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Two third of patients who undergo cataract surgery have at least one chronic systemic disease.\textsuperscript{1}

Hypertension (HTN) is the most prevalent co-morbidity and present in almost 50% individuals.\textsuperscript{2,2} In this article, we shall discuss the definition, peri-operative risk and management of patients with HTN posted for ophthalmic surgery.

Table 1 gives the classification of blood pressure (BP)\textsuperscript{3}

<table>
<thead>
<tr>
<th>BP Category</th>
<th>Systolic BP</th>
<th>Diastolic BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;120 mm Hg</td>
<td>&lt;80 mm Hg</td>
</tr>
<tr>
<td>Elevated</td>
<td>120–129 mm Hg</td>
<td>&lt;80 mm Hg</td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 1</td>
<td>130–139 mm Hg</td>
<td>80–89 mm Hg</td>
</tr>
<tr>
<td>Stage 2</td>
<td>≥140 mm Hg</td>
<td>≥90 mm Hg</td>
</tr>
</tbody>
</table>

This categorization appears in the ACC/AHA (American college of cardiology / American Heart Association) 2017 guidelines.\textsuperscript{3} The previous JNC (Joint National committee)\textsuperscript{7} has a category called pre-hypertensives which includes the elevated HTN and Stage 1 HTN in the above table.\textsuperscript{4} The JNC 8 recommendation mainly focuses on the management of hypertension and target of BP to be achieved rather than classification of stage of hypertension.\textsuperscript{5}

**Ocular problems in hypertension**

Hypertension is known to cause conformational changes in lens protein which lead to the development of posterior subcapsular cataract.\textsuperscript{6}

Hypertensive retinopathy, choroidopathy and optic neuropathy are a spectrum of retinal manifestations that represent target end organ damage. Mild to moderate hypertensive retinopathy changes are reversible but in later stages, there is a permanent damage to blood retinal barrier causing devastating and irreversible retinal exudates and hemorrhages.\textsuperscript{7} The retina is a window to study human circulation. The retinal arterioles share anatomical and physiological similarities with the cerebral and coronary circulation. Stage 3 and 4 hypertensive retinopathy with features of hemorrhage, cotton wool spots, microaneurysm, hard exudates and optic disc swelling suggest increased risk of stroke and coronary events. Such findings in an ophthalmic patient should alert the anaesthesiologist to systemic risk even if the patient has not been evaluated for the same.\textsuperscript{8} Physicians use hypertensive retinopathy to predict the risk of stroke, cardiovascular morbidity and even mortality. Retinal arterial narrowing is associated with the risk of chronic kidney disease and microalbuminuria. It also closely correlates with decreased myocardial blood flow and perfusion reserve. Patients with hypertensive retinopathy have more than twice the risk of developing congestive heart failure.\textsuperscript{7}

Other ocular conditions that may affect hypertensive patients include anterior ischaemic optic neuropathy, retinal vein occlusion, retinal arteriolar emboli, age related maculopathy (AMD) and glaucoma.\textsuperscript{8}

**Hypertension: Population risk versus peri-operative risk**

Hypertension is often asymptomatic and identified on routine screening. Longstanding hypertension is associated with coronary artery disease, stroke, chronic kidney disease (CKD), heart failure and damage to the peripheral vascular tree.

The perspective and goal of hypertension management differs between the anaesthesiologist and the physician. While the anaesthesiologist is more concerned about the perioperative risk which is not much worrisome, the physician is more concerned about long term risk reduction of cardiovascular complications especially stroke. Hypertensive patients have greater fluctuation in BP during the perioperative period. This may translate into minor physiological derangements like hypertension, hypotension and arrhythmia at induction, laryngoscopy, intubation or emergence.
There may be an exaggerated hemodynamic response to nociceptive stimulus. Other factors like hypoxia, hypothermia or excessive fluid resuscitation may contribute to variation in BP. However, there is no conclusive evidence to show that they cause harm or major adverse event.9

In treating hypertension, it is necessary to consider the total cardiovascular risk factor. Patients with personal or family history of stroke are at greater risk of cerebral hemorrhage.

**Management of HTN: Evaluation and pharmacotherapy**

The ACC/AHA 2017 has revised the treatment goals based on overall cardiovascular risk. It is recommended that the target for BP control be identified after ascertaining the atherosclerotic cardiovascular disease (ASCVD) risk in patients.3 There is an online tool to calculate ASCVD risk.10 For adults with confirmed hypertension and known CVD, CKD, DM or 10-year ASCVD event risk of ≥ 10%, a BP target of <130/80 mm Hg is recommended. For adults with confirmed hypertension, but without additional markers of increased CVD risk, a BP target of <130/80 mm Hg is recommended as reasonable. It is important to screen for and manage other CVD risk factors in adults with hypertension: smoking, diabetes, dyslipidemia, excessive weight, low fitness, unhealthy diet, psychosocial stress, and sleep apnea. Basic testing for primary hypertension includes fasting blood glucose, complete blood cell count, lipids, basic metabolic panel, thyroid stimulating hormone, urinalysis, electrocardiogram with optional echocardiogram, uric acid, and urinary albumin-to-creatinine ratio. They recommend deferring elective major surgery at BP > 180/110.3

The recommended drugs for the first line treatment of hypertension are thiazide diuretics, calcium channel blockers, angiotensin receptor blockers and ACE inhibitors. Drugs like beta blockers may be added in specific patients. The drug therapy needs to be tailored in the presence of other co-morbidities like heart failure, diabetes and CKD. It requires 4 to 6 weeks of antihypertensive therapy to achieve adequate blood pressure control and normalization of flow pressure autoregulation of the major organs.15 In managing perioperative hypertension a cosmetic control of BP gives a false belief of security as coronary and cerebral vasoregulation require several weeks of HTN control.19 Patients who are taking anti-hypertensive medication should continue their drugs up to and including the day of surgery. Particularly, beta blockers and clonidine should not be withdrawn as there may be a rebound hypertension.3 They may be replaced parenterally if the patient is unable to take them orally. Rapid lowering of BP immediately prior to surgery is not advised.13 In labile hypertension, there is a pre-procedural increase in BP with normal recorded BP at home or previous clinic visits. It is short-lived and may be treated with anxiolytics and short acting anti hypertensive agents. They may be given prophylactically if the condition is identified earlier.14

