

PROGRAM

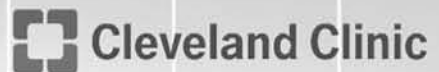
Ophthalmic Anesthesia Society

20th Annual Scientific Meeting

October 13-15, 2006
Westin Michigan Avenue Hotel
Chicago, IL

Program Co-Chairs
Richard Rivers MD PhD
Scott Greenbaum MD

Activity Director
Marc Allan Feldman MD MHS



Cleveland Clinic

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SCIENTIFIC AGENDA

FRIDAY, OCTOBER 13, 2006

- 12:50 **WELCOME REMARKS**
Richard Rivers MD PhD, President
Moderator: Richard Rivers MD PhD
- 1:00 **What's New: IOLs, Laser PKs, Bimanual Cataract Sx**
William W. Culbertson MD
Instructional Objectives: Describe new ophthalmic procedures that may require special anesthetic approaches
- 1:40 **Anesthesia for Strabismus Surgery**
Ezzat Aziz MBBCh MD FRCA MSc
Instructional Objectives: Compare types of anesthesia and effect on outcomes
- 2:20 **Obstructive Sleep Apnea: Ophthalmic Perspectives**
Howard D. Palte MB ChB FFA (SA)
Instructional Objectives: Describe the clinical features and sequelae of obstructive sleep apnea (OSA); discuss the impact on the conduct of ophthalmic anesthesia
- 3:00 **Questions and Answers**
- 3:10 **Break**
- 3:30 **The Patient with a Pacemaker or ICD: Why the Controversy?**
Marc A. Rozner PhD MD
Instructional Objectives: Describe the basic operation(s) of pacemakers and defibrillators; list reasons for pre-anesthetic evaluation patients with an implanted pulse generator; describe effects of electromagnetic interference on an implantable pulse generator; be aware that some ICD systems will deliver shock therapy inappropriately w/o ECG warning (even absent electromagnetic interference); organize appropriate post-anesthetic care of a patient with an implanted pulse generator.
- 4:10 **Topical and Cryoanesthesia in Cataract Surgery**
William W. Culbertson MD
Instructional Objectives: Describe indications, physiology and technique for topical and cryoanesthesia; Differentiate advantages, disadvantages and complications
- 4:50 **Questions and Answers**
- 5:00 **Anesthesia Jeopardy!!!**
Steven Gayer MD MBA, Game Host
Gary L. Fanning MD, Referee
Instructional Objectives: Game-show format testing knowledge of ophthalmic anesthesia techniques, solutions to case problems, and clinical challenges
- 6:00 **Adjourn**
- 6:00 **Reception**

SATURDAY, OCTOBER 14, 2006

- 7:50 **President's Welcome Remarks**
Moderator: Steven Gayer MD MBA
- 8:00 **History of Ophthalmic Anaesthesia: Have We Progressed?**
Anthony Rubin MB BCHIR FRCA
Instructional Objectives: Describe new drugs development which may require changes in techniques; discuss how surgical developments often allow for changes in anaesthetic technique; describe how changes in methods of preop assessment and care styles in hospital may dictate different anaesthetic techniques
- 8:40 **Punctuated Equilibrium: The Evolution of Recombinant Human Hyaluronidase**
Gregory I. Frost PhD
Instructional Objectives: Define the molecular, biochemical and pharmacologic properties of hyaluronidase; describe development of recombinant human forms of this enzyme
- 9:20 **USP 797: How this Standard May Affect the Way You Mix Medications.**
Serafin Gonzalez PharmD
Instructional Objectives: Provide a basic overview and introduction to USP <797>; Describe the applicatiohn to pharmacies and all health care-related facilities where medications are mixed; Outline methods to avoid non-compliance issues with USP <797>.
- 10:05 **Questions and Answers**
- 10:15 **Break**
- 10:30 **Anesthesia for the Syndromic Child Undergoing Eye Surgery**
Jacqueline L. Tutiven MD
Instructional Objectives: Describe risk factors and co-morbidities within this population to promote a safe peri-operative environment and guarantee a good surgical outcome. Reviewing common dysmorphism an anesthetic implications. Recognize potential difficult airway management of syndromic children Describe the prevalence and significance of CHD Anesthesia for the hypotonic child
- 11:10 **PONV after Eye Surgery: Myths and Facts**
Christian C. Apfel MD
Instructional Objectives: Assess the patients risk for PONV based on validated risk models; describe what can be expected from a single and combined antiemetic therapy; be able to implement a risk dependent antiemetic regimen in their own practice
- 11:50 **Questions and Answers**
- 12:00 **Lunch Break**
(continued)

SATURDAY, OCTOBER 14, 2006 *(continued)*

- Moderator:** Richard Rivers MD PhD
- 1:30 **Intraoperative Control of Intraocular Pressure**
Catherine Meschler MD
Instructional Objectives: Describe anatomical and physiological determinates of intraocular pressure; describe physiological and pharmacological interventions for controlling intraocular pressure intraoperatively
- 2:00 **Workshop Announcements**
- 2:15 **Workshops (Participants may attend two of three workshops)**
Instructional Objectives: Discuss the anatomical basis, technical aspects, and complications of orbital regional anesthesia. Describe orbital anatomy from the anesthesia provider's perspective; describe how to avoid complications through knowledge of anatomy and use of proper equipment; discuss the nomenclature of orbital regional anesthesia
- A. Parallel Approach to Orbital Blocks: Let's Track the Needle Tip**
Randolf R. Harvey BS CRNA
- B. Anatomy for Orbital Regional Anesthesia**
Gary L. Fanning MD
- C. Sub-Tenon's Technique: My Version**
Chandra Kumar MBBS DA FFARCSI FRCA MSc
- 3:15 **Break**
- 3:30 **Workshops (Second Session)**
- A. Repeat**
- B. Repeat**
- C. Sub-Tenon's Wet Lab**
Steven Gayer MD MBA and Scott Greenbaum MD
- 5:00 **Adjourn**

SUNDAY, OCTOBER 15, 2006

- 7:30 **Annual Meeting of the Membership (Non-CME)**
Moderator: Scott Greenbaum MD
- 8:00 **An Administrator's Perspective of a Successful Surgery Center**
Dan Simonson CRNA MHPA
Instructional Objectives: Describe the business viewpoint of running a surgery center; identify key ingredients for success
- 8:30 **Are There Any Complications of Sub-Tenon's Blocks?**
Chandra Kumar MBBS DA FFARCSI FRCA MSc
Instructional Objectives: Describe techniques for performing a safe sub-Tenon's block; describe how to prevent and treat complications of sub-Tenon's block
- 9:00 **Ophthalmic Anesthesia for the Cardiac Patient**
Marc Allan Feldman MD MHS
Instructional Objectives: Discuss preoperative precautions, and intraoperative monitoring for patients with cardiac disease
- 9:30 **Questions and Answers**
- 10:00 **Case Discussions**
Marc Allan Feldman MD MHS
Moderator
- 11:15 **Questions and Answers**
- 12:00 **Adjourn**

MEETING INFORMATION

ABOUT THIS MEETING

The purpose of the annual meeting of the Ophthalmic Anesthesia Society is to educate its members, as well as other interested healthcare professionals, and share information that will enable them to provide the highest level of anesthesia services during ophthalmic surgery.

THIS MEETING IS OF INTEREST TO:

- Anesthesiologists
- Ophthalmologists
- CRNAs
- RNs
- Ophthalmic Medical Professionals

ACCREDITATION

Nurse Anesthetist

Approved by the American Association of Nurse Anesthetists for 15 CE Credits: Code Number 28773, expiration date October 15, 2006.

Physician

This activity has been planned and implemented in accordance with the Essential Areas and policies of the Accreditation Council for Continuing Medical Education through the joint sponsorship of The Cleveland Clinic Foundation Center for Continuing Education and the Ophthalmic Anesthesia Society. The Cleveland Clinic Foundation Center for Continuing Education is accredited by the ACCME to provide continuing medical education for physicians.

The Cleveland Clinic Foundation Center for Continuing Education designates this educational activity for a maximum of 16.0 AMA PRA Category 1 Credit(s)%. Physicians should only claim credit commensurate with the extent of their participation in the activity.

This activity may be submitted for American Osteopathic Association Continuing Medical Education credit in Category 2.

DISCLAIMER

The information in this educational activity is provided for general medical education purposes only and is not meant to substitute for the independent

medical judgment of a physician relative to diagnostic and treatment options of a specific patient's medical condition. The viewpoints expressed in this CME activity are those of the authors/faculty. They do not represent an endorsement by The Cleveland Clinic Foundation. In no event will The Cleveland Clinic Foundation be liable for any decision made or action taken in reliance upon the information provided through this CME activity.

FACULTY DISCLOSURES

In accordance with the Standards for Commercial Support issued by the Accreditation Council for Continuing Medical Education (ACCME), The Cleveland Clinic Foundation Center for Continuing Education requires resolution of all faculty conflicts of interest to ensure CME activities are free of commercial bias.

The following faculty have indicated they have no relationship which, in the context of their presentation(s), could be perceived as a potential conflict of interest:

Christian C. Apfel MD PhD
Ezzat Samy Aziz MBBCh MD FRCA MSc
William W. Culbertson MD
Gary L. Fanning MD
Marc Allan Feldman MD MHS
Gregory I. Frost PhD
Steven Gayer MD MBA
Serafin Gonzalez PharmD
Scott Greenbaum MD
Randolf R. Harvey BS CRNA
Chandra Kumar MBBS DA FFARCSI FRCA MSc
Catherine Meschler MD
Howard D. Palte MB ChB FFA (SA)
Richard Rivers MD PhD
Marc A. Rozner PhD MD
Anthony Rubin MB BCHIR FRCA
Dan Simonson CRNA MHPA
Jacqueline L. Tutiven MD

MEETING INFORMATION

ABOUT THE OAS

The Ophthalmic Anesthesia Society is an organization of anesthesiologists, ophthalmologists, CRNAs, and nurses who are committed to sharing education and information that will enable them to provide the highest level of anesthesia services during ophthalmic surgery.

CONTACT INFORMATION

OAS
793-A Foothill Blvd., #119
San Luis Obispo, CA 93405
805 534 0300 (phone)
805 534 9030 (fax)
Info@EyeAnesthesia.org (email)
www.EyeAnesthesia.org

CO-CHAIRS & FACULTY

Meeting Co-Chairs



Richard Rivers MD PhD

Associate Professor
The Johns Hopkins University
Department of Anesthesiology &
Critical Care Medicine
Baltimore, MD
rrivers3@jhmi.edu



Scott Greenbaum MD

Associate Clinical Professor of
Ophthalmology
NYU School of Medicine
Greenbaum Eye Associates
Forest Hills, NY
TheCannula@aol.com

Activity Director



Marc Allan Feldman MD MHS

Head, Section of Anesthesia
Cole Eye Institute
Director, Cole Eye Institute
Operating Rooms
The Cleveland Clinic Foundation
Center for Continuing Education
Cleveland, Ohio
feldmam@ccf.org

Faculty



Christian C. Apfel MD PhD

Associate Professor
Department of Anesthesia
UCSF Mount Zion Hospital
San Francisco CA
Apfel@ponv.org
apfelc@anesthesia.ucsf.edu



Ezzat Samy Aziz MBBCh MD

FRCA MSc
Professor of Anaesthesia
Faculty of Medicine, Department of
Anaesthesia
Cairo University
Cairo Egypt
ezzataziz2002@hotmail.com



William W. Culbertson MD

Professor of Ophthalmology,
Director Bascom Palmer Eye
Institute Laser Vision Center
Department of Ophthalmology
Bascom Palmer Eye Institute,
University of Miami
Miller School of Medicine
Miami FL
wculbertson@med.miami.edu



Gary L. Fanning MD

Director of Anesthesiology &
Medical Director
Anesthesiology
Hauser-Ross Eye Institute
Sycamore IL
glfanning@aol.com



Gregory I. Frost PhD

Vice President &
Chief Scientific Officer
Halozyme Therapeutics, Inc.
San Diego CA
JMcDonald@halozyme.com



Steven Gayer MD MBA
Associate Professor of Anesthesiology & Ophthalmology, Director of Anesthesia Services
Bascom Palmer Eye Institute
Miami FL
Sgayer@miami.edu



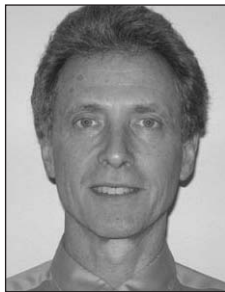
Howard D. Palte MB ChB FFA (SA)
Associate Professor of Ophthalmology
Director of Regional Anesthesia
Ann Bates Leach Eye Hospital
University of Miami
Miami FL
HPalte@med.miami.edu



Serafin Gonzalez PharmD
Director of Pharmacy Services
Bascom Palmer Eye Institute
Miami FL
SGonzale@med.miami.edu



Marc A. Rozner PhD MD
Professor of Anesthesiology & Cardiology
MD Anderson Cancer Center
The University of Texas
Houston TX
mrozner@mdanderson.org



Randolf R. Harvey BS CRNA
Chief of Anesthesia
Florida Eye Clinic / ASC
Altamonte Springs FL
rharvey5@cfl.rr.com



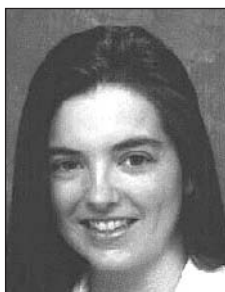
Anthony Rubin MB BCHIR FRCA
Consultant Anaesthetist
Wellington Eye Unit, London
London, England
rubin@easynet.co.uk



Chandra Kumar MBBS DA FFARCSI FRCA MSc
Professor of Anaesthesia
The James Cook University
Hospital and University of Teesside
Middlesbrough, UK
chandra.kumar1948@ntlworld.com
cmkumar@boas.org



Dan Simonson CRNA MHPA
Chief of Anesthesia,
Managing Partner
Spokane Eye Surgery Center
Spokane WA
DSimonson@mac.com



Catherine Meschler MD
Department of Anesthesiology & Critical Care Medicine
The Johns Hopkins University
Baltimore MD
cmeschl1@jhemi.jhmi.edu



Jacqueline L. Tutiven MD
Assistant Professor of Clinical Anesthesiology
Jackson Memorial Hospital /
Bascom Palmer Eye Hospital
University of Miami
Miami FL
JTutiven@med.miami.edu

FACULTY ABSTRACTS

What's New: IOLs, Laser PKs, Bimanual Cataract Surgery

William W. Culbertson, MD

Institute Laser Vision Center, Department of Ophthalmology
Bascom Palmer Eye Institute
University of Miami, Miller School of Medicine, Miami FL

In the last two years there have been significant advances in options for cataract and corneal surgery. New types of aspheric IOLs that correct spherical aberration and improve contrast sensitivity and visual acuity have been approved. These include the AMO Tecnis, the Alcon IQ and the Bausch and Lomb SofPort lens. Preoperative aberrometry is used to choose what lens is most suitable for an individual eye. An IOL that corrects both spherical refractive error and certain levels of astigmatism has been introduced by Alcon. Three presbyopia correcting IOLs, the AMO ReZoom, the Alcon Restor and the Eyeonics Crystalens have been introduced and are widely used to give appropriate patients simultaneous uncorrected reading and distance vision. Optimal visual results are obtained with these lenses when residual astigmatism is corrected either intraoperatively with corneal limbal relaxing incisions (LRIs) or postoperatively with astigmatic keratotomy (AK), conductive keratoplasty (CK) or LASIK. Intraocular clear lens extraction ("refractive lens exchange") and phakic refractive IOL placement with the AMO Verisyce or Staar ICL may be employed solely for correction of high levels of refractive error. In the case of cataract surgery, the ophthalmologist may make an supplemental uninsured surcharge to the patient for the additional evaluation and procedures necessary to choose and implant the appropriate aspheric IOL or presbyopic lens.

In corneal transplant surgery for corneal edema a new technique called DSEK or descemet's stripping endothelial keratoplasty, has become widely accepted. In this procedure, a thin lamella of the posterior donor cornea is placed on the back of the recipient cornea after its descemet's membrane has been stripped off. In contrast to full thickness corneal transplantation the DESK procedure takes 15 to 30 minutes, can be performed under topical anesthesia and visual recovery occurs in three to six weeks instead of the six to 24 months required for traditional penetrating keratoplasty.

