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Controversies in anaesthesia: anaesthesia technique and trabeculectomy failure

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Can anaesthesia technique influence the outcome of surgery? A good anaesthetic will provide optimum conditions for the surgeon, thereby reducing the likelihood of per-operative complications. But can the choice of anaesthetic have a more lasting effect on surgical outcome? In glaucoma surgery, the answer may be yes, though this is still a matter of controversy.

This issue was raised in a 1993 paper from Moorfields Eye Hospital. Sub-conjunctival anaesthesia (SCA) was being used for more and more trabeculectomies, until one of the clinicians noticed what appeared to be a high incidence of avascular, leaky blebs with this technique. A chronically leaking bleb is undesirable as it can lead to endophthalmitis or hypotony, each of which is potentially devastating to the vision. A review of cases showed that the prevalence of bleb leakage was indeed higher in the SCA group, though pressure control was good in both groups. At around one year post-operatively, bleb leak was seen in 77% for patients who had had SCA (2ml of 2% lidocaine), as opposed to 25% for those who had GA for their trabeculectomy surgery. The authors speculated that the LA might exert an inhibitory effect on conjunctival healing.

A large prospective study of trabeculectomy also concluded that sub-conjunctival anaesthesia may be undesirable, but for completely different reasons. The 1997 National Audit of Trabeculectomy looked prospectively at 1454 cases of primary trabeculectomy, done under the care of 382 UK consultant ophthalmologists. Only a small proportion of trabeculectomies (38/1454, 2.6%) were done using SCA. Data was collected for numerous aspects of the surgery, and follow-up was done at one year. Late bleb leakage was less than 3% in all groups. Sub-analysis suggested that trabeculectomy failure (defined in terms of intra-ocular pressure reduction) was more prevalent in the SCA group. It appeared that 'success' rates were only 39.5% in the SCA group, as opposed to 65-70% for GA or other LA techniques.

So where does this leave us? One study suggests that SCA leads to a very high likelihood of chronic leakage, but good pressure control. The other suggests that chronic leakage should not be a problem with any anaesthesia technique, but found poorer success rates when SCA was used. It must be remembered that the Moorfields study was retrospective and non-randomised, and the National Audit of Trabeculectomy was also a non-randomised observational study with relatively few SCA cases, and therefore other confounding factors could easily explain the apparent low success rates with SCA. The National

Audit investigators suggested a prospective randomised trial to look at whether SCA itself is a risk factor for failure. Studies on skin wounds have shown that infiltration with 1% or 2% lidocaine will significantly weaken a scar, whereas 0.5% lidocaine has little or no effect. Thus, it is possible that sub-conjunctival lidocaine has a dose-dependent anti-scarring effect on the conjunctiva, similar to that of the anti-metabolites that are frequently used to enhance success rates for trabeculectomy.

Many glaucoma specialists have been using SCA successfully for some years. My own practice is to use a low dose SCA, as 0.5ml 0.5% lidocaine SCA supplemented with intra-cameral lidocaine, for virtually all trabeculectomy surgery. I use this lower strength because of concerns about a possible detrimental effect of lidocaine on wound healing. Clinical audit has shown that the technique is highly acceptable to patients. Success rates are significantly better than the National Audit of Trabeculectomy, with no leakage at one year and virtually identical success rates to those of my colleague who routinely uses peribulbar LA. There is a suggestion of a higher rate of early bleb leakage, but this may be due to surgical technique rather than anaesthesia per se. Sub-conjunctival anaesthesia (SCA) does not appear to increase the risk of trabeculectomy failure or late bleb leakage. More work is needed to look into a likely dose-dependent 'anti-scarring' effect of lidocaine on the conjunctiva. An interesting possibility is that lidocaine may be as good as cytotoxics in modulating wound healing after trabeculectomy surgery.

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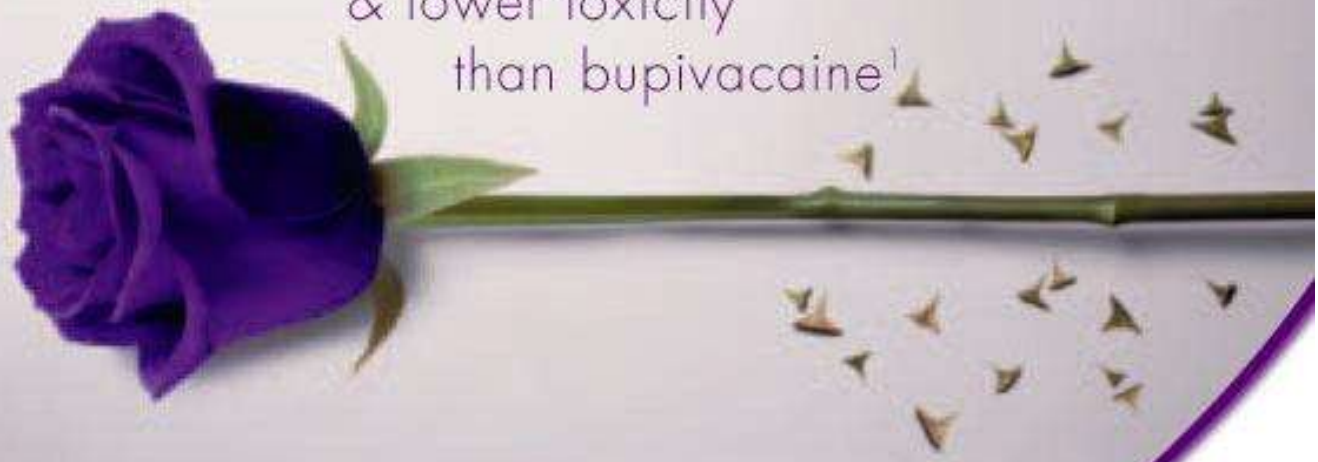