**Hypertension in Ophthalmic anaesthesia:**

Hypertensive patients with historic good BP control and without other major comorbidities present a larger incidence of preoperative rise in BP than normotensive individuals in cataract surgery.7 A retrospective review of medical records of 530 patients undergoing various ophthalmic procedures shows that hypertension is the commonest systemic disorder present in 359 (68%) patients. Of them, 161 (45%) had it newly diagnosed or an unstable condition was identified at pre-anaesthesia evaluation.9 An evaluation of 1006 consecutive cases of cataract surgery shows that the anesthetist’s intervention is required more often in hypertensives (41.4%) than in non-hypertensives (34.55%).16 A survey conducted among 104 ophthalmic anaesthetists from the British Ophthalmic Anaesthesia Society (BOAS) reveals that 78% of them (81/104) have cancelled a case due to hypertension. The cut off BP for abandoning the block was a systolic BP more than 200 mm Hg and diastolic BP >110 mm Hg for the majority. These upper limits are neither evidence based nor in accordance with the Royal College guidelines. Instead, they have been derived from local practice. The perceived risks are ocular (expulsive choroidal hemorrhage, orbital hemorrhage and increased intraocular pressure) and cardiac (angina, perioperative myocardial infarction and dysrhythmia).17 The use of 10% phenylephrine drops for pupillary dilatation may be associated with a mild (6-15 mm Hg) increase in blood pressure in a few patients. This may be due to an overdose, increased systemic absorption or
an idiosyncratic effect. It may be prudent to use plain tropicamide, cycloplegylate or 5% phenylephrine with 0.8% tropicamide with punctual occlusion in patients with elevated BP. Uncontrolled severe hypertension can result in suprachoroidal hemorrhage after cataract, glaucoma, keratoplasty or vitreoretinal surgery. Such expulsive hemorrhage is fortunately rare with an incidence of 0.04% after cataract surgery and 0.12% after vitreoretinal surgery but may lead to blindness. Systolic hypertension is an independent risk factor for development of delayed suprachoroidal hemorrhage post glaucoma and cataract surgery. Treating systemic hypertension prior to surgery also reduces the risk of post-operative hemorrhage after pars plana vitrectomy in proliferative diabetic retinopathy.

Controlled hypotension may be beneficial in lacrimal, oculoplastic and orbital procedures. The requirement for a bloodless field may be met by positioning, normotension and surgical site vasoconstriction. The physical health of the patient, the anaesthesiologist’s acquaintance with the technique and availability of drugs are more significant than surgeon preference and local practices in a decision to provide hypotensive anaesthesia. The BP is usually lowered up to 30% from normal in an ASA 1 patient. A hypertensive patient may require a higher mean arterial pressure (MAP) to maintain vital organ perfusion. The safety limit for the lowering of BP needs to be individualized.

**Peri-operative management of HTN - An overview of the existing guidelines:**

The evolving and changing landscape of management of HTN shows our continuous understanding of the disease which is a major risk factor for stroke, cardiac ischemia and chronic kidney disease. The focus has shifted from diagnosis and categorization to revised thresholds and aggressive management of HTN. Uncontrolled hypertension may increase the risks of systemic and ocular complications. However there is insufficient evidence to support a specific value above which surgery should be deferred.

It is interesting to note that the target BP to be achieved after treatment has changed from JNC 7 to AHA 2017 but the BP above which elective surgery is better deferred continues to be 180/110. The ACC/AHA guideline on management of cardiac conditions for non cardiac surgery is silent on the perioperative management of HTN except antihypertensive pharmacotherapy. Beta blockers should be continued in patients undergoing surgery who have been on beta blockers. Beta-blocker therapy should not be started on the day of surgery. Continuation of angiotensin-converting enzyme (ACE) inhibitors or angiotensin-receptor blockers (ARBs) perioperatively is reasonable.

A consensus document between the AAGBI and BHS published in 2015 is the only one available that liaises the concerns of treatment of hypertension in the community and before elective surgery. The salient features of this guideline include:

1. General practitioners should refer patients for elective surgery with mean blood pressures in primary care in the past 12 months less than 160 mmHg systolic and less than 100 mmHg diastolic. Patients may be referred for elective surgery if they remain hypertensive despite optimal antihypertensive treatment or if they decline antihypertensive treatment.

2. Secondary care should accept referrals that document blood pressures below 160 mmHg systolic and below 100 mmHg diastolic in the past 12 months.

3. Elective surgery should proceed for patients who attend the pre-operative assessment clinic without documentation of normotension in primary care if their blood pressure is less than 180 mmHg systolic and 110 mmHg diastolic when measured in clinic.

The joint guideline from RCOA and RCOO on local anaesthesia for ophthalmic surgery states that uncontrolled hypertension increases the risk of both ocular and systemic complications. However there is insufficient evidence to support a specific value above which surgery should be deferred.

The VISION 2020 guidelines for the management of cataract in India recommend a physician reference for patients with a BP > 170/100 during the PAE.
Table 1 lists some of the common drugs that are used in treating hypertensive surges.

Table 1:

<table>
<thead>
<tr>
<th>Drug</th>
<th>Class</th>
<th>Dose</th>
<th>Onset of action</th>
<th>Duration of action</th>
<th>Adverse effect</th>
<th>Contraindication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Esmolol</td>
<td>Cardioselective β1-receptor blocker</td>
<td>250-500 µg/kg bolus dose over one min followed by 50-100 µg/kg/min for 4 min; repeat boluses for further crises and increase maximum infusion dose to 300 µg/kg/min</td>
<td>2-10 min</td>
<td>10-30 min</td>
<td>Unopposed β-blockade may lead to α-storm Higher degrees of heart block</td>
<td>Use with caution in asthmatics/COPD</td>
</tr>
<tr>
<td>Labetalol</td>
<td>Combined α1 and non-selective β-receptor blocker</td>
<td>Loading dose 20 mg; if crises not controlled then 20-80 mg bolus (every 10 min) or alternatively 2 mg/min infusion</td>
<td>5-10 min</td>
<td>Single bolus 2-4 min; repeated bolus or infusion 2-6 h</td>
<td>Nausea Angioedema (rare)</td>
<td>Asthmatics COPD Higher degrees of heart block</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>Cardioselective β1-blocker</td>
<td>2.5 to 5 mg IV bolus over 2 minutes; may repeat every 5 minutes to a maximum dose of 15 mg</td>
<td>Immediate, peaks in 20 min</td>
<td>5-8 h</td>
<td>Hypotension, heart block, bradycardia, bronchospasm, Heart failure</td>
<td>AV block, Bradycardia, cardiogenic shock, decompensated heart failure, sick sinus syndrome, pheochromocytoma</td>
</tr>
<tr>
<td>Clinidipine</td>
<td>Dihydropyridine type of Calcium channel blocker</td>
<td>1-2 mg/h can be doubled every 90 second. Max 32mg/h</td>
<td>2-4 min</td>
<td>5-15 min</td>
<td>Non-specific</td>
<td>Allergic to soya or egg products Disorders of lipid metabolism such as pancreatitis, lipoid nephrosis</td>
</tr>
<tr>
<td>Drug</td>
<td>Class</td>
<td>Dose</td>
<td>Onset of action</td>
<td>Duration of action</td>
<td>Adverse effect</td>
<td>Contraindication</td>
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<td>-------------------</td>
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<td>--------------------------------------------</td>
</tr>
<tr>
<td>Diltiazem</td>
<td>nondihydropyridine calcium channel blocker</td>
<td>0.25 mg/kg (average 20 mg) IV over 2 min; may give 2nd bolus (0.35 mg/kg, average 25 mg) can be given 15 minutes later if HR &gt; 100 bpm), then 5 to 15 IV mg/hr</td>
<td>2-5 min</td>
<td>2-4 h</td>
<td>hypotension, heart block, HF</td>
<td>acute MI and pulmonary congestion, sick sinus syndrome, 2nd or 3rd degree AV block</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>Peripheral vasodilator</td>
<td>Initial dose is 10 mg slow intravenous bolus, every 4-6 h as required; bolus doses should not exceed 20 mg</td>
<td>10-30 min</td>
<td>2-6 h</td>
<td>Vascular collapse Peripheral neuropathy Thrombocytopenia Volume overload</td>
<td>CAD, Rheumatic MS, SLE</td>
</tr>
<tr>
<td>Sodium nitroprusside</td>
<td>Nitric oxide donor; acts on both arterial and venous smooth muscles</td>
<td>0.3-0.5 µg/kg/min; avoid doses &gt;2 µg/kg/min</td>
<td>Immediate</td>
<td>2-3 min</td>
<td>Cyanide toxicity Chromaturia (red urine) Erythema</td>
<td>Raised ICP</td>
</tr>
<tr>
<td>Nitroglycerin</td>
<td>Venodilator</td>
<td>5 µg/min up to maximum of 20 µg/min</td>
<td>2-5 min</td>
<td>5-10 min</td>
<td>Headache, Methaemoglobinemia -</td>
<td></td>
</tr>
</tbody>
</table>

There are different scenarios in which an anaesthesiologist encounters a patient with increased BP prior to ophthalmology surgery.

1. **In the PAC clinic prior to planned surgery**: A patient may be first identified as a hypertensive by the anaesthesiologist. They may never have been investigated or treated for high BP. Any patient with a BP >140/90 but <180/110 may be accepted for surgery but advised to have his BP monitored at home and seek physician/cardiologist consult for management of the same. It is prudent to order blood tests like blood glucose, creatinine and ECG to evaluate target organ damage and explain that this BP does not pose any major peri-operative risk but has long term effects on the heart, brain and kidney. If it is a patient with cardiac risk factor, CKD or diabetic, a referral for management of BP prior to elective surgery is reasonable. A BP ≥180/110 should prompt urgent referral for management of hypertensive crisis.
2. **On the day of surgery with a single elevated reading:** If it is a previously evaluated patient, an acute rise in BP may be a “white-coat” phenomenon and may only require anxiolysis. It is alright to proceed with ophthalmic surgery under regional anaesthesia if BP < 180/110. However, patients undergoing surgery under general anaesthesia are prone to hemodynamic lability. There is little evidence to show association between BP < 180/110 and perioperative complication. It is not uncommon in ophthalmic anaesthesia practice to encounter previously unevaluated patients on the morning of surgery with a high BP. A preoperative BP < 180/110 on the morning of surgery is acceptable and anything above this constitutes a hypertensive crisis.

If a BP > 180/110 is associated with signs of end organ damage (renal, retinal, cerebral, coronary), it is a hypertensive emergency and strong preference should be given towards deferring the case until the hypertensive emergency is treated with oral/parenteral anti-hypertensives and stabilized. Anxiolysis may form part of the treatment. Patients with certain medical conditions like renal failure may require specialist consultation when their BP is not adequately controlled. Ophthalmology surgery scheduled under regional anaesthesia is almost never an emergency and may be deferred until the HTN is medically managed. It is not recommended to acutely lower BP in a short time.

In the others, a decision to do, defer or cancel the surgery will have to be taken by the team based on patient factors like age, comorbidities, degree of hypertension as well as the type and urgency of the case.

3. **Emergency surgery:** It is appropriate to proceed with and anaesthesia after having explained the risk to the patient/relatives and be prepared for intra-operative and post-operative hemodynamic instability.

**Conclusion:**

Systemic hypertension is the most common cause for cancellation of cataract surgery. A thorough understanding of this condition helps the ophthalmic anaesthesiologist to approach and manage hypertensive patients in an evidence based and methodical way.

The condition itself may not affect the ophthalmic procedure but serious enough to warrant further primary care referral and evaluation.

**References:**


Over the hills and across the downs
A mini-review on EEG-guided multimodal general anaesthesia in ophthalmic surgery
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Email: lamicofritz@hotmail.com

Ophthalmic surgery is undertaken several thousand times every day across the world. Ranging from uncomplicated cataract-surgery to extended retinal tears requiring buckles or glaucomatous eyes having been operated many times before—ever so often the clinicians involved will decide on choosing general anaesthesia (GA) over locoregional (LA). Whilst the choice of regional anaesthesia has seen a lot of debate in the clinical societies involved with ophthalmic anaesthesia (OA) it seems that there’s more consensus amongst anaesthesiologists on how to perform GA in ophthalmic surgery for children, the elderly, frail and mentally disabled. The common motives are that the patient should be analgo-sedated deep enough so no movement or straining against the tracheal tube occurs. As is known, these might put vision at great peril: either through uncontrolled movements of head and eye whilst surgical tools are in the eye or through uncontrolled peaks of intraocular pressure limiting retinous perfusion or endangering ocular integrity in the context of “open-sky”-situation. To achieve a safe surgical field in the eye many methods are employed, deep analgosedation or thorough curarization being only the most frequently employed. But the fear of unanticipated patient-movements not rarely leads to a mindset that might be called “spiral of fear”. Without good endpoints to gauge anaesthesia-depth to, especially newcomers to the field of OA are wanton to oversedate a fair proportion of their very old or very small patients, having many of them come to deliriously after long emergence. As if this were not enough, occasionally patients will move under anaesthesia notwithstanding the high doses of opioids, propofol or halogenated ethers. Apart from reproachment from the surgeon the anaesthetist will be ever more inclined to go “deeper” with the next narcosis. Although there are certainly many ways out of this “spiral of fear”, I’d like to sketch my own approach arrived at after making many of the frustrating experiences alluded to in above lines.