A new laser that only to create corneal flaps for LASIK, called the IntraLase femtosecond laser, has been reconfigured to make intracorneal incisions to facilitate coadaptation of the edge of transplanted corneas. Typically the donor cornea and recipient cornea are cut with the laser in a refractive surgery facility. This area may be located in a different building or another area of the same building from the operating room where the surgery will be completed. Thus the patient will usually need to be moved from the refractive center to the operating room with the eye partially open. Modifications of the usual anesthesia used for penetrating keratoplasty are being explored to facilitate this double site procedure.

These new techniques for eye surgery present new paradigms for local and topical anesthesia. The differences between the anesthetic requirements for traditional ophthalmic surgery such as penetrating and anesthesia for these for these new alternative procedures will be discussed.

Strabismus Surgery Under Local Anaesthesia

Ezzat Samy Aziz MBBCh MD FRCA MSc DA(UK)

Cairo University, Egypt

From the earliest days of ocular surgery there have been significant advances in anaesthesia. Prior to the use of anaesthesia, ophthalmic procedures were performed rapidly to minimize the complications from patient movement. The development of topical anaesthesia afforded more time for the surgeon and markedly improved the comfort of the patient. When general anaesthesia became available, it enhances patient comfort yet it had its own set of complications. In the search for a better method of administering anaesthesia, regional blocks became popular as it limited the complications of general anaesthesia and still maintained adequate pain control.

Strabismus surgery with adjustable sutures has gained considerable popularity since its reintroduction more than 25 years ago (1). The general opinion is that the technique yields a better outcome, both in terms of alignment and long-term stability of the correction (2). Most surgeons use a two-stage approach, whereby muscle surgery is performed under general anaes-

thesia or regional block (3,4) with the postoperative adjustment made under topical anaesthesia. The timing of this adjustment varies from 3 hours to 4 days after surgery with the best results at least 6 hours after surgery (5). Using adjustable sutures during strabismus surgery allows the ophthalmic surgeon to prevent over or under correction of the squint in the postoperative period (6). Adjustable sutures can be attached to one or more ocular muscles. If the alignment is not satisfactory at the end of surgery, one or more of the muscles can be moved to a new position to produce the optimal alignment.

Although re-adjusting the sutures in this way helps to improve the success rate of strabismus surgery, it may have disadvantages. The patient may be reluctant to undergo another procedure, there may be Tissue oedema around or within the muscle to be adjusted may make the procedure difficult. Finally, operative complications such as suture slippage and suture breakage, all of which require return to the operating room for full exploration (7). Even after full adjustment, a residual over or under correction may still be present and necessitate another surgical procedure.

The popularity of the adjustable suture technique was the stimulus for introducing the use of topical anaesthesia in strabismus surgery(8). Unlike general anaesthesia and regional block techniques in eye surgery, topical anaesthesia avoids prolonged recovery of the eye muscles from motor block and allows suture adjustment at the time of surgery (rather than within 4 days), in one-stage adjustable suture strabismus surgery.

Intraoperative adjustment of the extraocular muscles under topical anaesthetic as a one-stage procedure has been well described but not widely practiced (8-14). Avoidance of the risks of general or regional anaesthesia are the obvious advantages of this approach, but this is somewhat offset by the need for patient cooperation and the fact that some discomfort is inevitably felt during the procedure(15). In order to decrease patient discomfort proper selection of patients is mandatory with avoidance of sedation to ensure full patient cooperation. Moreover, topical anaesthesia should be limited to horizontal strabismus than vertical or

oblique strabismus surgeries as these muscles are not easily accessible with greater patient discomfort(15). Further improvements in the area of pain control may increase the popularity of this approach.

A variety of anaesthetic agents and formulations have been used. Fells, (9) Chow, (10) and Kim et al (14) used topical anaesthetic drops only. Ruben and Elston (12) and Rauz and Govan (13) supplemented drops with subconjunctival infiltration of lidocaine with epinephrine over the muscle insertion Aziz and Rageh (15) used a sponge soaked in bupivacaine and placed it deep in the conjunctival fornix, as well as topical anaesthetic drops. Christopher et al (16) used cotton tipped applicators soaked in amethocaine 1% solution and apply this directly to the surgical field for approximately 1 minute before surgery begins.

Although most ophthalmic surgeons believe that pain in squint surgery results from pulling on a muscle, studies have proved that this is not altogether true. Pain also results from the retraction of an extraocular muscle causing traction on the pain sensitive periosteum at the muscle origin (17). Hooking a horizontal extraocular muscle (i.e. lateral or medial rectus) is much less painful if the patient is asked to look in the opposite direction to the hooked muscle. This causes relaxation of the muscle to be hooked so that traction on the periosteum is less with less discomfort (15). Hooking of the lateral rectus muscle was simple and painless, whereas hooking of the medial rectus muscle resulted in discomfort. The line of pull of the medial rectus is straighter than that of the lateral rectus because the arc of contact of the lateral rectus with the globe is greater. In addition there is an attachment between the medial rectus sheath and the pain-sensitive meninges around the optic nerve, but there is no such attachment in the lateral rectus. The absence of fascial attachments between the medial rectus and other muscles also results in a more direct pull on the periosteum. Slight backward pressure on the globe (retropulsion) with active abduction of the eye while hooking the medial rectus relieves the discomfort (15).

One stage adjustable suture strabismus surgery under topical anaesthesia is an effective method when patients are properly selected. Because it

does not interfere with muscle tone, topical anaesthesia produces a more accurate result than retrobulbar, peribulbar or sub-Tenon's anaesthesia. However, these regional blocks should be used in patients with vertical and oblique strabismus as the accessibility for these muscles are painful with topical anaesthesia.

References

1. Rosenbaum AL, Metz HS, Carlson M, Jampolsky AJ. Adjustable rectus muscle recession surgery. A follow-up study. *Arch Ophthalmol* 1977;95:817-20
2. Wygananski-Jaffe T, Wysanbeek Y, Bessler E, Spierer A. Strabismus surgery using the adjustable suture technique. *J Pediatr Ophthalmol Strabismus* 1999;36:184-8
3. Szymid SM, Nelson LB, Calhoun JH, Harley RD. Retrobulbar anaesthesia in strabismus surgery. *Arch Ophthalmol* 1984;102: 1325-7
4. Sanders RJ, Nelson LB, Deutsch JA. Peribulbar anaesthesia in strabismus surgery. *Am J Ophthalmol* 1990;109:705-8
5. Brown DR, Pacheco EM, Repka MX. Recovery of extraocular muscle function after adjustable suture strabismus surgery under local anaesthesia. *J Pediatr Ophthalmol Strabismus* 1992;29:16-20
6. Pratt-johnson JA. Adjustable-suture strabismus surgery. A review of 255 consecutive cases. *Can J Ophthalmol* 1985;20:105-9
7. Kraft SP, Jacobson ME. Adjustable suture techniques in strabismus surgery. *Ophthalmol Clin North Am* 1992;5:93-103
8. Diamond GR. Topical anaesthesia for strabismus surgery. *J Pediatr Ophthalmol Strabismus* 1989;26:86-90
9. Fells P. adjustable sutures. *Eye* 1988;2:33-5
10. Chow PC. Stability of one stage adjustable suture for the correction of horizontal strabismus. *Br J Ophthalmol* 1989;73: 541-6
11. Klyve P, Nicolaisen B. Topical anaesthesia and adjustable sutures in strabismus surgery. *Acta Ophthalmol (Copenh)* 1992;70:637-40
12. Ruben ST, Elston JS. One stage adjustable sutures; practical aspects. *Br J Ophthalmol* 1992;76:675-7
13. Rauz S, Govan JA. One stage vertical rectus muscle recession using adjustable sutures under local anaesthesia. *Br J Ophthalmol* 1996;80:713-8
14. Kim S, Yang Y, Kim J. Tolerance of patients and postoperative results: topical anaesthesia for strabismus surgery. *J Pediatr Ophthalmol Strabismus* 2000;37:344-8
15. Aziz ES, Rague M. Deep topical fornix nerve block versus peribulbar block in one step adjustable suture horizontal strabismus surgery. *Br J Anaesth* 2000;88:129-318.
16. Christopher BO, Victoria WY et al. Comparison of lidocaine 2% gel versus amethocaine as the sole anaesthetic agent for strabismus surgery. *Ophthalmology* 2003;110:1426-9
17. Lavrich JB, Nelson LB. Local anaesthesia for strabismus surgery. *Ophthalmol Clin North Am* 1992;5:131-41

Complications of Regional and Topical Anaesthesia in Ophthalmic blocks

By Dr Ezzat Samy Aziz Cairo University Introd

Obstructive Sleep Apnea: Ophthalmic Perspectives

Howard D. Palte MB ChB FFA (SA)

Ann Bates Leach Eye Hospital
University of Miami, Miami FL

Obstructive sleep apnea syndrome (OSAS) is an under-diagnosed condition affecting both adults and children. The true prevalence isn't really known but clinically evident cases probably represent the "tip of the iceberg." An increased awareness has prompted the ASA to recently issue "Practice Guidelines" for OSAS. Noteworthy, are comments on choice of venue and wisdom of ambulatory anesthesia in OSAS children.

The syndrome envelops both

- i) obstructive sleep apnea with airway collapse and diminished airflow despite active respiratory effort and
- ii) central sleep apnea with loss of respiratory drive and compensatory hyperventilation.

A mature association with obesity, cardiovascular disease and heart failure predisposes this subpopulation to increased peri-operative morbidity. Definitive diagnosis relies on polysomnographic evidence of sleep-disordered breathing and associated physiologic disturbances. Conservative management focuses on weight reduction and use of nocturnal continuous positive airway pressure (CPAP) devices.

Pre-operative case detection is vital since even short term CPAP therapy may alleviate significant co-morbidities. Anesthesia management is concentrated on the triad of airway control, prevention of cardiovascular compromise and avoidance of respiratory depressants. Regional anesthesia remains the technique of choice for ophthalmic surgery. However, general anesthesia remains mandatory for children and in those adults unable to tolerate regional techniques.

While sedation is frequently employed as an adjunct to regional techniques; its use may be hazardous in this scenario. The risk of airway compromise, inability to ventilate and respiratory embarrassment are ever present. In addition, attention must be paid to control of intra-

ocular pressure, post-operative airway edema and effective analgesia.

In managing these patients, the attending anesthesiologist requires appropriate skill in both sedation and regional anesthesia techniques. Incomplete block or over-sedation may cause unexpected patient movement with potential for poor visual outcome.

OSAS causes daytime somnolence and predisposes sufferers to enhanced potential for accidents and eye injuries. Select open globe injuries may be amenable to repair under regional block.

Ultimately, favorable outcomes depend on proactive planning, interpersonal communication and appropriate choice of venue.

The Patient with a Pacemaker or ICD: Why the Controversy?

Marc A. Rozner PhD MD

MD Anderson Cancer Center
The University of Texas, Houston TX

<p><u>Preoperative Key Points</u></p> <ul style="list-style-type: none">• Identify the manufacturer and model of the generator.• Have the pacemaker or defibrillator interrogated by a competent authority shortly before the anesthetic.• Obtain a copy of this interrogation. Ensure that the device will pace the heart.• Consider replacing any device near its elective replacement period in a patient scheduled to undergo either a major surgery or surgery within 25 cm of the generator.• Determine the patient's underlying rhythm / rate to determine the need for backup pacing support.• For conventional pacemakers, identify the magnet rate and rhythm, if a magnet mode is present and magnet use is planned.• Program minute ventilation rate responsiveness off, if present.• Program all rate enhancements off.• Consider increasing the pacing rate to optimize oxygen delivery to tissues for major cases.• Generally, suspension of antitachycardia therapy in a defibrillator is indicated. Although a magnet might work, in general, magnet therapy has been associated with inappropriate ICD discharge.	<p><u>Intraoperative Key Points</u></p> <ul style="list-style-type: none">• Monitor cardiac rhythm / peripheral pulse with pulse oximeter (plethysmography) or arterial waveform.• Disable the “artifact filter” on the EKG monitor.• Avoid use of the monopolar electrosurgical unit (ESU).• Use bipolar ESU if possible; if not possible, then pure cut (monopolar ESU) is better than “blend” or “coag.”• Place the ESU current return pad in such a way to prevent electricity from crossing the generator-heart circuit, even if the pad must be placed on the distal forearm and the wire covered with sterile drape.• If the ESU causes ventricular oversensing and pacer quiescence, limit the period(s) of asystole.• Perhaps avoid sevoflurane or isoflurane in the patient with long QT syndrome. <p><u>Postoperative Key Points</u></p> <ul style="list-style-type: none">• Have the device interrogated by a competent authority postop. Some rate enhancements can be re-initiated, and optimum heart rate and pacing parameters should be determined. The ICD patient must be monitored until the antitachycardia therapy is restored.
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Pacemakers

Almost 2,000 pacemaker models have been produced by 26 companies, and more than 220,000 adults and children in the US undergo new pacemaker placement yearly. Nearly 3 million US patients have pacemakers. Many factors lead to confusion regarding the behavior of a device and the perioperative care of the pacemaker patient, especially since literature reviews have not kept pace with technologic developments and some contain misinformation.

Intrathoracic cardiac gadgets consist of an impulse generator and lead(s). Leads can have one (unipolar), two (bipolar), or multiple (multipolar) electrodes with connections in multiple chambers. In unipolar pacing, the generator case serves as an electrode, and tissue contact can be disrupted by pocket gas.¹

<u>Position I</u>	<u>Position II</u>	<u>Position III</u>	<u>Position IV</u>	<u>Position V</u>
Chambers Paced	Chambers Sensed	Response to Sensing	Programmability	Multisite Pacing
O = None	O = None	O = None	O = None	O = None
A = Atrium	A = Atrium	I = Inhibited	R = Rate Modulation	A = Atrium
V = Ventricle	V = Ventricle	T = Triggered		V = Ventricle
D = Dual (A+V)	D = Dual (A+V)	D = Dual (T+I)		D = Dual (A+V)

Pacemaker Magnets

NOT ALL PACEMAKERS SWITCH TO A CONTINUOUS ASYNCHRONOUS MODE WHEN A MAGNET IS PLACED. Also, not all models from a given company behave the same way. (Table 3).⁴⁻⁶

Sinus Node Disease
Atrioventricular (AV) Node Disease
Long Q-T Syndrome
Hypertrophic Obstructive Cardiomyopathy (HOCM) ^{2,3}
Dilated Cardiomyopathy (DCM) ³

Preanesthetic Evaluation; Pacemaker Reprogramming

Preoperative management of the patient with a pacemaker includes evaluation and optimization of coexisting disease(s). ACC guidelines suggest that cardiac testing (stress tests, echocardiograms) should be dictated by the patient's underlying disease(s), symptomatology, interval from the last testing, and planned intervention.⁷ Both the ASA advisory and the ACC guidelines suggest a preoperative comprehensive evaluation of the generator.

Reprogramming the pacing mode to asynchronous pacing at a rate greater than the patient's underlying rate usually ensures that no over- or undersensing from EMI will take place. Note, however, that setting a device to asynchronous mode could have the potential to create a malignant rhythm in the patient with significant structural compromise of the myocardium.⁸ Reprogramming a device **will not** protect it from internal damage or reset caused by EMI.

