainage angle.

Assessment Points

System	Effect	Hx	PE	Test
HEENT	Acute ACG	Sudden unilateral painful eye Blurred vision Photophobia Colored halos around lights Headache N/V	Ocular injection Hazy cornea Mid-dilated pupil	Penlight Gonioscope Slit-lamp Ultrasound biomicroscopy
	Subacute ACG	Headaches (often mistaken for migraine) or asymptomatic		
	Chronic ACG	Generally asymptomatic		

Key References: Gayer S: Prone to blindness: answers to postoperative visual loss, *Anesth Analg* 112(1):11–12, 2011; Gayer S, Gedde SJ: Intraoperative management of increased intraocular pressure in a patient with glaucoma undergoing robotic prostatectomy in Trendelenburg position, *Anesth Analg Case Rep* 6(2):19–21, 2015.

Perioperative Implications

Preoperative Preparation
- Consider preop consultation with pt's ophthalmologist if planned procedure involves prolonged steep Trendelenburg or prone position.
- Avoid mydriasis, either due to stress, dim lighting, or drugs (particularly topical sympatholytic or parasympathomimetic agents).
- Consider checking lytes if pt is on a diuretic.
- Preop antisialagogues or scopolamine are microfractionally absorbed into the globe and thus are considered generally safe to administer parenterally. Antimuscarinic ophthalmic drops (atropine, scopolamine) can induce acute ACG.
- Glaucoma surgery is generally considered to be low risk for sight-threatening bleeding. The consensus of studies exploring this controversial issue suggests that surgery can be safely performed under regional anesthesia without the need to discontinue antithrombotic agents.
- Phospholine iodide (echothiophate) should be discontinued 4 to 6 wk prior to surgery. Systemic absorption can inhibit plasma cholinesterase, causing prolonged muscle paralysis after succinylcholine, as well as inhibit metabolism of ester local anesthetics, predisposing a pt to toxicity.
- Regional anesthesia:
  - Needle-based block: Intraconal (retrobulbar) or extraconal (peribulbar)
  - Cannula-based block: Sub-Tenon's

- Subconjunctival and/or intracameral injection and/or topical anesthetics

Induction

- Anesthetic goals center around minimizing interventions that may increase IOP or cause further damage to the optic nerve. Succinylcholine increases IOP by 9 mm Hg for 5–10 min; nonetheless it is acceptable. Direct laryngoscopy and endotracheal intubation causes a similar or greater increase in IOP. Bucking, coughing, and vomiting increase IOP by 30–40 and should be avoided. A supraglottic airway may be preferred.

Maintenance

- Avoid IOP elevating maneuvers, including constriction around the pt's neck, prolonged Trendelenburg or prone position, and hypercapnia, which may cause choroidal vascular congestion.
- Consider prophylactic administration of acetazolamide and/or mannitol for select pts—for example, the chronic untreated ACG pt for robotic assisted laparoscopic prostatectomy who will undergo prolonged steep Trendelenburg position coupled with potential elevated CO_2, or a similar pt having prolonged prone position spine surgery.

Extubation

- Carefully avoid IOP increasing events such as coughing, bucking, retching, and tight lid squeezing. Elevate head of bed.

Anticipated Problems/Concerns

- Acute ACG is an urgent vision-threatening condition. The challenge for the anesthesiologist is to distinguish whether a painful eye following nonophthalmic surgery is due to a corneal abrasion or acute ACG. Consult an ophthalmologist if the clinical scenario resembles acute ACG.
- Avoid use of ocular decompression devices such as the Honan balloon with regional anesthesia.
- Potentially life-threatening side effects: Systemic absorption of topical beta-blockers (especially Timolol) may exacerbate asthma, produce bradycardia or heart block, and induce CHF. Acetazolamide can cause diuresis and produce electrolyte abnormalities with chronic use. Mannitol can result in osmotic diuresis, CHF, pulmonary edema, and full bladder discomfort. For phospholine iodide (echothiophate), see the previous section.
- Controversial: Postop visual field "wipe-out" or worsening of vision. There is no obvious etiology; however, numerous surgical, anesthetic, and postop causes have been postulated. Theoretical anesthesia mechanisms include:
  - Local anesthetic mass effect: Pressure from local anesthetic, bleeding, or placement of a compression device
  - Direct trauma to the optic nerve
  - Hypoperfusion of the optic nerve head: Hypotension during general anesthesia and vasoconstrictors in local anesthetic

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Glaucoma, Open-Angle

James W. Ibinson | Laura H. Ferguson

Risk

- Open-angle glaucoma is the leading cause of blindness among African Americans and the second leading cause overall in USA.
- African American race, advanced age, elevated IOP, myopia, low diastolic perfusion pressures, and family Hx of open-angle glaucoma increase the risk for primary open-angle glaucoma.
- Incidence in US: Estimates suggest over 2.25 million Americans over age 40 have open-angle glaucoma.