It was developed especially for the care of elderly patients in ophthalmic anaesthesia. Ever so often they bring up the topic of post-operative delirium and not few of them suffered this severe complication in previous surgeries. What they relate are frightening hallucinations and misperceptions, some of them hurt themselves during agitation or are ashamed because they behaved indecently or insulted loved ones. Over the last years I tried to adapt my GA’s to the specific situation of elderly and frail patients, using the following strategies as part of our standard Bernese ophthalmic anaesthesia—protocol

1) EEG-guided GA
2) Use of dexmedethomidine as part of the hypnoto-analgesic “cocktail”
3) Multimodal, opioid-sparing-anaesthesia
4) Combination of GA with locoregional anaesthesia
5) Engaging subjective cognitive content of the patient through hypnotic suggestion or music

All of these aspects will be addressed briefly as they are parts of one protocol. It’s my conviction that the application of each of them to ophthalmic anaesthesia would merit a mini-review in their own right. While being particularly cautious not to make any hyperbolic claims or recommendations—many of strategies to be outlined in the following lines still await objective proof in prospective randomized trials, I nevertheless want to state clearly that outlined protocol has made emergence delirium vanish completely in our small unit at Berne’s university eye hospital. Extubation and turn-around time have been drastically reduced and patient satisfaction increased significantly. All ophthalmic surgeries are to some extent undertaken to improve an old person cognitively (Jefferis) and increasing evidence shows it does (Maharani). Hence, with paradigmatic demographic changes underway leading to increased old and very old patients being operated on in ophthalmology, it’s time ophthalmologists and anaesthesiologists factor in GA into the equation and come up with Strategies preserving cognition to the best of their abilities (Jefferis).
EEG-guided GA

Many an ophthalmic-anaesthetist has learned the hard way that clinical signs of arousal —like accelerated heart rate, raised blood-pressure etc—before a patient makes unwanted movements of the head—are unreliable predictors. As much as patients and anaesthetists fear awareness with recall (AWR) stemming from “too little” anaesthesia we should fear oversedation just the same, as the latter entails severe cognitive complications (Avidan) like delirium. Choosing an anaesthesia-protocol with short spans of curarization or none at all helps to reduce the risk of AWR while titrating „anaesthesia-depth“ towards an EEG helps to avoid oversedation. The last two decades have seen an upsurge in literature reflecting our communities learning-curve with perioperative EEG-monitors. For a thorough review the work of the head of ophthalmic anaesthesia at the university hospital in Liège, Belgium, Dr. Nicolas Marchant, is highly recommended. The basic idea is that EEG-monitors analyze a raw EEG measured by frontal electrodes, perform Fourier-transformation of the raw data and after internal comparison of EEG-changes to an internal dataset provide a dimensionless index number. This number (0-100) is arrived at by calculations applying company-owned algorithms unknown to the scientific community (Marchant) and taken to indicate sufficient (< 60) anaesthesia. Yet every now and then titrations according to the dimensionless index will confront the observing anaesthetist with problems, especially when clinical signs of wakefulness persist although the EEG-monitor indicates a degree of sufficient “anaesthesia-depth”. Applying “isolated-forearm-technique” where use of a tourniquet prevents paralysis of the right arm demonstrated that variable percentages of patients before or after laryngoscopy would react to being addressed by name and cued to shake hands (Sanders) even if their EEG-monitor indicated sufficient “narcotic depth”. Comparing several studies one would find that roughly one third of the anaesthetized patients shaking hands with the researcher after eventless intubation would not recall the event, another third would recall it indifferently and the last third recall pain. Although the studies Dr. Sanders cites were methodologically very heterogeneous in design, one cannot help but arrive at the conclusion that our management of narcotic unconsciousness is more complex than usually appreciated in daily clinical life.

Relating the cortico-thalamic system with “consciousness” and the complex subcortical networks (brainstem, midbrain, limbic system etc) with “responsiveness” and using these two factors to describe varying degrees of “connectedness” during different levels of wakefulness, sedation and anaesthesia represents a seminal change in our conceptual framework of narcotic unconsciousness (Sleigh J).

Another very courageous study from Australia aptly demonstrates the limitations of dimensionless indices to titrate anaesthesia. P. Schuller and colleagues tested changes in BIS-monitor-indices in 11 fasted volunteers, all of them anaesthetists. Using isolated-forearm technique to communicate by handshakes, subjects were curarized with either succinycholine or esmerone and ventilated by face-mask without any sedatives being administrated. Although raw-EEGs indicating wakefulness remained unaltered, BIS-indices dropped to 80 in a first movement and then leveled out at values as deep as 50 while communication via handshakes indicated perfectly normal awake consciousness. These results shed some doubtful light on the reliability of numerical indices to quantify “depth-of anaesthesia” especially demonstrating some significant overlap in oscillations in the beta-band shared by scalp-musculature and frontal cortex. So, after 30 years of experimenting and learning to administer GA according to “numerical EEGs” are we again left with nothing and the mistrust to the “new” technology warranted? (Andrzejowski JC). While mistrusting dimensionless numerical indices as substrate for different degrees of consciousness is certainly justified, new ways of taking “cortical activation-patterns” into account are being practiced. Better grasp of the raw EEG by anaesthesiologists—taking into account basic patterns—will lead to better end-points in anaesthesia especially of the old and frail. The basic patterns are easy to learn and free online teaching programs from some of the leading study groups in the field, like www.anesthesiaEEG.com (Brown E & Purdon P) as well as http://icetap (Avidan M) provide outstanding learning experiences. There’s ample instruction on recognizing raw EEG-patterns as well as using spectrograms to recognize changes in cortical activation patterns. Especially analogies to sleep-EEGs are of interest to the anaesthesia-community, as similarities in EEG-patterns and their sequence reflect how thalamocortical oscillations come under control of sub-cortical structures, being slowed and synchronized in the process (Brown).
Imaging (fMRI) and EEG-studies foster the assumption that GA mediates its effects in part by hijacking sleep networks that physiologically elicit loss of wakeful consciousness through control of of circadian rhythms and the sleep homeostat (Poulsen). Strikingly, anaesthetists monitoring patient’s induction by frontal EEG can observe transition from busy, low-amplitude Beta-wave dominated EEG signals through a stage of light sedation, often hallmarked by eye-movement and blinking artifact and high EMG-activity. This signaled passage to deeper anaesthesia and the appearance of frontal alpha-waves was known as early as 1941 (Burford).