In general, rate responsiveness and "enhancements" (dynamic atrial overdrive, hysteresis, sleep rate, A-V search, etc.) should be disabled by programming.^{7,9,10} Special attention must be given to any device with a minute ventilation (bioimpedance) sensor (Table 5), since pacemaker mediated tachycardia (PMT) has been observed secondary to mechanical ventilation,^{11,12} monopolar ("Bovie") electro-surgery,^{11,13,14} and connection to an EKG monitor with respiratory rate monitoring.¹⁵⁻²⁰ Sometimes, this PMT has led to inappropriate therapy.¹²

Table 3: Pacemaker Magnet Behavior
No apparent rhythm or rate change
No magnet sensor (some pre-1985 Cordis, Tele models)
Magnet mode disabled (possible with Biotronik, CPI, Guidant, Pacesetter, St Jude models)
EGM mode enabled (CPI, Guidant, Pacesetter, St Jude)
Program rate pacing in already paced patient (many CPI, Intermedics, Pacesetter, St Jude, Tele)
Brief (10-100 beats) asynchronous pacing, then return to program values (all Intermedics; most Biotronik models when programmed to their default state)
Continuous or transient loss of pacing
Discharged battery (some pre-1990 devices)
Diagnostic "Threshold Test Mode" (Siemens)
Asynchronous "high rate" pacing
Medtronic (most models) 85 bpm, 65 if battery depleted
Guidant Medical / CPI (current models since 1990, magnet mode enabled) > 85 bpm (max 100), 85 if battery depleted
Pacesetter / St Jude Medical (current models since 1990, magnet mode enabled) > 87 bpm (max 98.6 bpm), 86.3 if battery depleted
ELA Medical (current models since 1989) > 80 bpm (max 96 bpm), 80 if battery depleted. ELA devices take 8 additional asynchronous cycles (six at magnet rate, then two at programmed rate) upon magnet removal. Magnet placement increases the pacing voltage to 5v
Biotronik (when programmed to asynchronous mode, [not the default state]) 90 bpm, 80 if battery depleted
Asynchronous pacing without rate responsiveness using parameters possibly not in patient's best interest

Table 4: Pacemaker Reprogramming Likely Needed
Any rate responsive device - see text (problems are well known, ^{21,22} problems have been misinterpreted with potential for patient injury, ^{10,11,16,20} and the FDA has issued an alert regarding devices with minute ventilation sensors – see Table 5 ¹⁸
Special pacing indication (HOCM, DCM, pediatric patient)
Pacemaker-dependent patient
Major procedure in the chest or abdomen
Rate enhancements are present that should be disabled
Special Procedures Lithotripsy Transurethral or Hysteroscopic Resection Electroconvulsive Therapy Succinylcholine use MRI (generally contraindicated by device manufacturers), but perhaps possible in selected patients ^{23,24}

Table 5: Devices with Minute Ventilation Sensors
ELA Medical Symphony (new) Brio (212, 220, 222) Opus RM (4534) Chorus RM (7034, 7134) Talent (130, 213, 223)
Guidant Medical and/or CPI Pulsar (1172, 1272) Pulsar Max (1170, 1171, 1270) Pulsar Max II (1180, 1181, 1280) Insignia Plus (1194, 1297, 1298)
Medtronic Kappa 400 series (401, 403)
Telectronics / St Jude Meta (1202, 1204, 1206, 1230, 1250, 1254, 1256) Tempo (1102, 1902, 2102, 2902)

Intraoperative (or Procedure) Management of Pacemakers

No special technique or monitoring is needed for the pacemaker patient, but attention must be given to a number of concerns (Table 6). Magnet placement during electrosurgery might prevent aberrant pacemaker behavior. Spurious reprogramming with magnet placement is unlikely. If monopolar electrosurgery is to be used, then the ESU current-return pad must be placed to ensure that ESU current path does not cross the pacemaking system. Some authors recommend placement of this pad on the shoulder for head and neck procedures or the distal arm (with sterile draping of the wire) for breast and axillary procedures.²⁵

Pacemaker Failure

Pacemaker failure has three etiologies: 1) failure to capture; 2) lead failure; or 3) generator failure. Failure to capture can result from myocardial ischemia / infarction, acid-base disturbance, electrolyte abnormalities, or abnormal antiarrhythmic drug level(s). External pacing might further inhibit pacemaker output at pacing energies that will not produce myocardial capture.^{26,27} Sympathomimetic drugs generally lower pacing threshold. Outright

generator and/or lead failure is rare.

Post Anesthesia Pacemaker Evaluation

Any pacemaker that was reprogrammed for the operating room should be reset appropriately. For non-reprogrammed devices, most manufacturers recommend interrogation to ensure proper functioning and remaining battery life if any electrosurgery was used. Both the ASA practice advisory and ACC Guidelines recommend a post-procedure interrogation.⁷

EKG monitoring of the patient must include the ability to detect pacemaker discharges (“artifact filter” disabled)
Perfused (peripheral) pulse must be monitored with a waveform display
The pacemaker rate might need to be increased due to an increased oxygen demand
BiV and HOCM patients probably need beat-to-beat cardiac output monitoring
Appropriate equipment must be on hand to provide backup pacing and/or defibrillation

Implantable Cardioverter-Defibrillator (ICD) Overview

The development of an implantable, battery powered device able to deliver sufficient energy to terminate ventricular tachycardia (VT) or fibrillation (VF) has represented a major medical breakthrough for patients with a history of ventricular tachydysrhythmias or cardiomyopathy. Initially approved by the US FDA in 1985, nearly 100,000 new devices will be placed this year, and more than 240,000 patients have these devices today.

Indications for ICD placement initially centered around the patient who presented with sudden cardiac arrest, VT, or VF. Indications were broadened with time to include the patient with an inducible ventricular tachydysrhythmia. Currently, indications include prophylactic use in the patient with cardiomyopathy, whether they have experienced spontaneous or inducible VT or VF. Table 7 shows current indications for ICD placement.

Ventricular tachycardia
Ventricular fibrillation
Post-MI patients with EF 30% (MADIT II) ²⁸
Cardiomyopathy from any cause with EF 35% (SCD-HeFT) ²⁹
Hypertrophic cardiomyopathy
Awaiting heart transplant ³⁰
Long Q-T syndrome ³¹
Arrhythmogenic right ventricular dysplasia ⁶⁰
Brugada syndrome (right bundle branch block, S-T segment elevation in leads V1-V3) ^{32,33}

Like pacemakers, ICDs have a four-place code (Table 8).³⁴ The Pacemaker Code can be used instead of Position IV for robust identification of the ICD behavior.

Table 8: NASPE / BPG Generic Defibrillator Code (NBD)			
<u>Position I</u>	<u>Position II</u>	<u>Position III</u>	<u>Position IV</u> (or use Pacemaker Code)
Shock Chambers	Antitachycardia Pacing Chambers	Tachycardia Detection	Antibradycardia Pacing Chambers
O = None	O = None	E = Electrogram	O = None
A = Atrium	A = Atrium	H = Hemodynamic	A = Atrium
V = Ventricle	V = Ventricle		V = Ventricle
D = Dual (A+V)	D = Dual (A+V)		D = Dual (A+V)

Inappropriate ICD shocks

Despite improvements in detection of ventricular dysrhythmias and differentiation from tachycardias that are untreatable with ICDs (Table 9),³⁵ more than 10% of shocks are for rhythm other than VT or VF.³⁶ Supraventricular tachycardia remains the most common etiology of inappropriate shock therapy,^{37,38} and causes of inappropriate shock have been reviewed elsewhere.³⁹

Table 9: ICD Features to Reduce Undesired Shock
Onset criteria - usually, onset of VT is abrupt, whereas onset of SVT has sequentially shortening R-R intervals
Stability criteria - usually, the R-R interval of VT is relatively constant, whereas the R-R interval of atrial fibrillation with rapid ventricular response is quite variable
QRS width criteria - usually, QRS width in SVT is narrow (<110 msec), whereas QRS width in VT is wide (>120 msec)
"Intelligence" in dual chamber devices attempting to associate atrial activity to ventricular activity
Morphology waveform analysis with comparison to stored historical templates

Inappropriate shock therapy also can result from a failing lead. At this time, there are at least two right ventricular ICD leads that develop “chatter” (chatter is the electrophysiology term for phantom R wave signals appearing on an ICD lead) and can lead to inappropriate shock.⁴⁰ These inappropriate therapies will be delivered without warning or prior electrocardiographic evidence of a problem, since they result from this intrinsic noise signal. Often, the first evidence of a problem is the occurrence of an inappropriate shock.

ICD Magnets

Most ICDs suspend tachydysrhythmia detection (and therefore therapy) when a magnet is appropriately placed. Some devices from Angeion, CPI, Guidant, Pacemaker, St Jude Medical or Ventritex can be programmed to ignore magnet placement. Depending upon programming, **antitachycardia therapy in some Guidant or CPI devices can be permanently disabled by magnet placement for 30 seconds, and patients have been discovered with their ICD antitachycardia therapy unintentionally disabled.**⁴¹ Guidant has recently recommended suspending magnet function in some of their ICDs.⁴²

Preanesthetic Evaluation; ICD Reprogramming

In addition to evaluating and optimizing comorbid disease(s), every ICD patient should undergo preoperative ICD interrogation. In general, for most patients, antitachycardia therapy should be disabled during their procedure (see ASA Advisory⁴³ and ACC Guidelines⁷). In a setting where no monopolar electrocautery is to be used, where the device function is verified preoperatively, where the device and leads are not on recall, and where blood loss and fluid administration will likely be kept to a minimum, non-suspension of antitachycardia therapy could be considered acceptable. The comments in the pacing section apply here for antibradycardia pacing.

Intraoperative (or Procedure) ICD Management

At this time, no special monitoring or anesthetic technique (owing to the ICD) is required for the ICD patient. EKG monitoring and the ability to deliver external cardioversion or defibrillation must be present during the time of ICD disablement.

Should cardioversion or defibrillation be needed, the defibrillator pads should be placed to avoid the pulse generator to the extent possible. Nevertheless, one should remember that the patient, not the ICD, is being treated. The recommendations in the section "Intraoperative (or Procedure) Management of Pacemakers" apply here as well. ICDs should be disabled prior to insertion of a central line to prevent inappropriate shock and possible ICD failure.⁴⁴

Post Anesthesia ICD Evaluation

The ICD must be reinterrogated and re-enabled. All events should be reviewed and counters should be cleared. The pacing parameters must be checked and reprogrammed as necessary.

Summary

Electronic miniaturization has permitted the design and use of sophisticated electronics in patients who have need for artificial pacing and/or automated cardioversion / defibrillation of their heart. These devices are no longer confined to merely keeping the heart beating between a minimum rate (pacing function) and a maximum rate (ICD functions), as they are now being used as therapy to improve the failing heart. Both the aging of the population and our ability to care for a patient with increasingly complex disease suggest that we will be caring for many more patients with these devices, and we must be prepared for this situation. Safe and efficient clinical management of these patients depends upon our understanding of implantable systems, indications for their use, and the perioperative needs that they create.

Reference List

1. Lamas GA et al. Pacemaker malfunction after nitrous oxide anesthesia. *Am J Cardiol* 1985; 56(15):995.
2. Hayes DL. Evolving indications for permanent pacing. *Am J Cardiol* 1999; 83(5B):161D-165D.
3. Auricchio A et al. The pacing therapies for congestive heart failure (PATH-CHF) study: Rationale, design, and endpoints of a prospective randomized multicenter study. *Am J Cardiol* 1999; 83(5B):130D-135D.
4. Rozner MA et al. Perioperative care of the patient with a pacemaker. In: Stone DJ, Bogdanoff DL, Leisure GS, Spiekermann BF, Mathis DD, eds. *Perioperative care: anesthesia, surgery and medicine*. Philadelphia: Mosby; 1997: 53-67.
5. Purday JP, Towey RM. Apparent pacemaker failure caused by activation of ventricular threshold test by a magnetic instrument mat during general anaesthesia. *Br J Anaesth* 1992; 69(6):645-646.
6. Bourke ME. The patient with a pacemaker or related device. *Can J Anaesth* 1996; 43(5 Pt 2):24-41.
7. Eagle, K. A. et al. ACC/AHA guideline update for perioperative cardiovascular evaluation for noncardiac surgery. Published January, 2002. Available at: http://www.acc.org/clinical/guidelines/perio/update/pdf/perio_update.pdf. Accessed June 29, 2002.
8. Preisman S, Cheng DC. Life-threatening ventricular dysrhythmias with inadvertent asynchronous temporary pacing after cardiac surgery. *Anesthesiology* 1999; 91(3):880-883.
9. Andersen C, Madsen GM. Rate-responsive pacemakers and anaesthesia. A consideration of possible implications. *Anaesthesia* 1990; 45(6):472-476.
10. Levine PA. Response to "rate-adaptive cardiac pacing: implications of environmental noise during craniotomy". *Anesthesiology* 1997; 87(5):1261.
11. Madsen GM, Andersen C. Pacemaker-induced tachycardia during general anaesthesia: a case report. *Br J Anaesth* 1989; 63(3):360-361.
12. von Knobelsdorff G et al. [Interaction of frequency-adaptive pacemakers and anesthetic management. Discussion of current literature and two case reports]. *Anaesthesist* 1996; 45(9):856-860.
13. Van Hemel NM et al. Upper limit ventricular stimulation in respiratory rate responsive pacing due to electrocautery. *Pacing Clin Electrophysiol* 1989; 12(11):1720-3.
14. Wong DT, Middleton W. Electrocautery-induced tachycardia in a rate-responsive pacemaker. *Anesthesiology* 2001; 94(4):710-1.
15. Chew EW et al. Inappropriate rate change in minute ventilation rate responsive pacemakers due to interference by cardiac monitors. *Pacing Clin Electrophysiol* 1997; 20(2 Pt 1):276-282.
16. Rozner MA, Nishman RJ. Pacemaker-driven tachycardia revisited. *Anesth Analg* 1999; 88(4):965.
17. Wallden J et al. Supraventricular tachycardia induced by Datex patient monitoring system. *Anesth Analg* 1998; 86(6):1339.
18. Interaction between minute ventilation rate-adaptive pacemakers and cardiac monitoring and diagnostic equipment. Center for Devices and Radiologic Health. Published October 14, 1998. URL= <http://www.fda.gov/cdrh/safety/minutevent.html>. Accessed December 1, 2002.
19. Southorn PA et al. Monitoring equipment induced tachycardia in patients with minute ventilation rate-responsive pacemakers. *Br J Anaesth* 2000; 84(4):508-509.
20. Rozner MA, Nishman RJ. Electrocautery-induced pacemaker tachycardia: why does this error continue? *Anesthesiology* 2002; 96(3):773-4.
21. Schwartzburg CF et al. Rate-adaptive cardiac pacing: implications of environmental noise during craniotomy. *Anesthesiology* 1997; 87(5):1252-1254.
22. Aldrete JA et al. Pacemaker malfunction due to microcurrent injection from a bioimpedance noninvasive cardiac output monitor. *J Clin Monit* 1995; 11(2):131-133.
23. Martin ET et al. Magnetic resonance imaging and cardiac pacemaker safety at 1.5-Tesla. *J Am Coll Cardiol* 2004; 43(7):1315-1324.
24. Rozner MA et al. Pacemaker complication during MRI. *J Am Coll Cardiol* 2005; 45(1):161-162.
25. Frankina MF et al. Pacemakers: Perioperative evaluation, management and complications. *Anesthesiology* 2000; 93:A1193.
26. Mychaskiw G, Eichhorn JH. Interaction of an implanted pacemaker with a transesophageal atrial pacemaker: report of a case. *J Clin Anesth* 1999; 11(8):669-671.
27. Moskowitz DM et al. External chest wall stimulation to suppress a permanent transvenous pacemaker in a patient

- during endovascular stent graft placement. *Anesthesiology* 1998; 89(2):531-533.
28. Moss AJ et al. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. *N Engl J Med* 2002; 346(12):877-883.
29. Bardy GH et al. Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure. *N Engl J Med* 2005; 352(3):225-237.
30. Sandner SE et al. Survival benefit of the implantable cardioverter-defibrillator in patients on the waiting list for cardiac transplantation. *Circulation* 2001; 104(12 Suppl 1):I171-I176.
31. Khan IA. Long QT syndrome: diagnosis and management. *Am Heart J* 2002; 143(1):7-14.
32. McKenna WJ et al. Diagnosis of arrhythmogenic right ventricular dysplasia/cardiomyopathy. Task Force of the Working Group Myocardial and Pericardial Disease of the European Society of Cardiology and of the Scientific Council on Cardiomyopathies of the International Society and Federation of Cardiology. *Br Heart J* 1994; 71(3):215-218.
33. Brugada P, Geelen P. Some electrocardiographic patterns predicting sudden cardiac death that every doctor should recognize. *Acta Cardiol* 1997; 52(6):473-484.
34. Bernstein AD et al. North American Society of Pacing and Electrophysiology policy statement. The NASPE/BPEG defibrillator code. *Pacing Clin Electrophysiol* 1993; 16(9):1776-1780.
35. Swerdlow CD. Supraventricular tachycardia-ventricular tachycardia discrimination algorithms in implantable cardioverter defibrillators: state-of-the-art review. *J Cardiovasc Electrophysiol* 2001; 12(5):606-612.
36. Hurst TM et al. Inappropriate management of self-terminating ventricular arrhythmias by implantable cardioverter defibrillators despite a specific reconfirmation algorithm: a report of two cases. *Pacing Clin Electrophysiol* 1997; 20(5 Pt 1):1328-1331.
37. Prasad K et al. Inappropriate shocks in patients receiving internal cardioverter-defibrillators for malignant ventricular arrhythmias. *Indian Heart J* 1997; 49(4):403-407.
38. Schumacher B et al. Radiofrequency catheter ablation of atrial flutter that elicits inappropriate implantable cardioverter defibrillator discharge. *Pacing Clin Electrophysiol* 1997; 20(1 Pt 1):125-127.
39. Rozner MA. The patient with an implantable cardioverter-defibrillator. *Progress in Anesthesiology* 1999; 13:43-52.
40. Ellenbogen KA et al. Detection and management of an implantable cardioverter defibrillator lead failure: incidence and clinical implications. *J Am Coll Cardiol* 2003; 41(1):73-80.
41. Rasmussen MJ et al. Unintentional deactivation of implantable cardioverter-defibrillators in health care settings. *Mayo Clin Proc* 2002; 77(8):855-9.
42. Guidant. Urgent medical device safety information and corrective action (Contak Renewal [3,4,RF] ICD [magnet switch]). Published June 23, 2005. Available at: http://www.guidant.com/physician_communications/RENEWAL3_RENEWAL4.pdf. Accessed June 23, 2005.
43. Practice advisory for the perioperative management of patients with cardiac rhythm management devices: pacemakers and implantable cardioverter-defibrillators: a report by the American Society of Anesthesiologists Task Force on Perioperative Management of Patients with Cardiac Rhythm Management Devices. *Anesthesiology* 2005; 103(1):186-198.
44. Varma N et al. Central venous access resulting in selective failure of ICD defibrillation capacity. *Pacing Clin Electrophysiol* 2001; 24(3):394-395.