Perioperative Risks

- Vision loss secondary to optic nerve damage from pressure or ischemia

Worry About

- Interactions between ophthalmologic drugs and anesthetics
- Increases in IOP
- Periop derangements in electrolytes secondary to ophthalmologic drugs

Overview

- Glaucoma is a degenerative optic neuropathy characterized by optic-nerve cupping that results in progressive vision loss and possibly blindness if not treated. Treatment does not reverse the blindness.
- Elevated IOP is often found in glaucoma but is not required for the diagnosis. Nonetheless, treatment for all forms is aimed at maintaining a low-normal IOP.
- Onset is gradual, bilateral, and often unnoticed. While juvenile forms exist, it is much more common in those >40 y.

Etiology

- Likely caused by sclerosis of the trabecular meshwork near the canal of Schlemm, which decreases aqueous humor outflow and elevates IOP.
- Normal pressure, open-angle glaucoma is thought to be caused by insufficient blood flow leading to optic nerve damage, but treatment is the same as with primary open-angle glaucoma.

Usual Treatment

- Treatment goal is to maintain a low-normal IOP. Treatment is most successful if disease is detected early.
- Medical treatment includes topical timolol, betaxolol, epinephrine, echothiophate, or dipivefrin and oral acetazolamide.
- Surgical treatment includes laser trabeculoplasty, trabeculectomy, Baerveldt and Ahmed device implantation, and cycloablation.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Optic nerve damage, increased IOP	Visual changes, family Hx of glaucoma, myopia	Decreased visual acuity, increased optic cup-to-disk ratio, visual field losses	Slit lamp exam Tonometry Visual fields Visual acuity
CV	Excessive beta blockade	Fatigue, syncope or near-syncope, SOB, chest pain	Hypotension, bradycardia	

Key References : Kwon YH, Fingert JH, Kuehn MH, Alward WL: Primary open-angle glaucoma, *N Engl J Med* 360(11):1113–1124, 2009; Jaakola MI, Ali-Melkkilä T, Kanto J, Kallio A, Scheinin H, Scheinin M: Dexmedetomidine reduces intraocular pressure, intubation responses and anaesthetic requirements in patients undergoing ophthalmic surgery, *Br J Anaesth* 68(6):570–575, 1992.

Perioperative Implications

Preoperative Preparation

- Maintain miosis by continuing topical and systemic treatment medications except for echothiophate, which should be stopped 4 wk prior to elective surgery.
- Pts taking acetazolamide, a carbonic anhydrase inhibitor, should have electrolytes checked preop with specific attention to Na^+, K^+, and bicarbonate levels.
- Antisialagogue premedication with glycopyrrolate or atropine is not contraindicated; however, several texts suggest scopolamine should be avoided due to its greater mydriatic effect.

Induction

- Blunt increases in IOP during laryngoscopy and intubation with the use of IV agents, which tend to decrease IOP. Controversy surrounds ketamine's effect.
- Succinylcholine is safe for induction and intubation, provided echothiophate has been discontinued.
- Hypotension should be avoided due to optic nerve perfusion concerns.
- Use of LMA may not increase IOP as much as direct laryngoscopy and intubation.

General Anesthesia

- All inhalational agents decrease IOP. This should be taken into account when providing anesthesia for eye exams.
- Hypercarbia should be avoided, since it increases IOP. Hypothermia, on the other hand, lowers IOP.
- Timolol is systemically absorbed and can cause asthmatic crises and severe sinus bradycardia, especially when other beta-blockers are administered. Betaxolol is more oculospecific, but additional beta blockade during anesthesia should be done with extreme caution. Effects of calcium channel blockers like verapamil are addictive and should be administered with care.
- Pneumoperitoneum and head-down positioning can increase IOP, but maintaining adequate anesthetic depth likely eliminates any measurable pressure increase.