Alpha-waves seem to play a particularly important role in making their appearance in frontal EEG-leads while disappearing from the occipital leads- a process called “Alpha-anteriorization”, typical for propofol-induction (Feshchenko). Mark that the signature-EEGs of various anaesthetic drugs differ significantly. The following sequence of deepening narcotic unconsciousness is indicated by EEG-signatures typical for NREM-sleep stages: increasing proportions of delta-waves coupled with alpha-waves and sleep-spindles. These particular oscillation patterns have received increasing attention in anaesthesia-research as hallmarks of thalamo-cortical uncoupling from the external stimuli (Sleigh) and orderly sequence in passing through different sequences on induction and emergence is tied to reduced risk of post-operative delirium in aged patients (Hesse S). Their abrupt disappearance from EEG-traces while anaesthesia is continued at the same depth possibly indicates intrusion of nociceptive input into autonomous thalamo-cortical oscillation (Hagihira). What is more, the physiological connection of spindles to memory-stabilization in sleep furthers speculation that spindle-predominant EEGs during GA might imitate a particularly beneficial brain mechanism. It’s consequences in maintaining cognitive function across GA in the elderly remain to be seen.

As for detection of “nociception”– not “pain”, as the patient would need to be conscious to experience a noxious cue as pain– the hunt for their EEG-signatures is still on (Hagihira), but is becoming increasingly fruitful. An Oxford based research-group led by Prof. Rebecca Slater continues to publish impressive data on pain-signatures in the EEG of premature babies undergoing heel-lancing and eye examinations to detect retinopathy of prematurity (Hartley C). Apart from EEG-changes that follow partial awakening by means of nociception– like “Beta-arousal”, “spindle-loss” and frontal “Alpha-loss”– interestingly the Slater-group showed increase in delta-frequency in premature babies exactly time-locked to noxious stimuli under sevoflurane anaesthesia.

As for orderly emergence from GA (Hesse S), there are many points to be made for leaving on EEG-electrodes until the anaesthesiologist is able to converse with the extubated patient. EEG-diagnosis of post-operative delirium is improving and signifies that an objective measure of this cognitive complication of surgery and anaesthesia is in the hands of anaesthesiologist (Numan). This is particularly important as the literature shows that two-thirds of delirium-patients present hypoactive rather than in agitation, often times going unnoticed. The debate is furthered by detailed description of EEG in different delirium-situations connected to hepatopathic or sepsis-associated encephalopathies (Palanca BJA)

Combining general anaesthesia with locoregional blocks

A practice described extensively during the first decade of the 21st century is “combined ophthalmic anaesthesia”. Locoregional anaesthesia is used here to replace or reduce the use of narcotic agent (halogenated ethers) and opioid-analgesia. In the context of opioid-free/opioid-sparing anaesthesia currently debated in the anaesthesiological community, this receives new actuality. In studies performed in children undergoing VR-surgery, A. Chhabra et al elegantly demonstrated improvement of intraoperative oculo-cardiac-reflex and intra-and postoperative opioid-consumption when comparing sub-Tenon’s-Block with fentanyl as part of a standard GA. It is to be presumed that their results pertaining to postoperative analgesia would have been even more striking, had a STB-top-up also been performed at the end of surgery, as is often done in daily clinical practice. AD Farmery et al arrived at similar conclusions in their study of adult VR-surgery-patients concerning intraoperative bradycardia and postoperative analgesia consumption.

L. Bergman et al demonstrated that preoperative ropivacaine-STB in sevoflurane-anaesthetized patients undergoing buckle-surgery for retinal detachement led to better intraoperative hemodynamic control, reduced need of sevoflurane, better post-operative analgesia with lower opioid-consumption and shorter time spent in recovery unit.

Our clinical observations are in tune with these published results: shorter time-span to spontaneous respiration with airway secure, shorter time to extubation and a different recovery, where patients—after waking-up and replying meaningfully to standard questions—tend to sleep another hour or two but then come to orderly and refreshed.

An unsettled question pertains to retinal perfusion under GA in combination with ophthalmic regional blocks. Theoretically one would need to be apprehensive of increasing intra-ocular pressure while perfusion of the eye is reduced in the context of GA-related reduced forward-flow.

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**ACKNOWLEDGEMENT**

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support in bringing out this Newsletter
During retrobulbar block, injury to optic nerve subsequent or inadvertent dural puncture of the optic nerve sheath and local anaesthetic injection into the cerebrospinal fluid space can cause spread of the local anaesthetic solution to the various centres in the mid-brain. Since peribulbar block is given away from the optic nerve such complications are remote. Shivering is one of the many complication of retrobulbar block. But there are no reports of shivering following peribulbar block. We had a case of violent shivering following peribulbar block.

**Case Report**

A case of a severe shivering following perbulbar block is presented. The shivering occurred soon after completion of peribulbar block administered for phacoemulcification extracapsular cataract extraction and intraocular lens implantation in the left eye in a 63 year old female patient. She had no history of allergy to drugs or injections. She had a previous eye surgery in the right eye with no complication. Preanaesthetic evaluation revealed a history of systemic hypertension for the past 2 years and was under control with Tablet Amlodipine 5mg once daily. Her blood pressure was 150/90 mmHg and her random blood sugar was 150 mg/dL. Before administering the peribulbar block her blood pressure was 140/87 mm Hg, Heart Rate: 80 – 84 / minute, SaO2 : 99% She was administered peribulbar block with 8 ml of 2% xylocaine with hyaluronidase under monitored care of blood pressure heart rate and pulse oximetry. Soon after block she complained of cold and started shivering. She was covered with a blanket. She still felt cold and was shivering. So a heater was provided and hot air blown under the blanket. She was administered oxygen 3 lit/min via nasal cannula. When she felt better she was transferred into the operation theatre. She started complaining of severe cold and shivering severely. The shivering was so severe as to be misjudged as a seizure, but its onset appeared to be slower than a seizure. The patient remained conscious during the episode of shivering. We continued warming the patient and when the shivering lessened and the patient felt better surgery was started.

During surgery the blood pressure rose to 210/114 mm Hg and her heart rate rose to 127 bpm. She never lost consciousness. IV. midazolam 1mg was given. Her blood pressure came down to 160/105 (120) mm Hg and surgery was completed. Post operative blood sugar was 89 mgm/dL. Her nails and tongue were pale. We gave 25% dextrose IV 20 ml and 100 mg hydrocortisone IV was given. (Table 1. Shows perioperative vital signs of the patient.) After 2 hours she was completely normal following which she was discharged.