Topical and Cryoanesthesia in Cataract Surgery

William W. Culbertson, MD

Institute Laser Vision Center, Department of Ophthalmology
Bascom Palmer Eye Institute
University of Miami, Miller School of Medicine, Miami FL

Anesthesia for cataract surgery has come full circle back to where it started i.e. topical anesthesia or even no anesthesia in certain cases. The majority of routine cataract surgery is now performed under topical anesthesia with supplemental monitored intravenous sedation. Indications for topical anesthesia include anticipated routine surgery and patient cooperation, long axial length, anticoagulation or bleeding diathesis. Often topical anesthesia is supplemented with intraocular lidocaine anesthesia particularly in younger patients and or when iris manipulation is anticipated. When patients have known allergic sensitivity to topical or local anesthetic agents, cryoanesthesia may be considered. In cryoanesthesia the eye is first cooled with a 15 minute icepack. Then the clear corneal surgery is performed with chilled balanced salt solution irrigated around the incision and in the infusion bottle. Authors from India have described no anesthesia cataract surgery in which incisions are made quickly and external stimulation of the eye is minimized. Incision sizes have been minimized and in bimanual techniques the incision size may be reduced from 2750 microns down to 700 microns. Small external incisions have proven to require less or no anesthesia. Thus in the last 250 years we have indeed come full circle back to the days of couching of cataracts with no topical or peribulbar anesthesia and tiny peripheral incisions.

Faculty Abstracts: Saturday, October 14, 2006

History of Ophthalmic Anaesthesia: Have We Progressed?

Anthony Rubin MB BCHIR FRCA

Wellington Eye Unit, London, England

Reference will be made to "couching"; and a video of the technique will be shown (kindly provided by Mr Brian Little, an eminent ophthalmic surgeon in London).

This talk will then review the events of 1884, a

dramatic year marked by the introduction of cocaine by Koller, an ophthalmologist working in Vienna, the first injection of local anaesthetic into the orbit by Knapp, and the first reference to sub-Tenon's block by Turnbull, oculist to the German Hospital of Philadelphia.

Once the local anaesthetic properties of cocaine had been established, concerns were expressed as to its effects on the cornea. Rabbit experiments undertaken by Koller suggested that it was exposure and drying rather than cocaine that led to corneal opacity.

The combination of cocaine with adrenaline causing less bleeding and prolonging the action while reducing the absorption and the chance of toxicity was already understood by the time of Allen's textbook of 1915.

By 1915, Allen wrote, "the use of general anesthesia has been reduced to a minimum and by some is almost completely discarded". Allen also described the difficulty of obtaining anaesthesia of the iris with topical anesthesia alone, advocating stronger solutions of cocaine and more time for it to penetrate or the use of intracamerular or subconjunctival routes.

It was felt that for cataract surgery, topical with or without subconjunctival injection would suffice but for deeper operations, "the conjunctiva should be seized and the point of the needle inserted as deeply as possible into Tenon's capsules where some solution should be injected".

For enucleation, it was considered necessary to block the recti muscles close to their origins and use injections deep into the orbit to block the region of the ciliary ganglion.

As sub-Tenon's block has become the most popular local anaesthetic technique in the UK, its history will be traced up to the present day and parallel developments will be discussed as well. The talk will end with a peek into the future.

References

Tenon JR. Sur une nouvelle tunique de l'oeil. Memoirs et observations sur l'Anatomie, la Pathologie et la Chirurgie. Paris, Tome 1, 1806

Koller C. Bericht uber die sechszehnte Versammlung der ophthalmologischen Gesellschaft. Heidelberg 1884. Rostock, Universitats-Buchdruckerei von Adler's Erben 60-63

Knapp H. On cocaine and its use in ophthalmic and general surgery. Arch Ophth 1884; 13: 402-448

Turnbull CS The hydrochlorate of cocaine, a judicious opinion of its merits. Editorial Med Surg Rep (Boston) 1884; 29: 628-629.

Koller C. Hand Book Med Sci 1901; iii: 156

Allen CW. Local and Regional Anesthesia WB Saunders Company Philadelphia and London 1915 p580

Atkinson WS. Arch Ophthalmol 1936;16:501

Stevens JD, Franks WA, Orr G et al. Four-quadrant local anaesthesia technique for vitreoretinal surgery. Eye 1992; 6: 583-586

Stevens JD. A new local anaesthesia technique for cataract extraction by one quadrant sub-Tenon's infiltration. Br J Ophthalmol 1992; 76: 670 - 674

Ripart J, Lefrant J-Y, Lalourcey L et al. Medial canthus (Caruncle) single injection periorcular anesthesia. Anesth Analg 1996; 83: 124-1238

Punctuated Equilibrium: The Evolution of Recombinant Human Hyaluronidase

Gregory I. Frost PhD

Halozyme Therapeutics, Inc., San Diego CA

Hyaluronidases are a family of β 1-4 endoglycosaminidase enzymes that depolymerize hyaluronan, a high molecular weight linear viscous polysaccharide found in the interstitial matrix. Animal extracted forms of these enzymes have been utilized for many years in periorcular blocks such as retrobulbar anesthesia to promote the dispersion of anesthetics such as bupivacaine and lidocaine. The development of a recombinant form of this enzyme was based upon the discovery of the human hyaluronidase gene, PH20, which normally exists as a glycosylphosphatidyl inositol (GPI)-anchored protein locked to the plasma membrane of cells. By successive carboxy-terminal deletion mutagenesis, a soluble, enzymatically active deletion variant of human PH20 was found that, when introduced into a mammalian expression system, resulted in the secretion of a 447 amino acid glycoprotein. Purification of the full length rHuPH20 protein from chemically defined medium was achieved by successive chromatographic steps resulting in a highly purified protein with a specific activity greater than 100,000 USP Units/mg. The purified enzyme was found to depolymerize hyaluronan *in vitro*, and was capable of dispersing intradermally injected trypan blue tracer dye in animal models in a dose dependent fashion. This spreading activity was shown to be reversible in animal models within 24 hours of administration. We conclude that enzymatically active recombinant human hyaluronidase can be genetically engineered for secretion in mammalian cells and purified to high specific activity.

USP 797: How this Standard May Affect the Way You Mix Medications

Serafin Gonzalez PharmD

Bascom Palmer Eye Institute, Miami Fl

	<p>USP <797>: How this Standard May Affect the Way You Mix Medications</p> <p>Serafin Gonzalez, PharmD Director of Pharmacy Services Bascom Palmer Eye Institute</p>

	<p>Compounding</p> <p><i>The combining, mixing, or altering of ingredients to create a <u>customized medication</u> for an individual patient ...</i> <small>http://www.fda.gov/ola/2003/pharmacycompound1023.html</small></p> <p><i>The professional act of incorporating ingredients to create a finished product for dispensing to a patient or for administration by a practitioner or his agent; and shall specifically include the professional act of preparing a <u>unique finished product</u> ...</i> <small>FL Rule Section 64B16-27.700</small></p>
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	<p>Compounding</p> <p><i>... does <u>not</u> include mixing, reconstituting, or similar acts that are performed in accordance with the directions contained in approved labeling provided by the product's manufacturer and other manufacturer directions consistent with that labeling.</i></p> <p><small>Food and Drug Modernization Act (http://www.fda.gov/cder/fdama/difconc.htm)</small></p>
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	<p>Compounding in the News (2001 – 2003)</p> <p>Walnut Creek Incident: 38 patients exposed to <i>Serratia</i>-contaminated betamethasone, three die. Four patients develop fungal meningitis from interspinal methylprednisolone, one woman dies. Inhalation solutions contaminated with <i>Pseudomonas cepacia</i> are released from a Missouri pharmacy, affecting 12,500 patients Michigan pharmacy issues Class I and II recalls of 900+ vials of contaminated methylprednisolone</p> <p><small>Ockerman AV. <i>US Pharmacist</i>. April 2004; 46-62.</small></p>
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	<p>FDA Survey of Compounded Drug Products</p> <p>Conducted from June to December 2001 29 products from 12 compounding pharmacies Ten (34%) of the 29 samples failed 1 test(s) Nine ophthalmic products, 3 (33%) failed assay/potency tests Two hyaluronidase samples, one failed assay/potency tests</p> <p><small>(http://www.fda.gov/cder/pharmcomp/survey.htm)</small></p>
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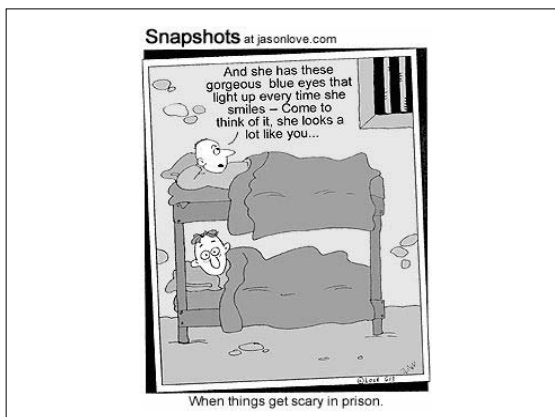
	<p>United States Pharmacopeia (USP) National Formulary (NF)</p> <p>Chapters 2000 apply to nutritional supplements</p> <p>Chapters 1000-1999 are advisory/guidelines</p> <p>Chapters 1-999 are U.S. medication standards and FDA-enforceable</p>
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USP 797: Sterile Preparations
<p>Establishes comprehensive QA standards for compounded sterile preparations (CSPs)</p> <p>Replaced USP <1206></p> <p>Goal – Protect patients</p> <p>Applies to <u>any</u> health care practitioner and practice setting</p> <p>Developed by an expert committee</p>

USP Expert Committee Target
<p style="text-align: right;">USP <797></p> <p>Poor ↓ Excellent</p> <div style="border: 1px solid black; width: 100%; height: 20px; margin: 5px 0;"></div> <p style="text-align: right;">↑ cGMP</p> <p style="font-size: small; text-align: center;">Adopted from <i>Hospital Pharmacy</i>, 2004;39:899-920</p>

Chapter Breakdown
<ul style="list-style-type: none"> Responsibilities of personnel Risk level classification Verification of accuracy/sterility Personnel aseptic training/evaluation Environmental quality and control SOPs Finished preparation release checks and tests Storage and beyond-use dating Patient monitoring and ADE reporting Quality assurance program content Etc... <p style="font-size: x-small; text-align: right;">Adopted from <i>Hospital Pharmacy</i>, 2004;39:899-920</p>

Legal Implications/Enforcement
<ul style="list-style-type: none"> JCAHO AAAHC State Boards FDA



	<p>Protecting yourself while ensuring the safety of your patients</p>
	<p>Don't compound for extended storage.</p> <p><u>Immediate-Use exemption</u></p> <ul style="list-style-type: none"> - Administration within 1 hour - 3 sterile non-hazardous commercial drugs - Not exposed to contact contamination - Requires label if not administered by person who prepared it

	<p>Protecting yourself while ensuring the safety of your patients</p>
	<p>Contract with a USP 797-compliant compounding pharmacy.</p> <ul style="list-style-type: none"> - When "immediate-use" is impractical or inappropriate - Some states require patient-specific prescriptions for CSPs

	<p>Considerations & Precautions</p>
	<p>Internet license search Inspect/Tour the pharmacy (if possible) Request copies of inspection reports Request written USP <797> statement Require copies of QA testing for each CSP Disclose use of CSPs in the informed consent Do not use a compounding pharmacy if commercial alternatives exist</p>

	<p>Take-Home Messages</p>
	<p>USP <797> is far-reaching and FDA-enforceable Review your medication-use process</p> <ul style="list-style-type: none"> - Identify CSPs - Gather information regarding safety - Standardize when possible - Use a Consultant RPh and Manufacturers - Develop and maintain detailed SOPs <p>Immediate-use exemption Do your homework when using a compounding pharmacy Submit comments to USP (www.usp.org)</p>

	<p>Thank you!</p> <p>Q & A</p>

Anesthesia for the Syndromic Child Undergoing Eye Surgery

Jacqueline L. Tutiven MD

Jackson Memorial Hospital / Bascom Palmer Eye Hospital
University of Miami, Miami FL

In large eye hospitals and centers, we come across pediatric patients who can present discreet features of an underlying congenital abnormality to complex syndromes with co-morbid issues. The primary evaluation of importance to the anaesthesiologist should include airway assessment, the presence of a congenital cardiac disease and/or neuromuscular disease. There are common dysmorphic features found in many of these affected children. There are also very discreet signs and symptoms of structural defects in the airway, heart and muscle system that may become apparent when the child is placed under general anaesthesia. This talk reviews important aspects for the peri-operative pediatric anaesthesia assessment in children with syndromes who present for eye evaluations and surgeries.

The Prevalence of children born with inherent genetic diseases and syndromes is relatively high. There are approximately 5 000 syndromic diseases and of these about 10-15% have significant oral maxillary deformities and dysmorphic craniofacial features. Children presenting for surgery and anaesthesia with congenital eye problems also have a variety of concomitant pathologies significantly impacting their perioperative management. The practice of pediatric ophthalmologic surgery requires the anaesthesiologist to interpret the physical dysmorphisms and symptoms found in congenital diseases and understand the musculoskeletal diseases that may impact perioperative care. The main focus of this article is to outline specific anaesthetic concerns when dealing with Syndromic children who present for evaluations under anaesthesia and/or comprehensive eye surgery. It is not this author's intention to categorize and discuss all the syndromes and congenital disorders that exist, but to delineate the important problems and features commonly found that may affect perioperative outcome.

PONV after Eye Surgery: Myths and Facts

Christian C. Apfel MD

UCSF Mount Zion Hospital, San Francisco CA

Educational Objectives

This presentation will provide a comprehensive overview on postoperative nausea and vomiting. After this lecture the clinician should be able to:

1. Understanding limitations and difficulties of risk factors,
2. Assess the patient's risk for PONV by using a simplified risk score,
3. Understand that the patients risk determines the benefit which can be expected from prophylactic therapy,
4. Realize that the effectiveness of a single antiemetic is limited, and
5. How they can apply a risk-dependent approach to prevent PONV.

Who is at Risk for PONV?

Estimating an individual's risk for PONV can indicate who will most likely benefit from prophylactic antiemetic therapy.

In adults, only a few baseline risk factors have consistently been shown to be independent predictors for PONV.(1-16) The most important patient specific predictors are female gender, none-smoking and the history of PONV. Anesthesia-related predictors are a general anesthesia with volatile anesthetics, nitrous oxide, and the use of postoperative opioids. The emetogenic effect of the inhalational anesthetics and opioids appear to be dose related so that longer procedures with concomitant longer anesthesia times and increased postoperative opioid consumptions are associated with an increased incidence of PONV. Many factors which are commonly believed to augment risk are not actually independent factors. These include obesity, anxiety, antagonizing neuromuscular blockage and many types of surgery.(3,4,10,15,17-21)

However, no single patient- or anesthesia-related risk factor is sufficiently sensitive or specific to provide a useful risk assessment.(15) Several risk models have therefore been developed. The simplified models of Apfel et al. and Koivuranta et al. have been shown to have

good predictive characteristics for patients undergoing general inhalational anesthesia in a variety of situations (see table 1).(3,7,8,11,12,22) To this end the simplified score of Apfel et al. considers four main risk factors which are female gender, non-smoking status, history of PONV and the need of i.v. postoperative opioids.(7) When 0, 1, 2, 3, or 4 risk factors are present the patients risk is approximately 10%, 20%, 40%, 60%, or 80%. While the actual incidence might deviate from the predicted risks — for example, during short ambulatory procedures — simplified risk models might allow clinicians to reliably estimate an individual patient's risk of PONV.