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Chirocaine (Levobupivacaine Hydrochloride) Prescribing Information. Presentation: Three strengths are available, 2.5 mg/ml, 5.0 mg/ml and 7.5 mg/ml of levobupivacaine as levobupivacaine hydrochloride. Each strength is available in 10ml polypropylene ampoules, in packs of 10. **Indications: Adults:** Surgical anaesthesia - Major, e.g. epidural (including for Caesarean section), intrathecal, peripheral nerve block. - Minor, e.g. local infiltration, peribulbar block in ophthalmic surgery. Pain management - Continuous epidural infusion, single or multiple bolus epidural administration for the management of pain especially post-operative pain or labour analgesia. **Children:** analgesia (Neurogenic/Analgesic/Postoperative Effects). **Dose and Administration:** The precise posology, and dispersal upon the procedure and individual patient concerned. Careful aspiration before and during injection is recommended to prevent intravascular injection. Where a large dose is to be injected, e.g. in epidural block, a test dose of 3-5 ml lidocaine (lignocaine) with adrenaline is recommended. An inadvertent intravascular injection may then be recognised by a temporary increase in heart rate and accidental intrathecal injection by signs of a spinal block. Aspirations should be repeated before and during administration of a bolus dose, which should be injected slowly and in incremental doses, at a rate of 75 - 30 mg/min, whilst closely observing the patient's vital functions and maintaining verbal contact. The recommended maximum single dose is 150 mg. The maximum recommended dose during a 24-hour period is 400 mg. For post-operative pain management, the dose should not exceed 18.75 mg/hour. For Caesarean section, higher concentrations than the 5.0 mg/ml solution should not be used. For labour analgesia by epidural infusion, the dose should not exceed 12.5 mg/hour. In children, the maximum recommended dose for analgesia (Neurogenic/Analgesic/Postoperative Effects) is 1.25 mg/kg/side. **Contra-indications:** Patients with a known hypersensitivity to local anaesthetic agents of the amide type; intravenous regional anaesthesia (Bier's block); patients with severe hypotension such as cardiogenic or hypovolaemic shock; and use in paracervical block in obstetrics. The 7.5 mg/ml solution is contraindicated for obstetric use due to an enhanced risk for cardiotoxic events based on experience with bupivacaine. There is no experience of levobupivacaine 7.5 mg/ml in obstetric surgery. **Precautions:** Epidural anaesthesia with any local anaesthetic may cause hypotension and bradycardia. All patients must have intravenous access established. The availability of appropriate fluids, vasopressors, anaesthetics with cardiovascular properties, oxygen/carbon, ventilation equipment and expertise must be ensured. Levobupivacaine should be used with caution for regional anaesthesia in patients with impaired cardiovascular function e.g. severe cardiac arrhythmias, and in patients with liver disease or with reduced liver blood flow e.g. alcoholics, or cirrhotics. **Interactions:** Metabolism of levobupivacaine may be affected by CYP3A4 inhibitors, eg: ketoconazole and CYP1A2 inhibitors, eg: enoxifen/cardiazac. Levobupivacaine should be used with caution in patients receiving cardiac arrhythmic agents with local anaesthetic activity, e.g. mexiletine, or Class III anti-arrhythmic agents since their toxic effects may be additive. No clinical studies have been completed to assess levobupivacaine in combination with adrenaline. **Side-Effects:** Adverse reactions with local anaesthetics of the amide type are rare, but they may occur as a result of overdosage or unintentional intravascular injections and may be serious. Accidental intrathecal injection of local anaesthetics can lead to very high spinal anaesthesia possibly with apnoea, severe hypotension and loss of consciousness. The most frequent adverse events reported in clinical trials irrespective of causality include hypotension (22%), nausea (13%), anaemia (11%), post-operative pain (8%), vomiting (8%), back pain (7%), fever (6%), dizziness (6%), frontal dizziness (6%) and headache (5%). Other side effects include: CNS effects: numbness of the tongue, light headedness, dizziness, blurred vision and convulsions followed by drowsiness, convulsions, unconsciousness and possible respiratory arrest. CVS effects: decreased cardiac output, hypotension and ECG changes; indicative of either heart block, bradycardia or ventricular tachyarrhythmias that may lead to cardiac arrest. Neurological damage is a rare but well recognised consequence of regional and particularly epidural and spinal anaesthesia. This may result in localised areas of paraesthesia or anaesthesia, motor weakness, loss of sphincter control and paraplegia. Rarely, these may be permanent. **Use in Pregnancy and Lactation:** Levobupivacaine should not be used during early pregnancy unless clearly necessary. The clinical experience of local anaesthetics of the amide type including bupivacaine for obstetrical surgery is extensive. The safety profile of each one is considered adequately known. There are no data available on excretion of levobupivacaine into human breast milk. However, levobupivacaine is likely to be transmitted in the mother's milk, but the risk of affecting the child at therapeutic doses is minimal. **Overdose:** Accidental intravascular injection of local anaesthetics may cause immediate toxic reactions. In the event of overdose, peak plasma concentrations may not be reached until 2 hours after administration depending upon the injection site used, therefore, signs of toxicity may be delayed. Systemic adverse reactions following overdose or accidental intravascular injection presented with being acting local anaesthetic agents involve both various CNS and CVS effects. **Special Storage Conditions:** No special storage precautions for the glass ampoules. Once opened, use immediately. **Legal Category:** POM. **Marketing Authorisation Number:** PL 0032/0308-0310. **Basic NHS Price:** 2.5 mg/ml pack: £16.60, 5.0 mg/ml pack: £19.30, 7.5 mg/ml pack: £28.50. Further information is available on request from Abbott Laboratories Ltd, Abbott House, Norden Road, Maidenhead, Berkshire SL6 4XE. PI/PD/1/005. **Date of Preparation:** March 2004. **Reference:**

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Administration: Deliver via a vaporiser specifically calibrated for use with sevoflurane. Induction can be achieved and maintenance sustained in oxygen or oxygen-nitrous oxide mixtures.
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Precautions: For use only by trained anaesthetists. Hypotension and respiratory depression increase as anaesthesia is deepened. Malignant hyperthermia. Experience with repeat exposure is very limited. Until further data are obtained, sevoflurane should be used with caution in patients with renal insufficiency. Levels of Compound A (produced by direct contact with CO_2 absorbents) increase with: increase in canister temperature; increase in anaesthetic concentration; decrease in gas flow rate and increase more with the use of Baralyme rather than soda lime.
The exothermic reaction that occurs with inhalational agents, including Sevoflurane and CO_2 absorbents, is increased when the CO_2 absorbent becomes desiccated (dried out).
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Side-Effects: Dose-dependent cardio-respiratory depression. The type, severity and frequency of adverse events are comparable to those seen with other inhalation anaesthetics. Most adverse events are mild to moderate and transient: nausea, vomiting, increased cough, hypotension, agitation and bradycardia. Hepatitis has been reported rarely. Convulsions may occur extremely rarely, particularly in children. Rare reports of allergic reactions, such as rash, urticaria, pruritus, bronchospasm, anaphylactic or anaphylactoid reactions have been reported. There have been very rare reports of pulmonary oedema. As with other anaesthetics, twitching and jerking movements, with spontaneous resolution have been reported in children during induction. Patients should not be allowed to drive for a suitable period after sevoflurane anaesthesia.
Use in Pregnancy and Lactation: Use during pregnancy only if clearly needed. It is not known whether sevoflurane is excreted in human milk – caution in nursing women.
Overdose: Stop sevoflurane administration, establish a clear airway and initiate assisted or controlled ventilation with pure oxygen and maintain adequate cardiovascular function.
Special Storage Conditions: Do not store above 25°C. Do not refrigerate. Keep cap tightly closed.
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Further information is available on request from Abbott Laboratories Ltd., Abbott House, Norden Road, Maidenhead, Berkshire SL6 4XE. **Ref:** PI/12/009.

Agents for sedation in ophthalmic surgery: a review of the pharmacodynamics and clinical applications

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Abstract

Sedation is often required to improve patient comfort during the placement of local blocks in eye surgery. The ideal sedative should have a rapid onset of action and be able to ensure immobility while allowing for patients to respond to verbal commands. Furthermore, ideal sedatives should provide amnesia, have a sufficiently short duration of action to facilitate patient cooperation during surgery, have minimal side effects and allow for a rapid return to home-readiness. Combined analgesic and sedative regimes are widely used in tandem with local/regional blocks, however, potential problems with sedative/narcotic agents are considerable. These complications include ventilatory depression and loss of airway control with hypoxia and hypercapnia, and may cause the patient to become confused intraoperatively.
The role of sedation in a wide variety of ophthalmic procedures as well as the variation in sedative agents used with regards to dosage regimes and drug combinations as applied in clinical practice can only be illustrated by referring to the published data. As a result, the goal of this paper is to examine the spectrum of sedative drugs that have been used in the process of eye surgery as published in the literature since 1990 with special attention being given to the pharmacodynamics and clinical applications of relevant drugs in current use.
In conclusion there does not seem to be one drug or one regime indicative of standardization of sedation practice in eye surgery. It rather seems as if there could be a competition between drugs and regimes for the position of the best and most favourable place in the armamentarium of sedationists and anaesthesiologists. There could be strong indications from the literature that drugs like midazolam, propofol and remifentanil are the favourites for sedation in eye surgery. It might be necessary to take another look at the group of alpha-2 adrenergic agonists and also perhaps at low-dose ketamine.

