- 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.

Regional Anesthesia
- Ester local anesthetics should be avoided in pts taking echothiophate due to reduced plasma cholinesterase activity and altered metabolism.

Extubation
- Avoid coughing and bucking, which can cause acute increases in IOP.
- Neuromuscular blockade reversal agents and antimuscarinics in usual dosages are considered safe.

Postoperative Period
- If emergency surgery is required in pts currently taking echothiophate, expect the need for prolonged postop ventilation.

Anticipated Problems/Concerns
- Avoid increases in IOP.
- Echothiophate therapy produces decreased plasma cholinesterase activity and should be stopped 4 wk prior to surgery to avoid a prolonged paralysis with the use of succinylcholine.
- Be aware that topical beta-blockers are systemically absorbed and can have systemic effects.

Glomus Jugulare Tumors

Ghaleb A. Ghani

Risk
- Account for 0.6% of head and neck tumors worldwide
- Male-to-female prevalence ratio: 1:2.5
- Slow-growing
- Can coexist with other paragangliomas
- Histologically benign but can be malignant with metastases
- Can be familial

Perioperative Risks
- Hypothermia
- Massive blood loss
- Venous air embolism
- Htn
- Bradycardia
- Hypotension
- Bronchospasm
- Tumor-parts embolization

Worry About
- Glomus jugulare tumors can appear in multiple locations; symptoms can persist after resection of the tumor.

Overview
- Tumors of neural crest at base of skull in jugular bulb area
- Highly vascular
- May extend into the posterior fossa
- May cause hydrocephalus
- May damage the lower cranial nerves (IX–XII)
- May involve internal carotid artery
- May grow into lumen of the jugular vein, as far as the RA
- May cause Horner syndrome
- May secrete catecholamines: 5%
- May secrete serotonin, histamine

Etiology
- Congenital (usually benign) hypertrophied arteriovenous anastomosis
- Epithelial cells with abundant capillary network

Usual Treatment
- Resection
- Embolization, alone or pre-resection
- Radiation
- Radiosurgery

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Cranial nerve injury	Hoarseness Dysphagia Tinnitus Vertigo	Tongue movement Soft palate motion Gag reflex Hearing test	Video laryngoscopy
CV	Htn Intravascular growth	Headache Palpitations	BP	Catecholamines level MRI/CT scans Angio (if indicated)
RESP	Aspiration	Cough Fever SOB	Rhonchi, wheezing	CXR
GI	Delayed gastric emptying	Heartburn Regurgitation		
GU		No different from normal		
CNS	Intracranial extension	Hearing loss Headache Dizziness Ataxia		CT scan MRI Paragangliomas in other locations

Key References: Jensen NF: Glomus tumors of the head and neck: anesthetic considerations, *Anesth Analg* 78(1):112–119, 1994; Heth J: The basic science of glomus jugulare tumors, *Neurosurg Focus* 17(2):E2, 2004.

Perioperative Implications

Preoperative Preparation
- Control Htn (in cathecholamine-secreting tumors). Preparation is similar to pheochromocytoma (see also Pheochromocytoma).
- Trial balloon occlusion of the internal carotid artery if there is a chance of ligating it intraop.
- Treat pneumonia.
- Metoclopramide for delayed gastric emptying.
- Adequate venous access for rapid fluid infusion.

Monitoring
- Consider arterial line and CVP.
- Monitor for venous air embolism (frequent ABG, $ETCO_2$, N_2; precordial Doppler).
- Cerebral oximetry.
- Facial nerve.
- Tenth nerve by using the NIM-EMG-ETT.

Maintenance
- Watch out for massive blood loss, Htn, hypotension, bradycardia, bronchospasm, venous air embolism, and tumor-parts embolization.
- Provide controlled hypotension if needed.
- Measure to decrease the ICP for intracranial extension:
  - Administer mannitol.
  - Assess for hyperventilation.
  - Optimize venous return from brain.
  - Assess CSF drainage.

Extubation
- Evaluate for airway swelling and neck hematoma.
- Evaluate for sequelae of pulm embolism.
- Evaluate for cranial nerves (IX–XII) injury.
- Evaluate for brain stem injury.

Adjuvants
- Controlled ventilation
- Muscle relaxants to prevent spontaneous ventilation intraop
- Controlled hypotension

Anticipated Problems/Concerns
- Loss of upper airway reflexes
- Airway obstruction
- Aspiration
- Delayed gastric emptying
- Ileus
- CNS insult
- CSF leak