In children, a number of papers have been published citing a variety of risk factors associated with the PONV.(23-25) However, evidence supporting these associations was lacking. More recently, a large series of pediatric patients in which a multivariable analysis was applied to identify PONV risk factors in children.(26) Four factors were independent predictors. These factors included duration of surgery ≥ 30 min, age ≥ 3 years, strabismus surgery, and history of POV (postoperative vomiting) or PONV of the relatives (26). Furthermore, they demonstrated that the risk for POV was 9%, 10%, 30%, 55% and 70% when 0, 1, 2, 3, or 4 of those independent predictors were present.

Use of prophylactic antiemetics should be based on valid assessment of the patient's risk for POV or PONV. In other words, antiemetic prophylaxis should be used only when the baseline risk multiplied by the relative risk reduction resulting from prophylaxis produces a clinically meaningful decrease in the risk of PONV. However, more liberal prophylaxis is appropriate in patients where vomiting poses a particular medical risk, including those with wired jaws, increased intracranial pressure, or gastric or esophageal surgery.

What can I do to Prevent PONV?

Because PONV is mainly caused by the emetogenic effects of volatile anesthetics, nitrous oxide and opioids, avoiding these drugs is apparently the most effective approach in patients at high risk for PONV. Examples are the extensive use of local or regional peripheral anesthetic techniques instead of general anesthetic.(27-29) However, if general anesthesia is needed in patients at high risk for PONV avoiding volatile anesthetics and nitrous oxide

together by using a "total intravenous technique" with propofol and air can reduce the risk by about *relative* 30%.(18) This means that if the baseline risk is 80% a TIVA will reduce the risk by 24% ($0.80 \times 0.30 = 0.24$), i.e. in every fourth patient. If however, the baseline risk is only 20% a TIVA will reduce the risk only by 6% ($0.20 \times 0.30 = 0.06$). Thus, it is really the baseline risk which determines whether an antiemetic prevention can lead to a clinically significant decrease of PONV.

However, in very high risk patients a TIVA would still leave about every second patient with PONV ($80\% - 24\% = 56\%$) so that additional antiemetics are needed. The most commonly used antiemetics in the states are the serotonin (5HT₃)-antagonists, i.e. 4 mg ondansetron, 12.5 mg dolasetron or 1 mg granisetron.(18,30,31) Of note, the minimal effective dose for granisetron is probably less than the FDA approved dose of 1 mg, however, 0.1 mg granisetron seem to be ineffective in two dose response studies(32,33) and a recent comparison trying to demonstrate non-inferiority of 0.1 mg granisetron compared to 4 mg ondansetron appears to be underpowered.(34) A new drug on the market is palonosetron which is the only serotonin antagonist on the market which has shown effectiveness and is approved for delayed chemotherapy induced nausea and vomiting(35) and a recent study by White, Scuderi and colleagues has shown it also to be effective for PONV. An even more recent development is the neurokinine (NK₁)-receptor antagonist aprepitant. A study by Gan et al. suggest it to be as effective against nausea as ondansetron but much more effective against vomiting.(ASA abstracts 2005) Palonosetron, aprepitant and the scopolamine patch(36) seem to have extended duration of actions which make these drugs especially useful for postdischarge nausea and vomiting.

A dose of 0.625 or 1.25 mg droperidol has been shown to be equally effective to 4 mg ondansetron.(18,37) Reports of severe cardiac arrhythmias have led the FDA to issue a black box warning which requests to use it only as last option if other drugs have failed and in that case to monitor the ECG for at least 6 hours. Recent investigations have shown that there are minor but short term effects on the QT prolongation which are not larger than those from a general anesthetic itself.(38,39) There are also reports of akathisia or anxiety associated with

low dose of droperidol, however, most studies have not reported these or any other side effects so that this issue remains unclear.(40-42)

A cost effective alternative to a 5-HT₃ antagonist or droperidol is 4 mg dexamethasone. Given at the beginning of the procedure it seems equally effective as 4 mg ondansetron or 1.25 mg droperidol, but without an increased incidence of side-effects.(18) Another alternative is now metoclopramide (Reglan®). While it was already well established that the standard dose of 10 mg is ineffective, (43) a recent dose-response study suggests that 25 mg may be the minimally effective dose and that 50 mg provides a better 24h coverage.(44)

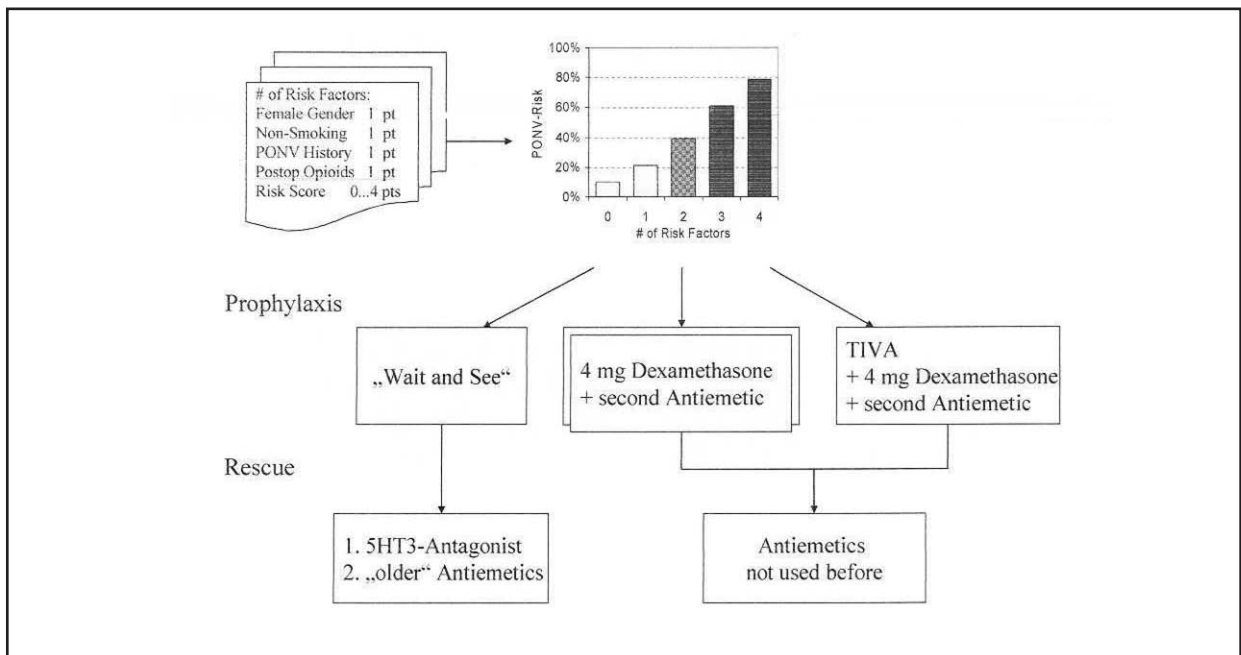
The limited efficacy of a single antiemetic has prompted numerous studies to combine antiemetics. The most successful study was conducted by Scuderi et al. where they were able to eliminate postoperative vomiting before discharge using a multimodal approach.(45) These impressive results triggered an International Multi-center Protocol to Assess the benefits of single and combined antiemetic interventions in a randomized Controlled clinical Trial (IMPACT) to investigate the benefits of ondansetron, droperidol, dexamethasone,

propofol (instead of volatile anesthetics), air (instead of nitrous oxide) and remifentanyl (instead of fentanyl).(46) The unique finding was that a) ondansetron, dexamethasone, droperidol or a TIVA each reduced the incidence by about 25 to 30 relative %, and b) that combining any of these drugs leads to additive effects (not synergistic nor antagonistic).(18) The relative risk reduction for each intervention was apparently independent for a wide range of absolute risks. Thus, interventions that comparably reduce relative risk will produce the greatest absolute risk reduction in patients most likely to experience PONV. A corollary is that the first antiemetic leads to a larger absolute reduction and any following antiemetic leads to a smaller absolute additional effect due to the already reduced risk after the first intervention.(18)

In conclusion, patients at low risk rarely benefit from prophylaxis, patients at moderate risk may benefit from a single antiemetic strategy and patients at high or very high risk should receive two or more prophylactic intervention to prevent PONV.(14)

* Further information are available at www.ponv.org

Fig. 1: Simplified risk score from Apfel et al. to predict the patients risk for PONV.(7) When 0, 1, 2, 3, or 4 of the depicted independent predictors are present; the corresponding risk for PONV is approximately 10%, 20%, 40%, 60% or 80%. The suggested selection of antiemetics could be adjusted to clinical circumstances and needs.(14)



Questions

1. Which statement regarding risk factors for PONV is correct?
 - a) Adult females suffer a higher incidence than do adult males.
 - b) The main causes in obese patients are gastric insufflation via facemask ventilation and increased storage of anesthetic agents in the fatty tissue.
 - c) The body mass index is an independent risk factor.
 - d) Smoking is associated with increased incidence.
 - e) Anxiety is an important risk factor for PONV.
2. Which factor is not considered in the simplified risk score?
 - a) Female gender
 - b) History of PONV
 - c) Postoperative Opioids
 - d) Nitrous oxide
 - e) Non-smoking status
3. Which class of drugs is not thought to cause PONV?
 - a) Muscle relaxants
 - b) Volatile anesthetics
 - c) Opioids
 - d) Induction agents such as thiopentone
 - e) Nitrous oxide
4. Which statement about risk scores is not true?
 - a) Risk scores cannot be universally applicable because they are derived from real data.
 - b) Multiple logistic regression analysis is a method to determine relevant risk factors
 - c) Validations should preferably be done in an independent population.
 - d) A risk score should be based on prospectively collected patient data
5. Which phase of the menstrual cycle is consistently associated with the highest risk for PONV?
 - a) The follicular phase
 - b) The peri-ovulatory phase
 - c) None, since the menstrual phase has no impact on PONV.
 - d) The luteal phase
 - e) The peri-menstrual phase
6. Which statement regarding droperidol is not correct?
 - a) 1.25 mg droperidol prevents PONV as well as 4 mg ondansetron.
 - b) 1.25 mg droperidol might be associated with anxiety and delayed akathisia.
 - c) Droperidol is no longer available in the US because of lethal arrhythmias.
 - d) It prolongs the QT-interval to a similar extent as drugs used during general anesthesia
 - e) Droperidol reduces the relative risk for PONV after a balanced anesthesia similar to a TIVA.
7. Which antiemetic intervention is most effective in preventing PONV?
 - a) A total intravenous anesthetic technique (propofol/air)
 - b) 4 mg i.v. ondansetron given at the end of the case
 - c) 4 mg i.v. ondansetron given at the end of the case
 - d) 1.25 mg droperidol
 - e) All mentioned interventions are similarly effective
8. Which drug is most effective in preventing vomiting?
 - a) Ondansetron
 - b) Dolasetron
 - c) Granisetron
 - d) Aprepitant
 - e) All mentioned interventions are similarly effective
9. Which drug might be favorable for postdischarge nausea and vomiting?
 - a) Scopolamin Patch because of the extended duration of action
 - b) Palonosetron
 - c) Aprepitant
 - d) All three above mentioned drugs.
10. Which statement for a rational risk adapted approach is not correct?
 - a) Patients at low risk rarely benefit from prophylaxis
 - b) Patient at moderate risk may benefit from a single intervention
 - c) Patients at high risk should receive a multimodal approach
 - d) A risk adapted approach is not cost effective as patients at high risk would receive too many antiemetics
 - e) Risk for potential medical complications should also play a role in the decision whether to prophylax or not.

Answers

1a, 2d, 3a, 4a, 5c, 6c, 7e, 8d, 9d, 10d.

Don't hesitate to contact me should you have any remaining questions: apfel@ponv.org

Literature

1. Palazzo M, Evans R. Logistic regression analysis of fixed patient factors for postoperative sickness: a model for risk assessment. *British Journal of Anaesthesia* 1993;70:135-40.
2. Cohen MM, Duncan PG, DeBoer DP, Tweed WA. The postoperative interview: assessing risk factors for nausea and vomiting. *Anesthesia and Analgesia* 1994;78:7-16.
3. Koivuranta M, Laara E, Snare L, Alahuhta S. A survey of postoperative nausea and vomiting. *Anaesthesia* 1997;52:443-9.
4. Apfel CC, Greim CA, Haubitz I et al. A risk score to predict the probability of postoperative vomiting in adults. *Acta Anaesthesiologica Scandinavica* 1998;42:495-501.
5. Apfel CC, Greim CA, Haubitz I et al. The discriminating power of a risk score for postoperative vomiting in adults undergoing various types of surgery. *Acta Anaesthesiologica Scandinavica* 1998;42:502-9.
6. Sinclair DR, Chung F, Mezei G. Can postoperative nausea and vomiting be predicted? *Anesthesiology* 1999;91:109-18.
7. Apfel CC, Laara E, Koivuranta M et al. A simplified risk score for predicting postoperative nausea and vomiting: Conclusions from cross-validations between two centers. *Anesthesiology* 1999;91:693-700.
8. Eberhart LH, Hogel J, Seeling W et al. Evaluation of three risk scores to predict postoperative nausea and vomiting. *Acta Anaesthesiologica Scandinavica* 2000;44:480-8.
9. Apfel CC, Kranke P, Greim CA, Roewer N. What can be expected from risk scores for predicting postoperative nausea and vomiting? *British Journal of Anaesthesia* 2001;86:822-7.
10. Junger A, Hartmann B, Benson M et al. The use of an anesthesia information management system for prediction of antiemetic rescue treatment at the postanesthesia care unit. *Anesthesia and Analgesia* 2001;92:1203-9.
11. Pierre S, Benais H, Pouymayou J. Apfel's simplified score may favourably predict the risk of postoperative nausea and vomiting. *Canadian Journal of Anesthesia* 2002;49:237-42.
12. Apfel CC, Kranke P, Eberhart LHJ et al. A comparison of predicting models for postoperative nausea and vomiting. *British Journal of Anaesthesia* 2002;88:234-40.
13. Stadler M, Bardiau F, Seidel L et al. Difference in risk factors for postoperative nausea and vomiting. *Anesthesiology* 2003;98:46-52.
14. Apfel CC, Roewer N. Risk assessment of postoperative nausea and vomiting. *Int Anesthesiol Clin* 2003;41:13-32.
15. Apfel CC, Kranke P, Eberhart LH. Comparison of surgical site and patient's history with a simplified risk score for the prediction of postoperative nausea and vomiting. *Anaesthesia* 2004;59:1078-82.
16. Pierre S, Corno G, Benais H, Apfel CC. A risk score-dependent antiemetic approach effectively reduces postoperative nausea and vomiting--a continuous quality improvement initiative. *Can J Anaesth* 2004;51:320-5.
17. Hovorka J, Korttila K, Erkola O. Gastric aspiration at the end of anaesthesia does not decrease postoperative nausea and vomiting. *Anaesth Intensive Care* 1990;18:58-61.
18. Apfel CC, Korttila K, Abdalla M et al. A factorial trial of six interventions for the prevention of postoperative nausea and vomiting. *New England Journal of Medicine* 2004;350:2441-51.
19. Kranke P, Apfel CC, Papenfuss T et al. An increased body mass index is no risk factor for postoperative nausea and vomiting. A systematic review and results of original data. *Acta Anaesthesiologica Scandinavica* 2001;45:160-6.
20. Van den Bosch JE, Moons KG, Bonseel GJ, Kalkman CJ. Does measurement of preoperative anxiety have added value for predicting postoperative nausea and vomiting? *Anesth Analg* 2005;100:1525-32.
21. Cheng CR, Sessler DI, Apfel CC. Does neostigmine administration produce a clinically important increase in postoperative nausea and vomiting? *Anesth Analg* 2005;101:1349-55.
22. van den Bosch JE, Kalkman CJ, Vergouwe Y et al. Assessing the applicability of scoring systems for predicting postoperative nausea and vomiting. *Anaesthesia* 2005;60:323-31.
23. Rowley MP, Brown TC. Postoperative vomiting in children. *Anaesth Intensive Care* 1982;10:309-13.
24. Sossai R, Johr M, Kistler W et al. Postoperative vomiting in children. A persisting unsolved problem. 1993;3:206-8.
25. Rose JB, Watcha MF. Postoperative nausea and vomiting in paediatric patients. *British Journal of Anaesthesia* 1999;83:104-17.
26. Eberhart LH, Geldner G, Kranke P et al. The development and validation of a risk score to predict the probability of postoperative vomiting in pediatric patients. *Anesth Analg* 2004;99:1630-7.
27. Pusch F, Freitag H, Weinstabl C et al. Single-injection paravertebral block compared to general anaesthesia in breast surgery. *Acta Anaesthesiologica Scandinavica* 1999;43:770-4.
28. Song D, Greilich N, Tongier K et al. Recovery profiles of outpatients undergoing unilateral inguinal herniorrhaphy: a comparison of three anesthetic techniques. *Anesthesia and Analgesia* 1999;88:S30.
29. Friedberg BL. Propofol-ketamine technique: dissociative anesthesia for office surgery (a 5-year review of 1264 cases). *Journal of Clinical Anesthesia* 1999;23:70-5.
30. Graczyk SG, McKenzie R, Kallar S et al. Intravenous dolasetron for the prevention of postoperative nausea and vomiting after outpatient laparoscopic gynecologic surgery. *Anesthesia and Analgesia* 1997;84:325-30.
31. Kranke P, Apfel CC, Eberhart LH et al. The influence of a dominating centre on a quantitative systematic review of granisetron for preventing postoperative nausea and vomiting. *Acta Anaesthesiol Scand* 2001;45:659-70.
32. Wilson AJ, Diemunsch P, Lindeque BG et al. Single-dose i.v. granisetron in the prevention of postoperative nausea and vomiting. *British Journal of Anaesthesia* 1996;76:515-8.
33. Mikawa K, Takao Y, Nishina K et al. Optimal dose of granisetron for prophylaxis against postoperative emesis after gynecological surgery. *Anesthesia and Analgesia* 1997;85:652-6.