Key words: sedation agents, ophthalmic surgery, pharmacology, dosage regimes, clinical application.

"performing painless eye blocks without sedation is probably the safest route"
BP Gallacher [1].

"Although many ophthalmic procedures can be performed on the standing horse, the decision to perform a procedure under sedation rather than general anesthesia must be made on the basis of the temperament of the horse, severity of the injury, and skill of the veterinarian." DA Wilkie [2].

Introduction

Sedation is often required to improve patient comfort during the placement of local blocks in eye surgery. The ideal sedative should have a rapid onset of action and be able to ensure immobility while allowing for patients to respond to verbal commands. Furthermore, ideal sedatives should provide amnesia, have a sufficiently short duration of action to facilitate patient cooperation during surgery, have minimal side effects and allow for a rapid return to home-readiness. Drugs used for sedation can be divided into two broad categories, namely anxiolytic and sedative-hypnotics. Sedative hypnotics, as a class, can be further subdivided into benzodiazepines, barbiturates, antihistaminics, narcotic analgesics, and intravenous anaesthetics. Combined analgesic and sedative regimes are widely used in tandem with local/regional blocks; however, potential problems with sedative/narcotic agents are considerable. These complications include ventilatory depression and loss of airway control with hypoxia and hypercapnia, and may cause the patient to become confused intraoperatively.
The role of sedation in a wide variety of ophthalmic procedures as well as the variation in sedative agents used with regards to dosage regimes and drug combinations as applied in

clinical practice can only be illustrated by referring to the published data. As a result, the goal of this paper is to examine the spectrum of sedative drugs that have been used in the process of eye surgery as published in the literature since 1990 with special attention being given to the pharmacodynamics and clinical applications of relevant drugs in current use, namely propofol, midazolam, alfentanil, fentanyl, remifentanil, ketamine, diazepam, lorazepam, methohexital, piritramide and clonidine/dexmedetomidine. For sedation in combination with eye blocks, the literature presents a multitude of combinations that are used in clinical practice viz; ketamine/alfentanil; alfentanil/midazolam; propofol/ketamine; propofol/remifentanil; propofol/alfentanil; propofol/fentanyl/alfentanil; propofol/diazepam; propofol/midazolam; alfentanil/droperidol; fentanyl/droperidol; fentanyl/diazepam.

Combinations of drugs can be used to sedate patients in order to limit movement during the block and to provide a cooperative, alert and haemodynamically stable patient during the operative procedure. Excessive dosage of these drugs however, may result in hazardous respiratory depression in this patient population particularly in the higher dose ranges.

SEDATIVE AGENTS: PHARMACOLOGY, DOSAGE REGIMES AND CLINICAL APPLICATION

Benzodiazepines

Midazolam, diazepam and lorazepam are the most commonly used benzopdiazepines associated with eye surgery and local eye blocks. Midazolam is the most popular mainly due to its high lipid solubility, which results in a fast onset of action as well as its rapid rate of clearance, while diazepam and lorazepam have largely lost favour for this indication. Diazepam, due to its slow clearance, is associated with delayed recovery particularly in large doses while, relative to midazolam, lorazepam has a comparatively slower onset of action due to its lesser degree of lipid solubility.

Pharmacology:

The most widely accepted hypothesis for the mechanism of action of benzodiazepines is that gamma aminobutyric acid (GABA) receptors and benzodiazepine receptors are coupled to a chloride channel. When both receptors are occupied, membrane hyperpolarization and neuronal inhibition occurs. As result, benzodiazepines have been shown to exhibit anticonvulsant, anxiolytic and muscle relaxant activity. The action of the benzodiazepines is readily reversed by flumazenil, a benzodiazepine receptor antagonist, making the benzodiazepines the only class of sedatives that can be reversed by a specific antagonist.

Midazolam

Pharmacokinetics and pharmacodynamics

Midazolam, a water soluble bezodiazepine, has a wide margin of safety and a high therapeutic index. The elimination half-life of midazolam ranges between 2 to 5 hours while the duration of action varies between 1.5 to 3.5 hours - shorter than most other benzodiazepines. Although the half-life of a given agent does not predict the duration of its sedative effect after a single, pre-operative dose it is important to remember that the elimination half-life is prolonged in patients with cardiac, hepatic and renal impairment. This, coupled with absence of long-acting active metabolites, makes midazolam a suitable short-acting sedative agent that produces minimal to no residual psychomotor impairment 8 hours following oral doses and 3 to 4 hours following intravenous (IV) administration. Other advantages include minimal pain or local intolerance following intramuscular (IM) and IV injection (as opposed to diazepam) due to its water solubility. Furthermore, in contrast to diazepam, midazolam is almost completely absorbed following IM administration

Dose

Usual doses for the induction of anaesthesia range between 0.15 to 0.3 milligrams/kilogram (mg/kg) given intravenously. For sedation, however, the recommended dose of midazolam is 0.04 mg/kg but this should only be taken as a guide as patient response varies considerably. Subsequent incremental doses should be at doses of 0.015mg/kg. It is suggested that midazolam should be injected at a rate of 2mg/min with the drug taking effect within approximately 1 to 1.5 minutes following IV administration.

Midazolam has been associated with both respiratory depression and respiratory arrest most often when combined with other central nervous system (CNS) depressants. Elderly patients and patients who have taken alcohol are more sensitive to this drug and it is recommended to

reduce the maximum total dose by 30% in patients over 60 years of age or debilitated patients. Pharmacodynamic studies have consistently shown that from the ages of 20 to 80 years the sensitivity of the brain towards midazolam increases by at least 75%. As a result, a bolus dose of midazolam given to an 80 year old patient should be reduced to 25% of that intended for use in a patient who is 20 years old [3]. In cases when midazolam is intended to be used in combination with an opiate, it should be borne in mind that only 25% of the usual recommended dose of each agent should be administered, giving opiate first followed by a titration of the midazolam. [4]. Metabolites of midazolam have substantial pharmacological activity and can accumulate in patients with renal failure and in patients with reduced CYP3A4/5 activity, while clearance is reported to be 30% lower in patients suffering from congestive heart failure [5].

The mean dose of midazolam given alone was 1.82 ± 1.09mg. The dose given with opioids was 1.44 ± 0.84 in 19,250 for cataract operations in nine centers [6].