<table>
<thead>
<tr>
<th>Time</th>
<th>Blood Sugar</th>
<th>Blood Pressure</th>
<th>Pulse Rate</th>
<th>Respiratory Rate</th>
<th>SpO2</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preanaesth</td>
<td>RBS 102mg/dL</td>
<td>140/90 mmHg</td>
<td>80/min</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>evaluation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AB ward</td>
<td>RBS 150mg/dL</td>
<td>150/90 mmHg</td>
<td>82/min</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Admission</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preblock</td>
<td>146/85 mmHg</td>
<td>80/min</td>
<td>16/min</td>
<td>100%</td>
<td></td>
<td>Peribulbar block given</td>
</tr>
<tr>
<td>Postblock</td>
<td>153/102 mmHg</td>
<td>98/min</td>
<td>16/min</td>
<td>100%</td>
<td></td>
<td>I.V.Midazolam 1mg Given</td>
</tr>
<tr>
<td>10-39 AM</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>shivering Covered with blanket</td>
</tr>
<tr>
<td>10-48 AM</td>
<td>136/94 mmHg</td>
<td>100/min</td>
<td>19/min</td>
<td>100%</td>
<td></td>
<td>Transferred to OT</td>
</tr>
<tr>
<td>10-58 AM</td>
<td>153/102 mmHg</td>
<td>108/min</td>
<td>22/min</td>
<td>100%</td>
<td></td>
<td>shivering</td>
</tr>
<tr>
<td>11-10 AM</td>
<td>210/114 mmHg</td>
<td>127/min</td>
<td>27/min</td>
<td>100%</td>
<td></td>
<td>HTN, Tachycardia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Severe shivering – hot air under blanket</td>
</tr>
<tr>
<td>11-14 AM</td>
<td>160/105 mmHg</td>
<td>113/min</td>
<td>27/min</td>
<td>100%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11-25 AM</td>
<td>RBS 85mg/dL</td>
<td>141/92 mm Hg</td>
<td>115/min</td>
<td>32/min</td>
<td>100%</td>
<td>I.V. Dextrose, Hydrocortisone</td>
</tr>
</tbody>
</table>
Discussion

The mechanism of shivering appeared to be the central spread of local anaesthetic solution into the brain stem, along the optic nerve

Spread to contralateral eye

Shivering is one of the manifestation of brain stem anaesthesia which may include convulsion, confusion, loss of consciousness, change in blood pressure, heart rate apnea or change in respiratory pattern, vomiting, shivering, hemiparesis, dysphagia, tinnitus, vertigo, hearing loss etc. Anyone of these complications may appear singly or in combination.

The severe shivering observed appear to have been directly related to central spread rather than the patient’s coincidental exposure to a cold environment.

The nature of shivering observed in the present case was quite unique. It was so severe as to be misjudged as a seizure. The patient could not understand why she was shivering so severely.

The episode of shivering was probably the result of local anaesthetic solution spreading along the optic nerve sheath contacting an area of the brain stem linked to the shivering mechanism. Stimulation of this area can produce shivering. In animals experiment, local anaesthetic solution applied to the ventro medial reticular formation of the brain stem facilitates shivering whereas application to the lateral pontine reticular formation inhibits shivering.

Diagnosis

Pupillary dilatation and partial akinesia of the extraocular muscles of the contralateral eye may occur with or without any other sign. This sign is pathognomonic of central spread of local anaesthetic agent. This sign should be looked whenever any abnormal reaction occurs following the block.

Such cases require monitoring until signs and symptoms resolve. Hypotension and bradycardia have been observed with brainstem anesthesia, but more commonly hypertension and tachycardia result. The hypertension and tachycardia are either due to vagolysis or blockage of the carotid sinus reflex via the glossopharyngeal nerve.

Conclusion

Any abnormal reaction following peribulbar block, should exclude central spread as the cause for the symptoms. As more and more anaesthetists are now becoming involved in administering and monitoring local ophthalmic anaesthesia, a greater understanding of these complication should be reached and good quality of patient care and monitoring for patients undergoing surgery under local anaesthesia.

References

Perioperative Anaphylaxis
Dr Jagadeesh V
Director, Department of Anaesthesiology,
Sankara Nethralaya, Chennai
E-mail: drjv@snmail.org

Introduction:
Anaphylaxis is a severe, potentially life threatening systemic hypersensitivity reaction. The perioperative incidence is 1 in 10,000 1 in 20,000 anaesthetic procedures and mortality less than 0.001%. It is difficult to identify the causative agent as many drugs are given in the perioperative period. It requires careful analysis of clinical presentation and the time gap between administration of the possible causative drug and the beginning of the reaction.

Pathophysiology – Immunological: IgE mediated and Non IgE mediated.
Non immunological - Idiopathic
IgE mediated is caused by crosslinking of IgE. Degradation of mast cells, basophils, release of mediation, histamine, prostaglandin, proteoglycans and cytokines; 60-70% of periop Anaphylaxis IgE mediated.
Non immunological – direct stimulation of most cells by agent resulting in deregulation and release if mediators. It is transient and may frequent with cutaneous lesions only.
Idiopathic Anaphylaxis when allergen cannot be identified.
Anaphylaxis generally occurs on re-exposure to a specific antigen but can also occur on first exposure as there may be cross reactivity among many drugs.

Risk Factors
1. Patient with history, signs and symptoms suggestive of allergic reactions.
2. History of allergy to specific drugs - analgesics, antibiotics etc
3. Patients who have undergone several operations particularly children with spine bifida due to frequent exposure to latex.
4. History suggestive of latex allergy.

Peri-op Triggers
A small quantity is sufficient to care Anaphylaxis. In the periop setting the common agents involved are antibiotic, neuromuscular blocking agents (NMBA) and latex. The less common ones causing reactions are colloids, hypnotic agents, opioids, dyes and chlorhexidine.
Non IgE mediated anaphylaxis with drugs such as opioids, NSAIDS, Iodinated contrast agents.
NMBA commonly associated with anaphylaxis are Rocuronium and Suxamethonium; can occur cross reactivity with other non-depolarizing agents.
Non immunological opioids, Vancomycin NMBA (atracurium) Heparin, Protamine, oxytocin, local anaesthetics and blood transfusion.
Latex exposure in periop glucose, catheters contact with skin or mucus membrane.

Clinical Features
Grading according to severity of symptom done by Ring and Messner in 1977. The modification is as follows.
Grade I – Cutaneous signs – Erythema, urticaria and angioedema.
Grade II – Moderate multi-organ involvement, hypotension, tachycardia, difficulty in breathing, cough, bronchial hyper reactivity.
Grade III – Life threatening multi-organ involvement cardiovascular collapse, arrhythmias, bronchospasms.
Grade IV – Cardiac / Respiratory arrest

Diagnosis
Anaphylaxis is suspected if there is unexplained hypotension refractory to vasopressors or unexplained difficulty in ventilation; periop anaphylaxis occurs within seconds or minutes after exposure to causative agents.
**Investigations**

**Tryptase**
Serum Tryptase released by mast cell degranulation. Serum Tryptase above 25 µg/ml suggest IgE related mechanism. Serum Tryptase increase is not specific to Anaphylaxis and may be raised in myocardial infraction, trauma, amniotic fluid embolism. Tryptase level may be normal if anaphylaxis is basophil mediated. The half life is 2-4 hrs and returns to baseline in 12-14 hrs. Increased level of Tryptase after 24 hrs suggest late onset reaction, biphasic reaction on mast cells disorders.