34. Gan TJ, Coop A, Philip BK. A randomized, double-blind study of granisetron plus dexamethasone versus ondansetron plus dexamethasone to prevent postoperative nausea and vomiting in patients undergoing abdominal hysterectomy. *Anesth Analg* 2005;101:1323-9.
35. Siddiqui MA, Scott LJ. Palonosetron. *Drugs* 2004;64:1125-32; discussion 33-4.
36. Kranke P, Morin AM, Roewer N et al. The efficacy and safety of transdermal scopolamine for the prevention of postoperative nausea and vomiting: a quantitative systematic review. *Anesth Analg* 2002;95:133-43.
37. Fortney JT, Gan TJ, Graczyk S et al. A comparison of the efficacy, safety, and patient satisfaction of ondansetron versus droperidol as antiemetics for elective outpatient surgical procedures. S3A-409 and S3A-410 Study Groups. *Anesthesia and Analgesia* 1998;86:731-8.
38. Charbit B, Albaladejo P, Funck-Brentano C et al. Prolongation of QTc interval after postoperative nausea and vomiting treatment by droperidol or ondansetron. *Anesthesiology* 2005;102:1094-100.
39. White PF, Song D, Abrao J et al. Effect of low-dose droperidol on the QT interval during and after general anesthesia: a placebo-controlled study. *Anesthesiology* 2005;102:1101-5.
40. Melnick B, Sawyer R, Karambelkar D et al. Delayed side effects of droperidol after ambulatory general anesthesia. *Anesth Analg* 1989;69:748-51.
41. Foster PN, Stickle BR, Laurence AS. Akathisia following low-dose droperidol for antiemesis in day-case patients. *Anaesthesia* 1996;51:491-4.
42. Lim BS, Pavy TJ, Lumsden G. The antiemetic and dysphoric effects of droperidol in the day surgery patient. *Anaesth Intensive Care* 1999;27:371-4.
43. Henzi I, Walder B, Tramer MR. Metoclopramide in the prevention of postoperative nausea and vomiting: a quantitative systematic review of randomized, placebo-controlled studies. *British Journal of Anaesthesia* 1999;83:761-71.
44. Wallenborn J, Gelbrich G, Bulst D et al. Prevention of postoperative nausea and vomiting by metoclopramide combined with dexamethasone: randomised double blind multicentre trial. *Bmj* 2006.
45. Scuderi PE, James RL, Harris L, Mims GR, III. Multimodal antiemetic management prevents early postoperative vomiting after outpatient laparoscopy. *Anesthesia and Analgesia* 2000;91:1408-14.
46. Apfel CC, Korttila K, Abdalla M et al. An international multicenter protocol to assess the single and combined benefits of antiemetic interventions in a controlled clinical trial of a 2x2x2x2x2 factorial design (IMPACT). *Control Clin Trials* 2003;24:736-51.

Intraoperative Control of Intraocular Pressure

Catherine Meschler MD

The Johns Hopkins University, Baltimore MD

The anesthesiologist in the operating room has several tools at his disposal to alter intraocular pressure. This is especially important in cases that involve an open globe, such as intracapsular cataract extractions and drainage procedures. Maintaining a low normal pressure prior to incision can help prevent the extrusion of contents. Lower IOP can also help the surgeon and provide for a more efficient operation. Intraocular pressure is determined by the balance of the forces within the eye pushing outward and extraocular muscle tone and scleral compliance pushing inward. The forces within the eye include aqueous humor (AH), vitreous humor, and blood volume and each of these can be manipulated perioperatively. The preoperative use of the carbonic anhydrase inhibitor acetazolamide inhibits the production of AH while reverse trendelenberg position and manual compression of the orbit increase AH drainage. Many factors decrease intraocular pressure via their affect on choroidal blood volume (CBV), the most prominent being hyperventilation to decrease the PaCO₂ levels of blood causing vasoconstriction. Of less clinical importance hyperbaric oxygen, metabolic acidosis, and profound hypotension decrease IOP by their influence on CBV. Hvidberg studied sixteen patients undergoing laparoscopic surgery and concluded that maintaining reverse trendelenberg of 15 degrees and hyperventilating to a PaCO₂ of 25-30 were both effective methods of decreasing IOP. Due to autoregulation, changes in blood pressure within normal physiological range have little effect on IOP, but below MAP's of 85 arterial hypotension decreases choroidal blood volume and IOP. Classically hyperosmotic agents were thought to decrease IOP by dehydrating the vitreous humor of the eye. While this is still true, recent experiments by Mauger and others postulate a second central mechanism via osmoreceptors of the hypothalamus. One of the commonly

used hyperosmotic agents is mannitol. While the most effective dose of mannitol is not known doses from 1-2g/kg appear most frequently in the literature; Mauger has demonstrated a single dose as low as 12.5g may be beneficial. General anesthetics with the exception of ketamine all decrease IOP by several mechanisms including their influence via diencephalon centers of the brain which have been shown to centrally mediate IOP. Muscle relaxants with the exception of succinylcholine have little effect to a slight decrease on IOP. Succinylcholine consistently raises IOP although interestingly giving a full dose of this drug raises IOP less than giving a half dose. When clinically appropriate, the LMA has been shown to raise IOP less than an ETT. Obviously any prevention that can be taken to prevent vomiting, coughing, or straining will prevent rises in IOP. Which of these methods we use to decrease the pressure is determined by the clinical picture of both the patients' comorbidities and the type and timing of the surgery.

Selected References:

- Cunningham AJ, Barry P. Intraocular pressure—physiology and implications for anaesthetic management. *Can Anaesth Soc J* 1986; 33(2):195-208.
- Hvidberg A, Kessing SV, Fernandes A. Effect of changes in PCO₂ and body positions on intraocular pressure during general anesthesia. *Acta Ophthalmologica* 1981;59:465-475.
- Mauger TF, Nye CN, Boyle KA. Intraocular pressure, anterior chamber depth, and axial length following intravenous mannitol. *Journal of Ocular Pharmacology and Therapeutics* 2000;16:591-594.
- Murphy DF. Anesthesia and intraocular pressure. *Anesth Analg* 1985;64(5):520-530.

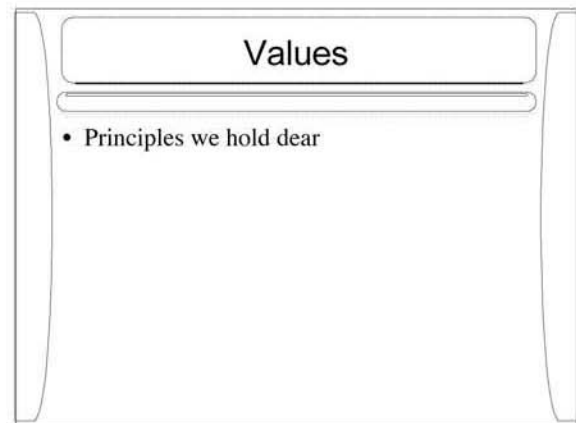
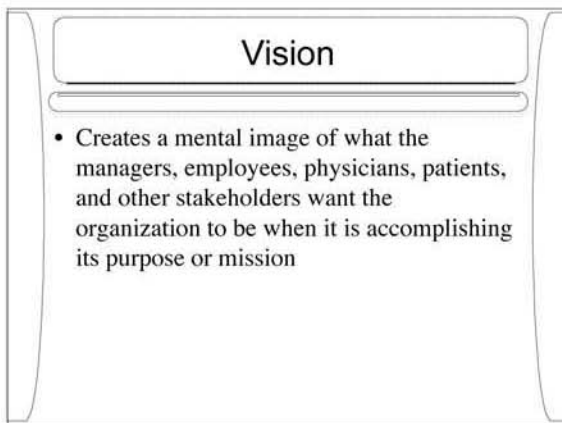
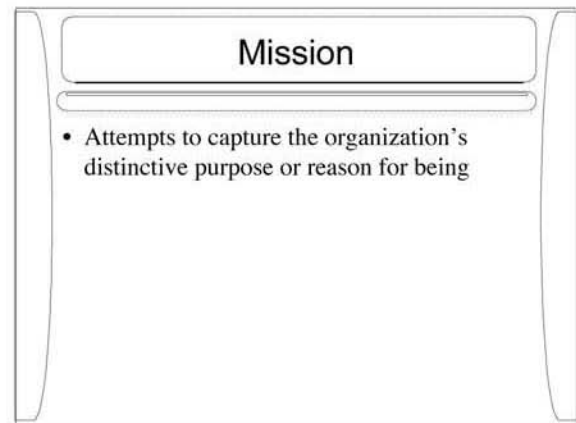
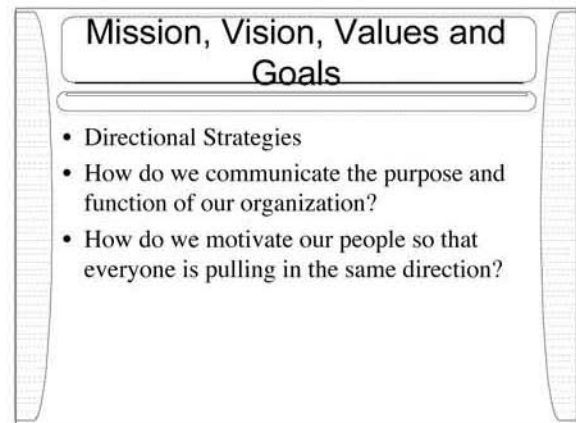
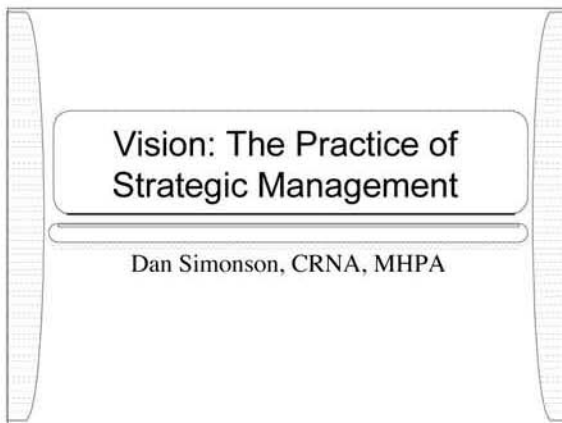
An Administrator's Perspective of a Successful Surgery Center

Dan Simonson CRNA MHPA

Spokane Eye Surgery Center, Spokane WA

Objective: Given a 50 minute presentation, the participants will be able to define the terms mission, vision, goals, and values as they relate to running ambulatory surgery centers.

Abstract: Mission, Vision, Goals, and Values are the four components of Strategic Management that are termed "Directional Strategies." They guide leaders of organizations when they make key organizational decisions. Many of us in health care are familiar with the terms, but not the body of knowledge that exists to help us understand, create, and utilize these important concepts. This lecture will review the history of these concepts and show, through concrete examples, how management teams can use them as they participate in the strategic planning for ambulatory surgery centers.



Goals

- Concrete objectives to be accomplished by the organization when adhering to their Mission, Vision, and Values

Examples

Strategic Management for Ambulatory Surgery Centers

- Mission:
 - Excellence
 - Safety
 - Quality

Strategic Management for Ambulatory Surgery Centers

- Vision:
 - What would an excellent surgery center look like?
 - What defines quality for our surgery center?

Resources

- **Ginter, P. M., Swayne, L. E., & Duncan, W. J. (2002). Strategic Management of Health Care Organizations. Malden, MA: Blackwell Publishers.**

Are There Any Complications of Sub-Tenon's Blocks?

Chandra Kumar MBBS DA FFARCSI FRCA MSc

The James Cook University, Hospital and University of Teesside, Middlesbrough, UK

Sub-Tenon's was re-introduced into the clinical practice as a simple, effective and safe technique alternative to commonly used needle blocks ¹. In this technique, the local anaesthetic agent is delivered under the Tenon's capsule through a blunt cannula thus avoiding the dangers of a sharp needle. As there were no reported complications, it was assumed that this technique is very safe then. As the technique started to be used more widely, complications were published in the literature.

Although the nature of complications is very similar to needle blocks but the incidence and severity are comparatively less. Unfortunately at present we do not have comparative data to predict the incidence of complications and relative safety of all orbital blocks. However, there are a few personal series and audit studies which suggest that sub-Tenon's block is a relatively safe technique. Major complications of sub-Tenon's block have all appeared as case reports.

Complications arising from sub-Tenon's block may be limited to the orbit and its contents or may manifest systemically ^{2,3,4}. Some complications arise immediately while others are delayed. Some complications are minor while others are life and sight threatening which may be directly related to the technique of block administration, local anaesthetic agent and adjuvant if used. Other medical adverse events unrelated to block are also known to occur.

Pain during injection

Minor to moderate pain during injection is reported in approximately 46% of patients ⁵. The severity of pain is usually of VAS <3 but some patients complain higher pain score and prediction is very difficult.

All injectable local anaesthetic agents produce mild sting or burning sensations on injection and this may be interpreted as pain especially around orbit. Some believe it is introduction of

cannula through the potential space into the posterior sub-Tenon's space; a feeling of pressure during injection, widening and stretching of the potential space (sub-Tenon) may cause pain.

Pain can not be completely abolished but severity can be reduced by gentle insertion of the cannula, slow injection of warm local anaesthetic agent and reassurance ³.

Chemosis

Chemosis is the swelling of conjunctiva and this occurs due to anterior spread of the local anaesthetic agent after injection. Mild to severe chemosis occurs after sub-Tenon's block and the incidence varies between 25 to 100% depending on the length of the sub-Tenon's cannulae used ^{5,6}.

Chemosis is unavoidable is more likely to occur if the dissection of Tenon's capsule is not adequate or higher volume of local anaesthetic is injected. This is usually limited to the site of injection but may spread to other quadrants of the globe.

Presence of chemosis usually does not interfere with cataract surgery but some glaucoma surgeons may not be satisfied.

Simple measures such as gentle pressure on the globe limits its spread or reduces the swelling.

Subconjunctival haemorrhage

Red looking eye is a common occurrence following sub-Tenon's block. Redness may be due to handling of the conjunctiva resulting in hyperaemia or it may be real subconjunctival haemorrhage.

Subconjunctival haemorrhage is evitable as small blood vessels are severed during blunt dissection. The incidence of redness varies from 20-100% depending on the length of cannula used ^{1,6}. Also the assessment of conjunctival haemorrhage is subjective therefore leads to under or over scoring. An objective method using comparison of photographs has been advocated ⁷. The haemorrhage may be limited to the area of dissection or spread to other quadrants. The incidence of conjunctival haemorrhage is higher in patients receiving anticoagulant, aspirin and clopidogrel ⁸.