Clinical application

Wong [7] studied the use of midazolam and alfentanil separately, or in combination in elderly patients undergoing cataract surgery under regional anaesthesia. The average age of 120 patients within this study was 73 years. Patients were randomized to receive one of normal saline, 1mg midazolam, 500µg alfentanil, or 0.5mg midazolam plus 250µg alfentanil. The midazolam-alfentanyl combination was shown to reduce pain perception while all IV sedation used reduced pain recall. Midazolam reduced systolic blood pressure and alfentanil reduced oxygen saturation with or without midazolam. The authors concluded that, firstly, the use of fine needles combined with a slow infusion rate of anaesthetic solution caused minimal discomfort and secondly, that the routine IV sedation may be unnecessary.

Diazepam

Pharmacokinetics

The onset of clinical effect following the oral administration of diazepam occurs within 30 minutes. Both oral and intramuscular absorption is rapid with peak levels occurring within 1 hour. Rectal absorption is also rapid with adequate serum levels occurring within 10 minutes and finally intravenous use produces peaks within 8 minutes. The elimination of diazepam is biphasic with an initial half-life of 7 to 10 hours, followed by a second half-life ranging from 2 to 6 days. Extensive hepatic metabolism occurs to active metabolites with less than 25% of the drug being excreted unchanged in the urine.

Clinical application

In a study conducted by Hampl and colleagues [8], diazepam was compared with propofol as intravenous sedation for retrobulbar block and eye surgery.

The combination of diazepam and propofol resulted in the highest comfort scores for both retrobulbar block and surgery. Diazepam alone did not produce adequate sedation for retrobulbar block and propofol alone did not provide any sedation during surgery.

Lorazepam

Lorazepam is a benzodiazepine derivative in clinical use as a sedative-hypnotic, anticonvulsant, anxiolytic, and muscle relaxant. Usual oral doses range from 2 to 6 mg daily, divided into 2 to 3 doses; parenteral doses of 0.05 mg/kg are recommended prior to surgical procedures. Paediatric dosing for surgical premedication has been 0.05mg/kg either parenterally or orally. In some cases, lorazepam administered by the sublingual route may result in a faster onset of therapeutic effect than orally administered lorazepam. Sublingual administration of lorazepam also compares favourably in time to onset with intramuscular injection of the drug.

Pharmacokinetics

Adequate absorption occurs with oral or intramuscular doses; onset of hypnosis is 20 to 30 minutes and the duration is 8 hours; protein binding is 85% to 91%; elimination half-life is 10 to 16 hours; hepatic metabolism to inactive metabolites occurs, with renal excretion and minimal faecal excretion.

Adverse effects include sedation, dizziness, vertigo, delirium, disorientation, agitation, hepatotoxicity, respiratory depression, and withdrawal symptoms.

Clinical application:

Lorazepam is an effective anti-anxiety and hypnotic agent for insomnia and as premedication to surgical procedures. The drug is also indicated parenterally for status epilepticus.

In eye surgery lorazepam in a dose range of 0.5 to1.0 mg, particularly in the elderly, will be used sublingually as an axiolytic.

Propofol

Propofol is an intravenous sedative hypnotic useful in the induction and maintenance of general anaesthesia in adults and children and for sedation during monitored anaesthesia care (MAC). Propofol is a hindered phenolic compound with intravenous general anaesthetic properties and is unrelated to any of the barbiturate, opioid, benzodiazepine, arylcyclohexylamine, or imidazole intravenous anaesthetic agents that are used currently. Propofol enjoys wide popularity as an agent for sedation in eye surgery evident from the many clinical applications referred to in the published literature. Low doses of propofol tend to lack analgesic properties and produce amnesia that is unreliable. As a result, this agent is often used in combination with other preparations.

Pharmacokinetics

Onset of anaesthesia usually occurs within 30 seconds of the end of the bolus infusion and the duration of effect last for approximately 3 to 10 minutes depending on the dose and the rate of administration. Propofol is highly lipophilic, has a large volume of distribution and is 97% to 99% protein bound. The initial distribution half-life in surgical and healthy volunteers ranges from 1 to 8 minutes. The half-life of the initial elimination phase is approximately 40 minutes, while the terminal elimination phase half-life is approximately 200 minutes. Mean recovery from anaesthesia (defined as the time when the patient could obey verbal commands) was 3, 6, and 8 minutes following propofol doses of 1, 2, and 3 mg/kg, respectively [5]. The most distinguishing property of propofol relates to its pattern of clearance. It is rapidly bio transformed into inactive metabolites not only in the liver but also in multiple tissue sites yet to be clearly defined, which provides for a rapid recovery even after prolonged administration. Propofol is well suited for sedation and at these low doses adverse respiratory and cardiovascular influences are minimal. Propofol also has notable antiemetic efficacy and nausea and vomiting are less frequent when propofol is a component of the anaesthetic regimen.

Pain on injection is listed as one of the most common adverse effects associated with propofol particularly when the drug is administered via the small veins on the dorsum of the hand or wrist. Apnoea, a reduction in blood pressure, and CNS effects are also relatively common with propofol anaesthesia. The lipid based formulation of propofol has a number of undesirable properties which include the pain on injection mentioned above as well as the possibility of serious allergic reactions, and rapid microbial growth with inadvertent contamination. Propofol also appears to increases the incidence of sneezing and might happen during placement of the block [9].

Dose

The adult induction dose of propofol is usually 2 to 2.5mg/kg. For maintenance of anaesthesia, continuous propofol infusions of 6 to 12 mg/kg/hour (0.1 to 0.2 mg/kg/min) have been used. For monitored anaesthesia care also called conscious sedation, most patients require an infusion of 6 to 9 mg/kg/hr, with maintenance rates of 0.3 to 3 mg/kg/hr. Higher maintenance doses may be required for ICU sedation.

Clinical application

It has been shown that propofol decreases intraocular pressure where a propofol bolus of 0.5mg/kg followed by a low-dose continuous infusion of 0.5mg/kg/hr in patients undergoing trabeculectomy under peribulbar block. Ocular pressure decreased 2 minutes after the start of the propofol infusion and remained significantly lower than in the control group [10]. Neel and co-authors published another study which examined the effects on intraocular pressure during single low dose (0.98mg/kg SEM 0.04) intravenous sedation with propofol before cataract surgery. A decrease in intraocular pressure of between 17 and 27%, with minimal side effects, was shown after sedation with intravenous propofol [11]. It was, however, shown that sedation techniques with other agents e.g. fentanyl/droperidol and alfentanil/droperidol combinations caused a similar reduction in intra-ocular pressure [12]. The reduction of intra-ocular pressure is therefore not unique to propofol sedation but more likely due to the sedation per se.

Rewari, et al [13] studied remifentanil and propofol for analgesia and sedation during placement of retrobulbar block. Four groups of patients were randomized to receive either remifentanil 1µg/kg, remifentanil 0.5µg/kg and propofol 0.5mg/kg, remifentanil 1µg/kg and propofol 0.5 mg/kg or

saline 0.1ml/kg. No pain was observed during the placement of the block in any of the groups except the control group. Respiratory depression was observed to the greatest degree in patients receiving remifentanil 1µg/kg groups. The combination of remifentanil 0.5µg/kg with propofol 0.5mg/kg as a bolus was considered to provide excellent relief of pain and anxiety with least number of adverse effects being associated with the placement of the block.
The efficacy and safety of remifentanil, propofol or both for conscious sedation during eye surgery under retrobulbar block was compared in a prospective randomized study by Holas and colleagues [14].
In another study by Yee, et al [15] propofol and the addition of alfentanil were compared for sedation during placement of retrobulbar block for cataract surgery.
Sedation quality and recovery profiles in patients who received propofol or propofol-ketamine sedation during placement of retrobulbar block were compared in a study by Frey et al [16].
Propofol and ketamine was also used in a study by Senn, et al [17] who believed that deep sedation is needed while injecting the local anaesthetic for the eye block.