**Plasma Histamine**
Elevated plasma Histamine levels correlate with signs and symptoms of anaphylaxis. It increase in 5-15 min and falls to baseline by 60 min. Blood samples for histamine levels require special handling; should be drawn wide bore needle, kept cold, centrifuged and freeze the plasma properly. Urine Histamine levels increase and are more specific than plasma Histamine.

**Treatment**
The treatment should be initiated immediately and the suspected drug stopped.

**Airway**
High flow O₂ to be given if there is respiratory distress and airway should be secured immediately if required; may be difficult in patients where upper airway is edematous and distorted. Repeated attempts lead to respiratory obstruction. Crico-thyroidectomy may be required.

**Epinephrine**
Prompt administration of epinephrine with haemodynamic monitoring is the mainstay of treatment. The main factor associated with mortality by anaphylaxis is delay in epinephrine administration. Epinephrine increases peripheral vascular resistance decreases airway mucosal edema;
Beta1 effect increases heart rate, contraction and Beta2 effect causes bronchodilation.

**Epinephrine Dose**
10-20mcg IV for Grade II reaction
100-200mcg IV for Grade III reaction.
Additional doses should not be delayed. If patient requires repeat boluses infusion at 0.05-0.4mcg/kg/min to be started.

**Other drugs**
In persistent hypotension – Vasopressin or Nor-epinephrine could be used
Response to epinephrine blunted in patients on beta blockers, ACE inhibitors or person in spinal blockade.
Glucagon may be tried in patients on Beta blockers.
Sugammadex may reverse Vecuronium or Rocuronium anaphylaxis. Steroid and antihistamines may be considered to have slow onset of action and not been proved to improve clinical conditions. Corticosteroids may be useful to prevent late phase of anaphylactic shock.

**Prevention of Anaphylaxis**
Specific investigations should be conducted 4-6 weeks after reaction because there can be adaptation of mast cells and basophil mediators.
Skin test (Prick test, Intradermal test) are done 4-6 weeks after reaction to common allergens. NMBA, local anaesthetics, antibiotics, latex, chlorhexidine, blue dyes.
Skin test to NMBA remain positive for years.
At the end, if positive, the patient should be informed about the agent and warning card is issued and given advice for future surgeries.
General precautions – optimal control of asthma, slow administration of drugs, avoid beta blockers, ACE inhibitors or drugs which cause histamine release.
Regional anaesthesia preferred but periop anaphylaxis has been reported in these patients also.
Intraoperative anaphylaxis – patient to be monitored in ICU because reaction can be prolonged upto 3 hours and biphasic reactions occurs in 20% cases.
Introduction:
Paediatric anaesthesia is always a challenge for all anaesthesiologists owing to the differing physiology, increased sensitivity to the anaesthetic drugs and risk of toxicity and post-operative respiratory depression. In contrast to adults, the paediatric population are not candidates for local anaesthesia and almost always require general anaesthesia even for minor procedures. Although most patients are ASA-I or II and most are day care procedures, some patients present with eye problems related to congenital, chromosomal metabolic disorders or trauma.

CHALLENGES RELATED TO PHYSIOLOGY:
- During the first few weeks of life, neonates are vulnerable to a phenomenon of “flip-flop” circulation wherein the circulation can switch over from the adult to the fetal type due to factors such as hypoxia, hypercapnia, hypothermia, acidosis, infection etc., resulting in sudden increases in pulmonary artery pressure and subsequent shunting of blood past the lungs through a patent foramen ovale or the ductus arteriosus, which may reopen.
- The neonatal heart has poor tolerance to increased intravascular volume leading to biventricular failure. Also, the reduced cardiac calcium stores results in increased susceptibility to myocardial depression by the anaesthetic agents. In addition, the cardiac output in neonates is rate dependent.
- The neonatal airway differs from adult airway in 4 aspects. Larynx is located higher up in the neck, the glottis is shaped differently and angled over the inlet, the vocal cords are angled, the subglottic region (at the level of cricoid cartilage) is the narrowest portion of the larynx. Hence, straight blades are more useful than curved blades and uncuffed tubes are preferred.
- Renal functions are immature, but rapidly develop during the new born period achieveing adult levels by the age of 2 years. Particularly during the first month of birth, care should be taken in the frequency of dosing to avoid drug induced toxicity.
- Hepatic metabolic activity is also immature in the neonates. Some phase I reactions and all Phase II reactions reach maturity only by the first year of birth which is important while using drugs such as benzodiazepines and morphine.
- Former preterm infants and those who are anaemic are at increased risk of post-operative apnea.
- Investigations required are usually minimal in the form of complete blood picture routinely for all patients and an echocardiogram if there are findings of some congenital syndrome or any history of cardiac symptoms.
- Hypothermia is an important aspect to be kept in mind while handling paediatric population due to large body surface to weight ratio.
- Intraoperative fluid therapy has changed from 4-2-1 (Holliday Segar formula) to perioperative administration of 20-40ml/kg of isotonic solution (Ringers lactate). In the post-operative period, the new 2-1-0.5 rule applies (2ml/kg for the first 10 kg, 1ml/kg from 10-20 kg, 0.5ml/kg for each kg above 20 kgs).

Fasting Guidelines For Elective Procedures:
- Clear liquids – two hours
- Breast milk – four hours
- Infant formula – six hours
- Nonhuman milk – six hours
- Light meal – six hours
Any patient with history of diabetes or reflux should be advised longer fasting times.
hexachloride or perfluoropropane are introduced into the eye to tamponade detached surfaces, nitrous oxide diffuses from the blood into gas filled spaces cause a significant rise in intraocular pressure with subsequent ischemic damage. As well if nitrous oxide was used at the beginning of surgery, it will diffuse out of the bubble at the end of procedure, and the bubble will shrink which increase the incidence of recurrent detachment.

Care should be taken to observe for oculo-cardiac reflex (OCR) and oculo-respiratory reflex. A combination of ketamine and midazolam was found to be least associated with the occurrence of oculo-cardiac reflex in a recent study.

Care should be taken to avoid increases in the intraocular pressure (IOP) by preventing coughing or bucking during or after the procedure. Use of LMA or IGEL has minimal effects on IOP when compared to intubation. Lidocaine can also be used before extubation to avoid increases in IOP.