Unsubstantiated concerns are raised that subconjunctival haemorrhage may compromise the outcome of glaucoma surgery⁹.

Redness or subconjunctival haemorrhage can be minimised by careful dissection thus avoiding damage to fine vessels. Although not proven but epinephrine containing local anaesthetic agent or application cotton bud soaked with vasoconstrictor containing solutions are supposed to reduce the incidence of subconjunctival haemorrhage^{3, 4, 10}. Ophthalmologists can reduce the incidence of conjunctival haemorrhage by applying diathermy using operating microscope^{11, 12} but no such benefit was obtained when disposable diathermy was used by anaesthesia personnel¹³. Application of gentle pressure on the globe may limit the spread of haemorrhage. Patients should be informed that the eye may look red in the immediate postoperative period³.

Akinesia and eye lid movements

Rectus muscle and eyelid movements are reduced following sub-Tenon's block but they variable and unpredictable^{3, 4, 6}.

Akinesia is volume dependent and if 4-5mls local anaesthetic is injected, a large proportion of patients develop akinesia⁶. Superior oblique muscle and lid movements may remain active in significant number of patients.

As akinesia is not essential for modern phacoemulsification surgery and surgery is performed without significant problems. However residual rectus muscle movements may not provide good operating conditions for certain procedures.

Sight and life threatening complications have been described as case reports. **Orbital and retrobulbar haemorrhage** due to trauma to the blood vessels^{14, 15, 16}, **rectus muscle paresis & damage** probably due to direct trauma by the blunt cannula (ptosis and diplopia)¹⁷⁻²¹ and **orbital swelling** resulting from inflammation, allergy or excessive growth of orbital tissue have been reported²²⁻²⁷. Serious **life threatening complications** such as **central spread of local anaesthetic**²⁸ and unpublished **report of a death**²⁹ have also occurred. **Sight threatening complications** such as **globe perforation**³⁰, **retinal and**

choroidal and vascular occlusion³¹, **optic nerve problems** (dilated pupils, loss of accommodation, and optic neuropathy)³²⁻³⁴, **conjunctival inclusion cyst**³⁵, **intractable glaucoma**³⁶ and **cutaneous hypopigmentation**³⁷ have been reported.

Above described complications appear to be related to a faulty technique and perhaps deep insertion of long posterior sub-Tenon's cannula³⁸ into the posterior part of the sub-Tenon's space.

Careful dissection and slow introduction of posterior cannula without force is advocated^{3, 38}. If there is any resistance met during the insertion of a cannula, it should be reintroduced and repositioned⁴. The use of smaller & flexible cannulae may offer benefits but the incidence of chemosis and conjunctival haemorrhage increases³⁹.

Complications related to local anaesthetic agent

Topical local anaesthetic agent

All local anaesthetic eye drops produce sting on application but tetracaine appears to produce more stings⁴⁰. Some authorities are concerned that a significant increase in corneal thickness and opacification can result if local anaesthetics enter the anterior chamber of the eye.

Injectable local anaesthetic agent

Intravascular injection

The toxicity may be associated with local anaesthetic absorption, intravascular injection, allergic reaction or vasovagal attack. These complications have been reported after needle block but fortunately no such complication has occurred following sub-Tenon's block. Sub-Tenon cannula is blunt hence it is unlikely to enter into a major blood vessel of the orbit.

Utilization of a minimum effective dose, volume, concentration, aspiration before injection and slow injection in fractional amounts while maintaining verbal contact with the patients for reporting of possible toxic symptoms is a good clinical and safe practice.

Allergic reaction

Hypersensitivity reaction to amide local anaes-

thetic agent is rare. A history of previous exposure should be sought.

Adjuvant

Epinephrine

Admixture with epinephrine is commonly used to prolong the block and reduce absorption of local anaesthetic agent. A concentration (1:200,000) has no systemic effects⁴⁰.

No unwanted effects have been reported during sub-Tenon's block.

Epinephrine containing solution is generally avoided during orbital blocks.

Hyaluronidase

Hyaluronidase is used to improve onset, effectiveness and quality of sub-Tenon's block⁴¹ but good anaesthesia and akinesia is possible without it⁴².

The amount of hyaluronidase used during ophthalmic regional anaesthesia varies from 1-150 IU.ml⁻¹. British National Formulary^{43,44} recommends limiting the concentration of hyaluronidase to 15 IU.ml⁻¹.

Orbital pseudotumour⁴⁵ and orbital swelling after high dose hyaluronidase have been reported⁴⁶. Rare allergic reaction to hyaluronidase has been described during sub-Tenon's block⁴⁷.

There is no evidence of muscle dysfunction if hyaluronidase is omitted.

Pulsatile ocular blood flow during block

Ocular blood flow is known to decrease after all orbital blocks⁴⁸.

Complications related to sedation

Although sedation is common during topical anaesthesia but in selected patients, in whom explanation and reassurance have proved inadequate, may benefit from sedation during other techniques.

Routine use of sedation for orbital block is discouraged⁴⁹ because of increased intra-operative events^{50,51}.

It is essential that when sedation is administered, a means of providing supplementation oxygen, equipment and skills to manage any life-threatening events must be immediately accessible⁴⁹.

Other adverse medical events

A large prospective audit involving 6000 patients conducted in Auckland⁵² suffered no serious complication related to sub-Tenon's block but some patients suffered from cardiovascular complications unrelated to block.

Conclusion

Minor and major complications including life and sight threatening have occurred following sub-Tenon's block. The exact incidence of these complications is not known. At present we do not have an absolute safe technique of orbital block. Sound knowledge of orbital anatomy, ophthalmic physiology and pharmacology of the anaesthesia and ophthalmic drugs are prerequisites for performing orbital regional anaesthesia.

References

- 1 Roman SJ, Chong Sit DA, Boureau CM, Auclin FX, Ullern MM. Sub-Tenon's anaesthesia: an efficient and safe technique. *Br J Ophthalmol* 1997; 81: 673-6.
- 2 Kumar CM, Dowd TC. Complications of ophthalmic regional blocks: their treatment and prevention. *Ophthalmologica* 2006; 220: 3-82.
- 3 Kumar CM, Manickam B, Williamson S. A review of sub-Tenon's block: current practice and recent development. *Eur J Anaesthesiol* 2005; 22: 567-77.
- 4 Kumar CM, Dodds C. Sub-Tenon's Anesthesia. *Ophthalmol Clin North Am* 2006 Jun;19(2):209-19.
- 5 Stevens JD. A new local anesthesia technique for cataract extraction by one quadrant sub-Tenon's infiltration. *Br J Ophthalmol* 1992; 76: 670-4.
- 6 Kumar CM, Dodds C. Evaluation of Greenbaum sub-Tenon's block. *British J Anaesth* 2001;87: 631-3.
- 7 Kumar CM, Dowd TC, Adams WE, Puckering S. Methodology of evaluating conjunctival appearance following sub-Tenon's block for phacoemulsification cataract surgery. *Eye* 2005 (ahead of pubmed)
- 8 Kumar N, Jivan S, Thomas P, McLure H, Sub-Tenon's anesthesia with aspirin, warfarin, and chlopidogrel. *J Cataract Refract Surgery* 2006; 32: 1022-25.
- 9 Eke T. Anesthesia for glaucoma surgery. *Ophthalmol Clin North Am* 2006; 19: 245-55.

- 10 Chung RS Chua CN. Reduction of subconjunctival hemorrhage with sub-Tenon's anesthesia. *J Cataract Refract Surg* 2005; 31: 2031.
- 11 Greenbaum S. Parabolbar anesthesia. *Am J Ophthalmol* 1992; 114: 776.
- 12 Gauba V, Saleh GM, Watson K, Chung A. Sub-Tenon anaesthesia: reduction in subconjunctival haemorrhage with controlled bipolar conjunctival cautery. *Eye* 2006; 2006 Jun 2; [Epub ahead of print].
- 13 Kumar CM, Williamson S. Diathermy does not reduce subconjunctival haemorrhage during sub-Tenon's block. *Br J Anaesth* 2005; 95: 562.
- 14 Olitsky SE, Juneja RG. Orbital haemorrhage after the administration of sub-Tenon's infusion anaesthesia. *Ophthalmic Surg Lasers* 1997; 28: 145-6.
- 15 Rahman I, Ataullah S. Retrobulbar hemorrhage after sub-Tenon's anesthesia. *J Cataract Refract Surg* 2004; 30: 2636-7.
- 16 Dareau S, Gros T, Bassoul B, Causse L, Eledjam JJ. Orbital haemorrhage after medial canthus episclera (sub-Tenon's anaesthesia) *Ann Fr Anesth Reanim* 2003; 2: 474-6.
- 17 Spierer A, Schwalb E. Superior oblique muscle paresis after sub-Tenon's anesthesia for cataract surgery. *J Cataract Refract Surg* 1999; 25: 144-5.
- 18 Jaycock PD, Mather CM, Ferris JD, Kirkpatrick JNP. Rectus muscle trauma complicating sub-Tenon's local anaesthesia. *Eye* 2001; 15: 583-586.
- 19 Adams W, Morgan SJ. Diplopia following sub-Tenon's infiltration of local anaesthesia *J Cataract Refract Surg* 2002; 28: 1694-7.
- 20 Dal Canto AJ, Downs-Kelly E, Perry D. Ptosis and orbital fat prolapse after posterior sub-Tenon's capsule triamcinolone injection. *Ophthalmology* 2005; 112: 1092-7.
- 21 Merino P, Munoz-Sanz N, Gomez-de-Liano P, Gutierrez-Partida B, Seijas-Leal O. Diplopia after sub-Tenon's anesthesia for cataract surgery. *Arch Soc Esp Oftalmol* 2006; 81: 141-6.
- 22 Redmill B, Sandy C, Rose GE. Orbital cellulitis following corneal gluing under sub-Tenon's local anaesthesia. *Eye* 2001; 15: 554-6.
- 23 Dahlmann AH, Appaswamy S, Headon MP. Orbital cellulites following sub-Tenon's anaesthesia. *Eye* 2002; 16: 200-1.
- 24 Morgan SJ. Orbital cellulitis following corneal gluing under sub-Tenon's local anaesthesia. *Eye* 2003; 17: 284-5.
- 25 Muqit MM, Saidkasimova S, Gavin M. Acute orbital cellulites after sub-Tenon's eye block. *Anaesthesia* 2004; 59: 11-3.
- 26 Mukherji S, Esakowitz L. Orbital inflammation after sub-Tenon's anesthesia. *J Cataract Refract Surg* 2005; 31: 2221-3.
- 27 Quhill F, Bowling B, Packard RB. Hyaluronidase allergy after peribulbar anesthesia with orbital inflammation. *J Cataract Refract Surg* 2004; 30: 916-7.
- 28 Ruschen H, Bremner FD, Carr C. Complications after sub-Tenon's eye block. *Anesth Analg* 2003; 96: 273-7.
- 29 Personal communication (Anaesthesia Department, Eastbourne NHS Trust Hospital, UK).
- 30 Frieman BJ, Friedberg MA. Globe perforation associated with subtenon's anesthesia. *Am J Ophthalmol* 2001; 131:520-1.
- 31 Moshfeghi DM, Lowder CY, Roth DB, Kaiser PK. Retinal and choroidal vascular occlusion after posterior sub-tenon triamcinolone injection. *Am J Ophthalmol* 2002; 134: 132-4.
- 32 Ramsay AS, Ray-Chaudhuri N, Dayan M, Walshaw D. Quantification of relative afferent pupillary defects induced by posterior sub-Tenon's, peribulbar, and retrobulbar anaesthetics. *Br J Ophthalmol*.2001; 85: 1445-6.
- 33 Patel JI, Jenkins L, Benjamin L, Webber S. Dilated pupils and loss of accommodation following diode panretinal photocoagulation with sub-tenon local anaesthetic in four cases. *Eye* 2002; 16: 628-32.
- 34 Kim SK, Andreoli CM, Rizzo JF 3rd, Golden MA, Bradbury MJ. Optic neuropathy secondary to sub-tenon anesthetic injection in cataract surgery. *Arch Ophthalmol* 2003; 121:907-9.
- 35 Vishwanath MR, Jain A. Conjunctival inclusion cyst following sub-Tenon's local anaesthetic injection. *Br J Anaesth* 2005; 95: 825-6.
- 36 Huang SY, Tsai YY, Lin JM, Hung PT. Intractable glaucoma following posterior sub-tenon's triamcinolone acetamide for central retinal vein occlusion in a young adult. *Eye* 2006 [Epub ahead of print]
- 37 Gallardo MJ, Johnson DA. Cutaneous hypopigmentation following a posterior sub-tenon triamcinolone injection. *Am J Ophthalmol*.2004; 137: 779-80.
- 38 Greenbaum S. Orbital inflammation after posterior sub-Tenon's anesthesia. *J Cataract Refract Surg* 2006; 32: 1246-7.
- 39 Kumar CM, Dodds C, McLure H, Chabria R. A comparison of three sub-Tenon's cannulae. *Eye* 2004; 18: 873-6.
- 40 McLure HA, Rubin AP. Review of local anaesthetic agents. *Minerva Anesthesiol* 2005; 71: 59-74.
- 41 Guise P, Laurent S. Sub-tenon's block: The effect of hyaluronidase on speed of onset and block quality. *Anaesthesia & Intensive Care* 1999; 27: 179-81.
- 42 Radhakrishna, F. Shekawat and G. Furlong. Sub-Tenon's block without hyaluronidase. *Anaesthesia* 2004; 59: 411.
- 43 British National Formulary. A joint publication of the British Medical Association and the Royal Pharmaceutical Society of Great Britain, London, 2002.
- 44 Fanning GL. Hyaluronidase in ophthalmic anesthesia. *Anesth Analg* 2000; 91: 934-7.
- 45 Kempeneers A, Dralands L, Ceuppens J. Hyaluronidase induced orbital pseudotumour as a complication of retrobulbar anesthesia. *Bull Soc Belge Ophthalmol* 1992; 243: 159-166.
- 46 Kumar CM, Dowd TC, Dodds C, Boyce R. Orbital swelling following peribulbar and sub-Tenon's anaesthesia. *Eye* 2004; 18: 418-20.
- 47 Musa F, Srinivasan S, King CM, Kamal A. Raised intraocular pressure and orbital inflammation: a rare IgE-mediated allergic reaction to sub-Tenon's hayluronidase. *J Cataract Refract Surg* 2006; 32: 77-8.

48 Pianka P, Weintraub-Padova H, Lazar M, Geyer O. Effect of sub-Tenon's and peribulbar anesthesia on intraocular pressure and ocular pulse amplitude. *J Cataract and Refractive Surgery* 2001; 27: 1221-6.

49 Local Anaesthesia for Intraocular Surgery: The Royal College of Anaesthetists and The Royal College of Ophthalmologists 2001.

50 Katz J, Feldman MA, Bass EB, Lubomski LH, Tielsch JM, Petty BG, Fleisher LA, Schein OD. Adverse intraoperative medical events and their association with anesthesia management strategies in cataract surgery. *Ophthalmology* 2001; 108: 1721-6.

51 Haberer JP. Premedication and sedation complications during ophthalmic anaesthesia. *J Fr Ophtalmol* 2000; 23:901-906.

52 Guise PA. Sub-Tenon anesthesia: a prospective study of 6,000 blocks. *Anesthesiology* 2003; 98: 964-8.

WORKSHOP ABSTRACTS

Workshop A

(Repeated in Second Session)

Parallel Approach to Orbital Blocks: Let's Track the Needle Tip

Randolf R. Harvey BS CRNA

Florida Eye Clinic / ASC, Altamonte Springs FL

Since Dr. Atchinson described the retrobulbar block, orbital regional block techniques have continued to undergo refinements that have led to improved patient safety and comfort. The technique of utilizing a parallel approach to orbital blocks has been around for more than twenty years. Gills and Loyd described the technique in, AM Intra-Ocular Implant Soc. J-VOL 9, summer 1983, titled: "A Technique of Retrobulbar Block with Paralysis of Orbicularis Oculi."

Directing the needle tip away from the vital orbital structures is this techniques primary value. Secondly, the needle is inserted through the conjunctiva avoiding a skin puncture and reducing the potential for lid ecchymosis.

The needle, bevel up, is inserted infero-temporally, above the inferior orbital rim, approximately 3 to 5mm lateral to the lateral limbic margin of the globe, through the conjunctiva. The needle travels posteriorly, inferior to the globe. After passing the equatorial plane of the globe, the needle is angled cephalad and advanced into the intra-conal compartment of the mid-orbit to a depth of approximately 25mm, approximately 5mm posterior to the globe.