Opioid analgesics/narcotics
In this group of agents, fentanyl, remifentanil, alfentanil and piritrimide have been used in sedation for periorbital blocks and eye surgery. Most of these agents have been referred to in combination with propofol and /or midazolam [7, 13, 14, 18-26].
Opioids are generally included in anaesthetic regimens to provide analgesia and offset sympathetic responses to surgical stimuli. Opioids have relatively little effect on consciousness but can be used alone or in combination with other sedative agents to improve tolerance and the level of sedation during the placement of local eye blocks.
The most common side effects with all opioids are respiratory depression and hypotension within the context of sedation for eye blocks.

Remifentanil
Remifentanil is an ultrashort-acting opioid analgesic for use during surgical anaesthesia. It is selective for mu receptors and exhibits typical opioid pharmacologic effects, including analgesia, respiratory depression, sedation, hypertonus of skeletal muscle, and bradycardia. Acute effects are competitively antagonized by naloxone.
Remifentanil is structurally related to alfentanil, but is 20 to 50 times more potent and has a shorter elimination half-life. In surgical anaesthesia, remifentanil has been given as an intravenous bolus of 1 microgram/kilogram (µg/kg) followed by a continuous infusion of 0.1 to 0.8 µg/kg/minute; the duration of opioid effects is short, requiring immediate supplemental postoperative analgesia. Dose reductions are not required in renal or hepatic impairment.
Pharmacokinetics
Remifentanil is characterized by a rapid onset of analgesia and a very short duration of action (3 to 10 minutes); the time to a 50% reduction in the effective plasma concentration of the drug is about 3.5 minutes, significantly less than that of alfentanil. The brief duration of action of remifentanil is attributed to its rapid hydrolysis by blood and tissue esterases, to essentially inactive metabolites. Most of an intravenous dose is excreted in the urine as the carboxylic acid metabolite.
Remifentanil has an elimination half-life of 8 to 20 minutes, shorter than that of alfentanil. Unlike alfentanil, which is metabolized in the liver, remifentanil contains an ester linkage in its structure and is susceptible to rapid hydrolysis in tissues and blood by nonspecific esterases to essentially inactive metabolites.
Adverse effects of remifentanil resemble those of other opioid analgesics, and include hypotension, bradycardia, central nervous system effects (sedation, dizziness, euphoria), nausea, vomiting, muscle rigidity, skin rash, and respiratory depression; however, due to its rapid clearance, pharmacologic reversal should not be required.
Clinical application
Remifentanil is indicated as an analgesic component for the induction and maintenance of general anaesthesia for inpatient and outpatient procedures, and as an analgesic into the immediate postoperative period. It is also indicated as an analgesic component of monitored anaesthesia care (MAC). Remifentanil is shorter-acting than alfentanil and advantages of remifentanil are easy titration of intraoperative opioid effects, no accumulation during prolonged infusion, minimal risk of significant postoperative respiratory depression, and lack of dependence upon hepatic metabolism.

Simply waiting for opioid effects of remifentanil to subside appears sufficient for reversal of effects. Discontinuing the infusion of remifentanil was more effective than administration of naloxone [5].

Remifentanil has been used extensively in combinations with other agents for sedation during placement of local anaesthetic blocks. The place of remifentanil in the clinical application with reference to eye blocks has been defined in very good studies referred to in this article [9, 13, 14, 23].

Remifentanil has most often been used in doses ranging from; 0.03 ± 0.01µg/kg to 1.0µg/kg.

Fentanyl

Fentanyl citrate is a narcotic analgesic with pharmacologic effects similar to morphine and meperidine. Fentanyl is 50 to 100 times more potent than morphine on a weight basis; fentanyl 0.1 mg is approximately equivalent in analgesic activity to morphine 10 mg or meperidine 75 mg. Fentanyl is a mu-receptor agonist and interacts with receptors distributed in the brain, spinal cord, and other tissues.

Phamacokinetics

Fentanyl is a potent narcotic analgesic and intramuscular doses of 50 to 100 µg are effective as a premedicant and adjunct to regional anaesthesia.

Fentanyl produces analgesia almost immediately following IV, lozenge/sucker, and oral transmucosal use; the drug is metabolized in the liver and excreted in the urine primarily as metabolites (less than 7% unchanged drug); the half-life of fentanyl is 2 to 7 hours.

Fentanyl is metabolized primarily via human cytochrome P450 3A4 isoenzyme system. In humans the drug appears to be metabolized primarily by N-dealkylation to norfentanyl and other inactive metabolites that do not contribute materially to the observed activity of the drug.

The kinetics of fentanyl in geriatric patients has not been well studied, but in geriatric patients the clearance of intravenous fentanyl may be reduced.

Acute effects can however, be competitively antagonized by naloxone.

The most common side effects with all opioids are respiratory depression, apnoea, circulatory depression, hypotension, and shock. Muscle rigidity and chest wall spasm can occur following rapid intravenous administration and bradycardia, seizures and delirium have occurred. Hypoventilation may occur throughout the therapeutic range of fentanyl.

Clinical application

Parenteral fentanyl is indicated for anaesthesia, treating postoperative pain, and as a premedicant. Transdermal fentanyl is used for managing chronic pain in patients requiring opioids. Fentanyl lozenge/sucker (Oralet®) is indicated to induce anxiolysis and analgesia prior to surgery in paediatric and adult patients but is only indicated for use in a hospital setting as an anaesthetic premedication in the operating room area or to induce conscious sedation prior to a diagnostic or therapeutic procedure in other monitored anaesthesia care settings in the hospital [5].

The subjective and psychomotor-impairing effects of intravenous fentanyl were examined in a group of healthy volunteers [25]. Subjects were injected with 0, 25, 50 and 100 µg/70kg body weight. Some aspects of psychomotor functioning e.g. eye-hand coordination, were impaired by fentanyl and fentanyl produced dose-related increases in ratings of "high" and "sedated" but also tended to produce dysphoria and somatic symptomatology. Most subjects reported "liking" the effects of the two higher doses of fentanyl for at least a brief time after injection, but they varied widely in their "liking" ratings in the post drug injection period. Fentanyl did not score high in drug-induced euphoria.

The relative potencies of fentanyl and alfentanil for respiratory depression were determined in healthy volunteers. Both drugs decreased ventilation in a similar manner. The potency ratio for alfentanil: fentanyl is 1:45 with parallel analgesic and respiratory depressant effects. Equianalgesic concentrations of both drugs will lead to equally pronounced respiratory depression [26].

Alfentanil

Alfentanil is a tetrazole derivative of fentanyl. The drug is a member of the 4-anilinopiperidine class of opiate agonists. Alfentanil is an opioid analgesic used in anaesthesia, analgesia, and sedation.

The dosage should be individualized in each patient; for surgeries lasting less than 1 hour, typical induction intravenous doses range from 8 to 50 µg/kg /kg, followed by maintenance injections ranging from 3 to 15 µg/kg /kg or

continuous infusions of 0.5 to 1 µg/kg/min. Alfentanil 30 µg /kg IM is an effective analgesic. With reference to its clinical use alone or in combination with other sedative agents for sedation during eye blocks the dose ranges were from 5µg/kg to 15µg/kg.