Oculo-cardiac Reflex:
It is frequently encountered in paediatric patients, particularly during strabismus surgeries. It is defined as a 10-30% decrease in heart rate from the baseline. Continuous ECG monitoring during ocular surgeries is mandatory.

Traction on extraocular muscles, particularly the medial rectus muscle can trigger the OCR resulting in sinus bradycardia, ventricular ectopics, atrial ectopics, junctional rhythm, AV blocks etc. The reflex takes its afferent innervations from the ophthalmic division of the trigeminal nerve, relays via the sensory nucleus in the 4th ventricle, with the efferent impulse in the vagus nerve.

Intravenous atropine 20 mcg/kg or glycopyrrolate 10 mcg/kg can be given during surgery if the OCR occurs. And it is imperative to have the drugs available drugs available. The reflex can be counteracted by applications of topical local anaesthetic eye drops such as tetracaine, or by blocking the afferent limb of the reflex with a peribulbar block, which is not commonly used in paediatric patients due to the risk of globe perforation.
Oculorespiratory Reflex
Extraocular muscles manipulation can also provoke oculorespiratory reflex which results in reduction in tidal volume and respiratory rate; consequent hypercapnia and hypoxemia may occur, which in turn increase the risk of OCR. Oculorespiratory reflex has the same afferent pathways as in OCR which relays in brainstem respiratory control area, and the efferent impulses travel along phrenic nerve and other nerves involved in respiration.

Table 2 shows Ophthalmic Medications & Systemic Effects:

<table>
<thead>
<tr>
<th>Eye preparations</th>
<th>Indication</th>
<th>Systemic side effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beta-blockers: Tamsulosin or Betaxolol (hydrochloride)</td>
<td>Glaucoma</td>
<td>Bradycardia refractory to atropine, bronchospasm at high doses</td>
</tr>
<tr>
<td>Carbamide anhydride inhibitors: Acetazolamide (acetate)</td>
<td>Glaucoma</td>
<td>Metabolic acidosis, electrolyte abnormalities, or other serious reactions similar to Stevens-Johnson syndrome</td>
</tr>
<tr>
<td>Antimetabolites: Cytosine arabinoside or Thiotepa</td>
<td>Pseudotumor</td>
<td>Dry mucous membranes, coma and vomiting, tachycardia</td>
</tr>
<tr>
<td>Alpha-2 agonists: Phenylephrine 2.5%</td>
<td>Pupil dilation</td>
<td>Hypertension, tachycardia</td>
</tr>
<tr>
<td>NSAIDS: Diclofenac sodium, Ketorolac tromethamine 0.5%</td>
<td>Pain relief</td>
<td>Potential to worsen or precipitate acute asthma</td>
</tr>
<tr>
<td>Local anaesthetic agents: Acrimon (Tetracaine), Oxybuprocaine, Pramoxime</td>
<td>Pain relief or prevention</td>
<td>Local anaesthetic toxicity, particularly proteinaceous</td>
</tr>
</tbody>
</table>

Post Operative Analgesia:
Optimal postoperative analgesia is crucial as pain may partly be responsible for PONV, emotional distress, and discharge delay if not properly treated. Intraoperative local anaesthetic blocks (sub-tenon) are effective in reducing PONV as well as improving post-operative analgesia when compared with intravenous opioids.

To manage mild to moderate postoperative pain which occur in most ocular procedures, topical local anaesthetic agents, analgesics such as paracetamol or NSAIDs like ketorolac, diclofenac can be given preoperatively either orally, rectally or IV at induction of anaesthesia.

More severe pain as in vitreoretinal, enucleation and squint surgeries need strong analgesics like intravenous fentanyl.

Post Operative Nausea & Vomiting:
Nausea alone is difficult to be determined in children who usually had a higher incidence of PONV when compared to adults. Postoperative vomiting is very common in children after strabismus surgeries, especially in children over the age of 2 years. About two-thirds of children vomit after strabismus surgery, if no preventive measures are taken. Prophylactic strategies to avoid postoperative vomiting included the use of anticholinergic agents, dexamethasone, dimenhydrinate, anti-emetics - ondansetron or using the anti-emetic properties of propofol can be used. However, 5-HT3 (serotonin) antagonists has led to marked reduction in the incidence of PONV, if given intraoperatively. Ondansetron 0.1 mg/kg is very effective and smaller doses have been proved to be equally effective. Combination therapy (e.g. ondansetron and dexamethasone) is better than ondansetron alone. Recently a study demonstrated that an intraoperative lactated ringer’s solution at 50 ml/kg/hr is more effective in reducing PONV in strabismus surgery patients than a solution at 10 ml/kg/hr.

Conclusion:
Anaesthesia for Ophthalmology itself is a challenging field because most of them are day care procedure without prior evaluation. In addition to these challenges, paediatric patients coming for ophthalmologic procedures pose an even bigger challenge due to the physiological aspects discussed above. It is the need of the hour for introducing further updates in this field to keep the children more comfortable during the pre-, intra-, and post-operative period.

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Interactive Sessions
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Panel Discussions

REGISTRATION DETAILS

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IMPORTANT PRESENTATION-TOPICS

- Locoregional ophthalmic anaesthesia teaching safer blocks for the 21st century
- Use of Ultrasound to make locoregional anaesthesia safer
- Anatomy of ophthalmic regional blocks in humans, animals & teaching models
- The Trigemino-cardiac reflex
- Opioid-free & Multimodal General Anaesthesia
- Panel-discussion: Teaching a new generation of clinicians in OA
- Perioperative EEG as guidance for „GA-depth“-going beyond the BIS-index
- Intra-ocular pressure and retinal haemodynamics in GA & LA
- Sub-Tenon-blocks in treatment of retinopathy-of-premature babies
- New trends in pediatric OA (intra-nasal dexmedetomidine, i.v.-access etc)
  - OA in ophthalmic oncology surgery
  - Surgery and anaesthesia in ocular trauma
  - Use of hypnosedation in ophthalmic surgery
  - Anaesthesia-concepts for oculoplastic surgery

PRACTICAL IN-DEPTH, HANDS-ON AFTERNOON COURSES

- Locoregional ophthalmic anaesthesia
- Sub-Tenon’s and peribulbar blocks, lateral canthotomy
- Sonographic exam of the eye before and during ophthalmic regional anaesthesia
- Perioperative Electroencephalography
- Basic concepts of clinical hypnosis for ophthalmic anaesthesia

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2. There is no upper age limit for eye donation
3. Persons wearing spectacles, who have undergone cataract surgeries, treatment for diabetes and blood pressure can also donate eyes
4. If you donate your eyes, you give vision to two corneal blind people

Editors:  Dr. Jagadeesh V
Dr. Jaichandran V V
Dr. Kannan R

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