The needle remains parallel to the visual axis and lateral to the lateral limbic margin throughout the technique. Therein lays the difference from other needle based techniques, which have some degree of medial needle direction.

Anatomically, the needle tip rests in an area that has been described as a safe zone, relatively devoid of vital orbital structures. However, the eye should not look medial because it may place the optic nerve in line with the needle tip. In addition a retrobulbar hemorrhage can still occur if the orbital veins in this area are traumatized. The general proximity of vital orbital structures in relation to the pathway of the needle tip is illustrated below:

1. Structures **MEDIAL** to the needle tip pathway

<u>Nerves</u>	<u>Muscles</u>
CN II Optic	Superior Rectus
CN III Oculomotor	Inferior Rectus
CN IV Trochlear	Medial Rectus
CN V Trigeminal	Superior Oblique
Ciliary Ganglion/Nerves	Inferior Oblique
<u>Vasculature</u>	
Ophthalmic Artery	Superior Ophthalmic Vein
Central Retinal Artery	Central Retinal Vein
Ciliary Arteries	Venous Vortex Veins
2. Structures **LATERAL** to the needle tip pathway

<u>Nerves</u>	<u>Muscles</u>
CN VI Abducens	Lateral Rectus
3. Structures **SUPERIOR** to the needle tip pathway

<u>Nerves</u>	<u>Vasculature</u>
CN V Trigeminal/Lacrimal	Lacrimal Artery
	Lacrimal Vein
	Superior Ophthalmic vein
4. **Globe's relation to the needle tip pathway:**

Superior: The globe is superior to the needle tip, from its insertion point, until after the needle tip passes the equatorial plane of the globe and begins its rotation cephalad.

Posterior: The needle tip becomes posterior to the globe after passing the equatorial plane of the globe.

Medial: The posterior pole of the globe (macula) remains medial to the needle tip throughout the procedure. Along with the area inferior to the macula, where, posterior staphylomas may form.

As practitioners, we understand there is no anesthetic technique that is 100% safe. However, the parallel approach to orbital blocks incorporates a sound anatomical and technically safe approach for our needles to enter the mid-orbit for the administration of local anesthesia.

Objectives:

- (1) Review the appropriate orbital anatomy
- (2) Describe the needle pathway into the mid-orbit
- (3) Evaluate the position of the orbital structures in relation to the needle tip

Workshop B

(Repeated in Second Session)

Anatomy for Orbital Regional Anesthesia

Gary L. Fanning MD

Hauser-Ross Eye Institute, Sycamore IL

Orbital regional anesthesia (so-called retrobulbar and peribulbar blocks) has long been used to provide excellent anesthesia and akinesia for surgery of the eye. As there are major complications associated with orbital blocks, more recently other techniques, namely sub-Tenon's anesthesia and topical anesthesia, have been promoted as safer methods of anesthesia, especially for patients undergoing cataract surgery. As not all patients, procedures, and/or surgeons are suited to these newer techniques, orbital blocks will continue to be used. The purpose of this presentation is to examine the anatomy of the orbit vis-à-vis orbital regional anesthesia in order to determine if the safety of the procedure can be enhanced.

For many years a rather standard version of orbital regional anesthesia has been described and taught. In a publication as recent as April 2005 this classic description of an orbital block appears in an excellent paper by Lai et al¹: "A retrobulbar injection was performed by inserting an Atkinson needle through the lower eyelid at the junction of the lateral and middle thirds of the inferior orbital rim parallel to the orbital floor, first to a depth of 25mm, then angled up and medially and advanced toward the apex to the hub of the needle." This classic description includes what I like to call the three cardinal sins of orbital regional anesthesia: 1) the needle is too long, 2) the needle is inserted at the wrong point, and 3) the needle is aimed in the wrong direction. The remainder of this presentation will provide anatomical reasons why I consider these to be cardinal sins and will suggest alternative techniques.

Nomenclature: As I have previously opined², the terms "retrobulbar" and "peribulbar," commonly used to describe two different forms of orbital regional anesthesia, are anatomically

inadequate. In fact, the term "retrobulbar" simply means behind the eye. Virtually all orbital blocks involve putting a needle behind the eye. The term "peribulbar" means around the eye. All orbital blocks had better be around the eye as the only alternative is through the eye, something we try very hard to avoid. Although meant to describe the anatomical location of the needle tip during the block, neither of these terms is precise enough to be acceptable, in my opinion. When we refer to a "retrobulbar block," we really mean to denote a block in which the needle tip lies within the muscle cone. It would be preferable, therefore, to simply call it an "intraconal block." Likewise, the term "peribulbar block" is meant to denote a block in which the needle tip is outside of the muscle cone. Again, it makes more sense to call it an "extraconal block." Both forms of blocking are acceptable as local anesthetic can easily diffuse from one compartment to the other, as demonstrated well by Ripart et al³.

Needle Length: The standard Atkinson needle is 1 7/8" (38mm) in length (even though Atkinson⁴ described using a 1 3/8" (35mm) needle). It is too long. In 1989 Katsev et al⁵ published a study of the orbital length of 120 skulls. In that study, 20% of the orbits were short enough that a 1 7/8" needle would be able to reach within 7mm of the optic canal. In that area of the orbit, structures are packed tightly together and are vulnerable to damage by a needle. Katsev et al recommended that needles 1 3/8" (31mm) or less be used in order to avoid harm in patients with short orbital lengths. This author has used a 1" needle to perform a shallow, intraconal block quite successfully for about three years in all patients. Prior to that a 1 7/8" was used. The block results have been as good or better with the 1" needle. There is no need to use a long needle, and by using a shorter

needle several of the severe complications of orbital blocks will be less likely to occur, including significant retrobulbar hemorrhage, intravascular injection, brainstem anesthesia, and optic nerve injury. Avoiding the deep portion of the orbit, as recommended by Katsev et al, makes anatomical sense and does not adversely affect the quality of the block.

A final word on needles: the needle is an important tool in performing an orbital block. It is incumbent on each of us to know the exact length, gauge, and tip-type of the needles we use to perform blocks and to record it in the patient's record when doing a block.

Needle Insertion Point: When looking face-on at the orbit, we have several choices of points to insert a needle. The inferonasal quadrant is not a good point for insertion because the origin of the inferior oblique muscle lies there. The superonasal quadrant is not a good point, either, because many important and easily damaged structures are there, including the superior oblique muscle and the trochlear mechanism, the end branches of the ophthalmic artery, the beginning of the superior ophthalmic vein, and the end branches of the nasociliary nerve. The superotemporal quadrant is relatively devoid of structures except for the lacrimal gland, and several authors have advocated insertion of needles in this area to supplement blocks. The inferotemporal quadrant is the area most free of structures and is most frequently used in performing blocks. Through the years it has been most common to instruct people to insert the needle at the junction of the lateral one-third and medial two-thirds of the inferior orbital rim. When one looks at the frontal anatomy of the orbit, one can immediately see that at this point one can easily encounter the inferior rectus muscle or the neurovascular bundle of the inferior oblique muscle. Both muscles are among the most common demonstrating prolonged dysfunction following an orbital block. Atkinson⁴ is commonly credited with suggesting this entry point for an orbital block. In his paper, Atkinson actually states: "...an intradermal wheal is first raised a short distance below the inferior temporal margin of the orbit... The 3.5 cm needle is then introduced through the wheal...so that the point just clears the inferior orbital margin." Nowhere in this paper does he suggest the

junction of the lateral one-third and medial two-thirds. Unfortunately, one of the illustrations in this paper shows a needle entering at that point instead of at the inferior temporal of the orbit, as he stated. In fact, the inferior temporal margin or corner of the orbit is anatomically an excellent place to insert a needle. Behind this point is a compartment of fat that leads directly to the intraconal fat-filled space. By inserting the needle at this point, one has the best chance of entering the gap between the lateral and inferior rectus muscles, thus sparing them from direct needle damage and/or intramuscular injection. In my opinion, based on the anatomy of the orbit and on my own experience and that of others, the classic entry point should be abandoned in favor of an entry point at the extreme inferotemporal corner of the orbit.

A special statement needs to be made regarding the upper half of the orbit. When you closely examine the anatomy of the orbit, you will note that the major and largest vascular structures (ophthalmic artery and superior ophthalmic vein) lie in the upper half of the orbit. The further posterior in the orbit, the larger these vascular structures become. Thus, in order to avoid the vascular complications of orbital regional anesthesia (retrobulbar hemorrhage and intravascular injection), one should avoid the upper half of the orbit as well as the deep orbit.

A second entry point needs to be mentioned due to the necessity of occasionally supplementing blocks in order to achieve complete akinesia. Medial to the medial rectus muscle there lies a fat-filled space (the medial canthal space) separating the muscle from the medial wall of the orbit. This space can be easily entered, as will be described in greater detail subsequently. This is a very useful space for injecting local anesthetic to supplement a block, but some individuals use it as the compartment into which they inject primarily.

Needle Direction: After the needle has been inserted at the inferotemporal corner of the orbit, how does one direct it? As suggested earlier in the reference by Lai et al¹, many people direct the needle toward the apex of the orbit. In fact, this is a dangerous direction because of all the structures packed into a tight

area behind the globe that are headed toward or coming from the optic canal and annulus of Zinn. Behind the eye there is an intraconal fat pad which extends outward into the extraconal area. Local anesthetic spreads easily throughout the orbit when injected into this fat so long as sufficient volume is used. There is no need to aim toward the apex nor to have the needle tip deep in the orbit.

One begins by envisioning a line connecting the superonasal and inferotemporal corners of the orbit. The needle is inserted on this line at the inferotemporal corner of the orbit and aligned with it. With the patient's eye in neutral gaze, a sagittal plane is envisioned passing through the lateral limbus. The one-inch needle is then angled in such a way that the tip will pass tangential to the globe and just touch that limbal plane when it reaches the intraconal fat-filled space about 5mm behind the hind surface of the eye. The angle that one must use to attain this end point is determined by two factors that are different in every patient: the axial length of the eye and the degree to which the eye is either proptotic or deeply set. If the eye is deeply set and/or long, the angle of insertion will be considerably steeper than when the eye is short and/or proptotic. Aiming the needle in this way makes it very unlikely that one will damage the optic nerve, the major vessels, or the extraocular muscles.

[A technical note: I begin the block with the patient's eye in neutral gaze. I insert the needle with the bevel facing the globe. When the needle tip is beyond the equator (frontal plane) of the globe, I rotate the needle 180° so that the bevel is now facing the lateral orbital wall and I ask the patient to look toward my nose. By asking the patient to look laterally, the optic nerve and the posterior pole of the eye are directed well away from the tip of the needle. Rotating the needle helps bring the tip behind the eye without having to otherwise redirect the needle. I make no purposeful attempt to redirect the needle once I have determined the proper angle of insertion other than to rotate the bevel.]

The length of the patient's eye is an important factor to keep in mind when performing orbital blocks. Many authors have stressed the relationship of axial length and the incidence of

globe penetration or perforation. Edge and Navon⁶ described 7 cases of perforation in 50,000 blocks. In each case the eye was greater than 27mm in axial length and each had a staphyloma. For every case of cataract surgery, the axial length has been measured. The axial length in these cases should be recorded with the block technique. If the axial length is unknown, as in cases other than cataract surgery, one should at least attempt to find and record the patient's spherical equivalent. High myopes are very likely to have long axial lengths. In the absence of both axial length and spherical equivalent, one should try to determine and record whether or not the patient is a high myope.

The globe-orbit relationship is also important. Is this a long eye deeply set in a tight orbit or a short, proptotic eye in a very loose orbit? Not only is it wise to examine this relationship in every patient, it is very prudent to record it with the axial length or spherical equivalent in the space in the patient's record where the block is recorded.

Supplementing Blocks: Using a short needle to perform a shallow intraconal block, it is necessary to inject 4-8mL in order to get a good block. I routinely use 8mL unless the globe is beginning to become tight within the orbit as I inject. ***I also have my assisting nurse place her fingers gently along the inferior orbital rim to bolster the orbital septum in that area. Doing this helps to promote the flow of anesthetic upwards and backwards instead of into the lower lid.*** The incidence of greater than 95% akinesia should be greater than 90% after one injection. Due to the vagaries of the orbital connective tissue system, sometimes the medial and/or superior compartments are less well covered with local anesthetic. There are two alternatives: repeat the inferotemporal block or perform a medial canthal block. To perform a medial canthal block, one inserts a 1" needle into the little tunnel that lies posterior to the medial canthus and anterior to the caruncle, aiming toward the medial wall of the orbit. Upon just touching the medial wall, the needle is withdrawn about 1-2mm and redirected to be inserted into the orbit parallel to both the orbital wall and orbital floor. The needle must not be inserted aggressively, because the optic canal lies directly at

the posterior aspect of the medial orbital wall. Never use a needle longer than one inch in this area, and do not let the shoulder of the needle (where shaft and hub meet) go deeper than the plane of the iris. About 4mL injected here usually provides a perfect supplement to the block, although more or less may be required, depending on the patient. It is wise to wait at least five minutes after the inferotemporal injection before doing this supplemental injection. Some people use this approach as the primary block and inject about 8 or 9 mL. Other techniques have been described for blocking in this area^{7,8}, but the technique described by Husted et al⁹ has been very safe and effective in my hands.

Summary: It is unnecessary to use long needles to perform orbital regional anesthesia and doing so may increase the hazards of the procedure. Inserting the needle at the junction of the lateral one-third and medial two-thirds of the inferior orbital rim is not anatomically defensible, because it endangers the inferior rectus muscle and the neurovascular bundle to the inferior oblique. A more anatomically desirable insertion point is the extreme inferotemporal corner of the orbit. The needle should not be aimed at the apex of the orbit. By aiming a short needle to just intercept a plane going through the lateral limbus, one can avoid many of the most severe complications of orbital regional anesthesia.

References:

1. Lai MM, Lai JC, Lee W-H, et al. Comparison of retrobulbar and sub-Tenon's capsule injection of local anesthetic in vitreoretinal surgery. *Ophthalmology* 2005; 112:574-579.
2. Fanning G. Orbital regional anesthesia: let's be precise. *J Cataract Refract Surg* 2003; 29:1846-1847.
3. Ripart J, Lefrant J, de la Coussaye J, et al. Peribulbar versus retrobulbar anesthesia for ophthalmic surgery. *Anesthesiology* 2001; 94:56-62.
4. Atkinson WS. Retrobulbar injection of anesthetic within the muscle cone. *Arch Ophthalmol* 1936; 16:494-503.
5. Katsev DA, Drews RC, Rose BT. An anatomical study of retrobulbar needle path length. *Ophthalmology*. 1989; 96:1221-1224.
6. Edge R, Navon S. Scleral perforation during retrobulbar and peribulbar anesthesia: risk factors and outcome in 50,000 consecutive injections. *J Cataract Refract Surg* 1999; 25:1237-1244.
7. Ripart J, Lefrant J, Vivien B, et al. Ophthalmic regional anesthesia: medial canthus episcleral (sub-Tenon) anesthesia is more efficient than peribulbar anesthesia: a double-blind randomized study. *Anesthesiology* 2000; 92:1278-1285.
8. Nouvellon E, L'Hermite J, Chaumeron A, et al. Ophthalmic regional anesthesia: Medial canthus episcleral (sub-Tenon) single injection block. *Anesthesiology* 2004; 100:370-374.
9. Husted RF, Hamilton RC, Loken RG. Periocular local anesthesia: Medial orbital as an alternative to superior nasal injection. *J Cataract Refract Surg* 1994; 20:197-201.

General References:

- Dutton JJ. *Atlas of Clinical and Surgical Orbital Anatomy*. WB Saunders Company, Philadelphia, 1994.
- Gills JP, Husted RF, Sanders DR, eds. *Ophthalmic Anesthesia*. Slack, Inc., Thorofare, New Jersey, 1993.
- Kumar C, Dodds C, Fanning G, eds. *Ophthalmic Anaesthesia*. Swets & Zeitlinger, Lisse, The Netherlands, 2002.
- Fanning G. Orbital Regional Anesthesia. In Moster MR, Azuara-Blanco A, eds. *Ophthalmology Clinics of North America* 2006;19:221-232. Saunders, Philadelphia.

Workshop C
Session 1

Sub-Tenon's Technique: My Version

Chandra Kumar MBBS DA FFARCSI FRCA MSc

NOTES

Workshop C
Session 2

Sub-Tenon's Wet Lab

Steven Gayer MD MBA and Scott Greenbaum MD

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