Pharmacokinetics

The half-life ranges between 46 to 213 minutes; protein binding is 82 to 95%; clearance is 1.6 to 17.6 mL/min/kg; and volume of distribution is 0.6 to 2.5 L/kg. The onset of anaesthesia is two minutes, and the duration of analgesia with a single intravenous bolus is 10 minutes. Alfentanil is oxidatively metabolized in the liver and the inactive metabolites are excreted renally. Following single intravenous bolus injections of alfentanil, 50 or 125µg/kg, a 3-compartment model was described with a redistribution half-life of 4.6 to 21.6 minutes.

Duration of analgesia for alfentanil is approximately 10 minutes following a single intravenous bolus dose. An intramuscular dose of alfentanil 30 mcg/kg administered to six healthy volunteers had duration of action of about 1 hour. Most common adverse effects include muscle rigidity in 50 to 99% of patients; nausea (28%), vomiting (18%), bradycardia (14%), tachycardia (12%), hypotension (10%) and hypertension (18%) have been reported.

The effects of low bolus dose alfentanil, 10µg/kg, on central respiratory drive and respiratory pattern in spontaneously breathing ASA 1 patients were assessed in 17 patients for minor surgery. Alfentanil produced a significant reduction in arterial oxygen saturation and minute volume which needed careful monitoring of cardio-respiratory function [27].

Clinical applications

Alfentanil is a useful sedative and analgesic. Alfentanil has been used in a variety of combinations with other agents for sedation of patients undergoing eye surgery with local anaesthetic blocks. Doses ranged from 5µg/kg to 15µg/kg. [7, 15, 17, 18, 26].

Piritramide

Piritramide is a synthetic narcotic analgesic chemically related to meperidine (pethidine). It was developed in an attempt to reduce the incidence of adverse effects, primarily nausea and vomiting, associated with morphine and other strong analgesics. The actions and use of piritrimide are similar to those of other opioid analgesics and is indicated primarily for the treatment of postoperative pain.

For postoperative pain, the recommended dose of piritramide is 0.2 to 0.3 mg/kg intramuscularly and intravenous doses of 0.1 to 0.4 mg/kg have been used for maintenance of analgesia during surgery. There is some evidence that piritramide may even possess antiemetic effects. Slightly less respiratory depression has been reported with piritramide (20 mg) as compared to morphine (15 mg), however, this is of doubtful clinical significance [5].

Pharmacokinetics

Pharmacokinetic data for piritramide are lacking; maximal analgesia generally occurs within 1 hour of intramuscular injection; analgesic effects persist for 4 to 6 hours. Distribution half-life at steady state was 53.9 minutes after an intravenous bolus dose of 0.2 mg/kg and the elimination half-life is 478 minutes after an intravenous bolus dose of 0.2 mg/kg. Clinically, intramuscular piritramide, 15 to 20 mg can be expected to produce analgesia roughly similar to morphine 10-15 mg. There is some evidence that equianalgesic doses of meperidine and piritramide are 100 and 15 mg, respectively [5].

The most common adverse effect of piritramide is sedation; dizziness, confusion, hypotension, nausea, vomiting, respiratory depression, skin rash, and physical dependence have also been reported. Emetic effects of piritramide may be less than with morphine where as its sedative effects may be greater; anaphylaxis has occurred rarely.

Clinical application

Intramuscular piritramide has been effective and comparable to equianalgesic doses of morphine in the management of postoperative pain; additional studies comparing the efficacy and toxicity of this agent with morphine and other strong analgesics in various types of pain are needed to assess its place in therapy.

Reinhardt and others [28], investigated the effects of pre-block analgesia and sedation using piritramide in a randomized placebo-controlled study in 60 patients having cataract surgery and peribulbar block. The piritramide group received 0.05mg/kg piritramide (Dipidolor ®) intravenously. Their study suggests that using piritramide for analgesia and sedation prior to peribulbar block produces haemodynamic stability and reduces pain perception and endocrine stress response.

Ketamine

Ketamine is a non-barbiturate anaesthetic/analgesic agent structurally related to phencyclidine and cyclohexamine.

The drug is water soluble and its lipid solubility is approximately 10 times that of thiopental.

Ketamine is the only single-agent anaesthetic capable of producing a "dissociative" anaesthesia, which has been useful for a variety of outpatient and inpatient surgical procedures. This agent also produces potent analgesia at subanaesthetic, therapeutic concentrations and it is felt that analgesic and anaesthetic actions may be mediated by different mechanisms [5].

Numerous theories have been proposed to explain the effects of ketamine, including binding to N-methyl-aspartate (NMA) receptors in the central nervous system, interactions with opioid receptors at central and spinal sites, and interaction with norepinephrine, serotonin, and muscarinic cholinergic receptors. Data suggest that at least some effects of ketamine may be mediated via non-opioid mechanisms. Although analgesic effects of ketamine were reversed by naloxone no effect on dissociative properties was observed, even with high naloxone doses, in animal studies [5].

For induction of dissociative anaesthesia, recommended doses of ketamine are between 1 and 4.5 mg/kg intravenously or 6.5 to13 mg/kg intramuscularly; maintenance of anaesthesia is achieved with an intravenous infusion of 0.1 to 0.5 mg/minute or one-half to the full induction dose intravenously or intramuscularly, repeated as required. For sedation and analgesia, intramuscular doses of 2 to 4 mg/kg or intravenous doses of 0.2 to 0.75 mg/kg have been employed; ketamine has also been given orally, rectally, and epidurally.

With adequate anaesthetic doses, a trancelike, cataleptic state with amnesia is produced with no impairment of laryngeal and pharyngeal reflexes or depression of respiration.

Ketamine in sub-anaesthetic doses produces analgesia while preserving airway patency, ventilation, and cardiovascular stability. Side effects, such as hypertension and psychomimetic emergence reactions limit its use a sole agent for sedation. Ketamine preserve airway patency and airway muscle tone in doses of 1mg/kg in men 27-74 years old.

Pharmacokinetics

Peak serum levels of ketamine occur 5 to 30 minutes after intramuscular injection and 30 minutes following oral doses; anaesthesia is usually induced within 30 seconds and 4 minutes following intravenous and intramuscular induction doses, respectively. Similar to thiopental, ketamine is rapidly distributed to highly perfused tissues (e.g., brain, heart, lungs) following parenteral doses, and then redistributed to muscle, peripheral tissues, and fat; the drug is metabolized in the liver, a requirement for termination of activity. Norketamine is an active metabolite of ketamine; most of a dose of ketamine is excreted in the urine as hydroxylated and conjugated metabolites (less than 4% appears in urine as unchanged drug or norketamine). The elimination half-life of ketamine is 2 to 3 hours. Duration of anaesthetic action after a single IV dose is 5 to10 minutes and recovery (discharge) times following intravenous ketamine for outpatient anaesthesia have usually been 1 to 2 hours [5]. More prolonged recovery has been observed after intramuscular injections (e.g. 3 to 4 hours) and with combined use of benzodiazepines. Duration of anaesthesia after intramuscular injection is 12 to 25 minutes and analgesia 15 to 30 minutes [5].

Adverse effects associated with ketamine include emergence phenomena (vivid dreams, hallucinations, delirium), cardiovascular stimulation (tachycardia, hypertension), hypersalivation, elevation of intracranial and intraocular pressures, significant nausea, vomiting, skeletal muscle hyperactivity, nystagmus, and skin rash. Respiratory depression is not usually observed. Benzodiazepines have been useful in attenuating cardiovascular effects and preventing emergence phenomena.

Edwards et al [29] made an interesting observation when looking at the pharmacokinetics of ketamine and alfentanil alone and together in three groups of rats. They found that the plasma distribution co-efficient for ketamine in the presence alfentanil were significantly higher in the brain tissue. The finding that the distribution of ketamine into the brain was increased by low plasma concentrations of alfentanil could have important clinical applications for pain management.

Clinical application

Ketamine is useful as an anaesthetic and analgesic/sedative in a variety of specialized

procedures and clinical settings, particularly in children and asthmatics.
A low dose-dosage regime of three drugs: droperidol, diazepam, and ketamine was described by Cugini and others [30]. The low dose of ketamine used in this study was analgesic without affecting the intraoccular pressure and proved to be safe and useful for patients having eye surgery.
Propofol-Ketamine sedation used for retrobulbar nerve block as described by Frey et al [16] showed that ketamine in sub-anaesthetic doses produces analgesia without respiratory depression and intraoccular pressure was consistently lower in all groups.
Intravenous ketamine, 0.25mg/kg followed 30 seconds later by propofol, 0.5mg/kg was successfully used to eliminate or reduces undesired patient movement and apnea during the administration of regional blocks [31].

Methohexital
Methohexital is an oxybarbiturate that is considered ultra short-acting. The effect of methohexital on the central nervous system is similar to that of other barbiturates. With small doses, methohexital may increase the reaction to painful stimuli and patients occasionally experience over excitement instead of sedation; however, as the dose is increased the paradoxical excitement will disappear [5].
Anaesthetic induction is accomplished with intravenous doses of 50 to 120 milligrams delivered in a 1% solution. Anaesthesia can be maintained with intermittent injections of about 20 to 40 mg/mL (1% solution), given as required, or by continuous intravenous infusion of about 1 drop/second of a 0.2% solution. Methohexital is an effective agent for induction or maintenance of anaesthesia, as an agent for inducing a hypnotic state, or as supplementation with additional anaesthetic agents.
Pharmacokinetics
Onset of anaesthesia is rapid. Distribution half-life is 1 to 4 minutes following intravenous or rectal administration. Elimination half-life is 3.9 +/- 2.1 hours following intravenous doses of 2 to 2.8 mg/kg in adult patients undergoing minor surgery. Similar values have been measured for children (aged 4 - 7 years) and elderly (aged 54 - 65 years) patients after single intravenous doses of 1-2 mg/kg. (This compares to an elimination half-life for thiopental of 5.6-17.6 hours). Doses utilized were 1.2 mg/kg/dose intravenously or 20-25 mg/kg/dose rectally and the patient population included children aged 4-7 years and adults aged 24-65 years, respectively.
The most prominent adverse effects include restlessness, confusion, lethargy, coughing, pain on injection, and involuntary muscle movements; additionally, some degree of gastrointestinal upset, hypersensitivity reactions, and respiratory depression may occur. Rectal administration in paediatric patients [5].
Clinical application
References made in this presentation where methohexital was used in combination with; midazolam and fentanyl [20], propofol and midazolam [22], midazolam and alfentanil [31], midazolam and ketamine [32].

Dexmedetomidine
Parenterally administered dexmedetomidine produces clinical actions similar to those of clonidine, including sedation, reduced salivation, reduced blood pressure and heart rate, and tiredness. Like clonidine, dexmedetomidine has been used in anaesthesia to reduce anaesthetic and opioid requirements, attenuate the tachycardic response to endotracheal intubation, and provide intraoperative haemodynamic stability. Dexmedetomidine itself has anaesthetic properties and has been used as a sole anaesthetic in animal studies. Data from animal studies suggest that dexmedetomidine may reduce volatile anaesthetic requirements to a greater extent than does clonidine. Analgesic effects of dexmedetomidine have also been demonstrated in healthy volunteers and surgical patients [5].
The sympatholytic effects of dexmedetomidine attenuate the stress response in patients emerging from anaesthesia. Patients who receive dexmedetomidine during surgery do not experience increases in heart rate, systolic blood pressure, or in plasma norepinephrine concentrations. Precise mechanisms of anaesthetic effect of dexmedetomidine are unknown but are presumed to be related to stimulation of central postsynaptic, and possibly presynaptic alpha-2 receptors. Anaesthetic and hypnotic effects of the drug have been reversed with alpha-2 adrenoceptor antagonists that cross the blood-brain barrier. The analgesic effects of dexmedetomidine and clonidine appear to be mediated via both spinal and supraspinal mechanisms, although peripheral antinociception

via release of an enkephalin-like substance has also been postulated. Analgesic effects of dexmedetomidine have been blocked by atipamezole but not by naloxone. A close relationship between opioid mu and alpha-2 adrenergic actions in the modulation of pain pathways has recently been suggested.
Dexmedetomidine is indicated for short-term use as a sedative for patients undergoing mechanical ventilation in the intensive care setting.

Pharmacokinetics

Recommended dose for sedation is a loading infusion of 1µg/kg over 10 minutes, with sedation maintained by an infusion of 0.2 to 0.7 µg/kg /hour. Usual intravenous dose of dexmedetomidine for preanaesthetic medication is 0.5 to 0.6µg/kg; for postoperative pain, 0.4µg/kg. Elimination half-life is approximately 2 hours, with clearance of approximately 39 liters/hour.
Blood pressure and heart rate should be closely monitored due to the frequency of dexmedetomidine-induced hypotension and/or bradycardia, especially in hypovolaemic patients and patients 65 years of age and older. Dose reductions should also be considered in the presence of hepatic insufficiency due to impaired clearance of dexmedetomidine. Prolonged sedation has occurred with intramuscular doses [5].

Clinical applications

Premedication with dexmedetomidine attenuates cardiovascular responses to intubation and dexmedetomidine is also effective in providing post-intubation short-term sedation.
Dexmedetomidine has also been shown to reduce anaesthetic and narcotic requirements in a variety of surgical procedures, and provides effective control of postoperative pain. Dexmedetomidine reduces the dose requirements for opioids and anaesthetic agents in a dose as low as 0.63µg/kg [33].

Clonidine

Clonidine is a centrally acting alpha-adrenergic agonist antihypertensive and analgesic agent. Clonidine reduces sympathetic outflow from the brain secondary to direct stimulating effects on alpha-receptors in the vasomotor centre of the medulla. Decreases in blood pressure are associated with reductions in peripheral vascular resistance and decreases in heart rate secondary to increased vagal tone. Baroreceptor responses are usually blunted. Pressor responses and tachycardia upon standing are attenuated by clonidine which can worsen symptoms in patients with pre-existing orthostatic disorders. Although cardiac output falls by up to 20% with acute therapy as a result of reduced heart rate and stroke volume, chronic therapy is associated with only mild reductions or no change in cardiac output.
The usual initial oral dose is 0.1 mg twice daily, adjusted as necessary in 0.1 to 0.2 mg/day increments to desired response.

Pharmacokinetics

The onset of antihypertensive activity is 30 to 60 minutes after oral or intravenous dosing; peak antihypertensive activity occurs within 2 to 4 hours; duration of antihypertensive effect is 6 to 10 hours. Oral bioavailability ranges from 65% to 96%. Elimination half-life is 6 to 23 hours.
Adverse effects of clonidine may include rebound hypertension, atrioventricular block, bradycardia, hypotension, depression, psychotic reactions, drowsiness, fatigue, irritability, vomiting, dry mouth, constipation, sexual dysfunction, hepatotoxicity, and cutaneous reactions.

Clinical application

Clonidine is an effective antihypertensive as monotherapy or combined with other antihypertensive agents (it can be considered for oral therapy in hypertensive urgency). Clonidine epidural injection is approved for adjunctive use in the treatment of intractable cancer pain unrelieved by opioid analgesics alone.
The addition of clonidine to local anaesthetics prolongs anaesthesia and analgesia of local anaesthetics in various neural blocks as well as the duration of retrobulbar block.
Clonidine has been shown to decrease intraocular pressure and to have some analgesic and sedative effects when it is used in premedication for ophthalmic surgery.
Clonidine as premedication has been demonstrated to be effective in reducing the perioperative stress associated to retrobulbar anaesthesia.
When clonidine in doses ranging from 0.5µg/kg to 2.0µg/kg is added to lidocaine 2% with retrobulbar block, it causes a decrease in intraocular pressure, a sedative effect, and an increased duration of analgesia and akinesia, with a small but significant reduction of both systolic and diastolic blood pressure [34, 35].
To determine whether the administration of peribulbar or oral clonidine would enhance

analgesia and anaesthesia in patients undergoing ophthalmic surgery under peribulbar block was studied by Lauretti, et al [36]. Clonidine (15-30µg) added to the peribulbar anaesthetic solution decrease the onset time to anaesthesia and prolonged the time for rescue analgesics in patients under peribulbar block without increasing the incidence of adverse effects. Oral administration of clonidine (75-150µg) alone did not enhance these effects suggesting a local mechanism of action.

Antagonists of benzodiazepines and opioids

The opioids and benzodiazepines are analgesic and sedative drugs which have specific antagonists e.g. naloxone, naltrexone, nalmefene, and flumazenil.

Many of the central actions of drug groups used in conscious sedation can therefore be reversed by specific antagonists which add to the safety profile of these agents and patient care.

Conclusion

In conclusion there does not seem to be one drug or one regime indicative of standardization of sedation practice in eye surgery. It rather seems as if there could be a competition between drugs and regimes for the position of the best and most favourable place in the armamentarium of sedationists and anaesthesiologists. There could be strong indications from the literature that drugs like midazolam, propofol and remifentanil are the favourites for sedation in eye surgery. It might be necessary to take another look at the group of alpha-2 adrenergic agonists and also perhaps at low-dose ketamine.

Music has long been known to reduce anxiety, minimize the need for sedatives and to make patients feel more at ease. It has been shown by Cruise et al [37] that relaxing music increases satisfaction in elderly outpatients undergoing cataract surgery and that it was better than relaxing suggestions or white sound. “I could compare my music to white light which contains all colours. Only a prism can divide the colours and make them appear; this prism could be the spirit of the listener.” Arvo Pärt

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Weill-Marchesani syndrome-is it difficult to intubate?

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Introduction

Weill-Marchesani syndrome (WMS), also known as Spherophakia-Brachymorphia syndrome, is a rare genetic connective tissue disorder associated with fibrous tissue hyperplasia. It was first described by Georges Weill in 1932 [1] and further delineated by Oswald Marchesani in 1939 [2]. It has been suggested that it may have autosomal recessive (AR) or autosomal dominant (AD) inheritance. Autosomal dominant families with WMS were linked to chromosome 15q21.1, the fibrillin-1 gene, while autosomal recessive WMS has recently been mapped to chromosome 19p13.3-p13.2 [3].

The Weill-Marchesani syndrome is characterized by short stature: an unusually short, broad head (brachycephaly), and other facial abnormalities such as hypoplastic maxilla, thickened skin, hand defects, including unusually short fingers (brachydactyly), and distinctive ocular abnormalities [4]. The latter typically include unusually small, round lenses of the eyes (spherophakia) that may be prone to dislocating (ectopia lentis), as well as glaucoma and detached retina [3]. Due to such abnormalities, affected individuals may have varying degrees of visual impairment when presenting for anaesthesia and surgery. Joint stiffness is one of the features of this syndrome. Rennert described an affected 9-year-old boy with joint stiffness that had difficulty in extending his arms over his head [5]. Another report pointed to the difficulty of tracheal intubation due to facial abnormalities and joint stiffness [6].

I am reporting two cases with classical features of Weill-Marchesani syndrome who were subjected to different ophthalmic procedures and in whom the trachea was intubated with great difficulty.

Case reports

Case 1: A 66-year-old male patient, ASA class II, was admitted with the diagnosis of glaucoma in the right eye secondary to prior cataract surgery. He was to undergo pars plana lensectomy and Ahmed implant under general anaesthesia. His weight, height and BMI were 60 kg, 148 cm and 27.4 kg/m^2 respectively. Visual acuity was counting fingers at 1-2 feet right eye, and 20/40 left eye. Preoperative airway assessment was normal with Mallampati 2 classification, good mouth opening and adequate thyromental distance.

Anaesthesia was induced with fentanyl 2µg/kg and propofol 2mg/kg. Because of the ease of manual ventilation with normal preoperative airway assessment, atracurium 0.5mg/kg was given. Nothing could be visualized during the first attempt at laryngoscopy. Even with the help of the McCoy laryngoscope, with repositioning and maximum laryngeal compression, only the tip of epiglottis could be observed with great difficulty (Grade III of Cormak and Lehane classification). Tracheal intubation was successful with the use of a gum elastic bougie. Anaesthesia was subsequently carried out in a standard manner.

Case 2: A 25-year-old male patient, ASA class I, admitted with a diagnosis of dislocated lens, to undergo para plana lensectomy and pars plana vitrectomy in the left eye. Prior to this admission, he was involved in a road traffic accident 9 months earlier with severe head and facial trauma. His body mass index (BMI) was 24 kg/m^2. The patient had a history of surgical correction of LeFort I fracture and a tracheostomy scar was observed. Airway assessment was perfectly normal.

Anaesthesia was induced by fentanyl 2µg/kg and, propofol 2mg/kg. As it was easy to maintain manual ventilation, atracurium 0.5 mg/kg was given. During intubation, only the tip of the epiglottis was visualized with great difficulty. With the help of a McCoy laryngoscope and gum elastic bougie, the trachea was intubated at the fifth attempt. Anaesthesia was maintained with N_2O, O_2 and sevoflurane. Surgery lasted for 150 minutes. Extubation was performed when the patient was fully awake.

Discussion

Patients with WMS can present for cataract and glaucoma surgery as well as repair of retinal detachment. These complications are an integral part of this syndrome. Some of these patients may be unexpectedly difficult to intubate but easy to ventilate. This type of patients may undergo elective surgery because their primary pathology is associated with this syndrome or they may present for any emergency procedure.
Our two patients were difficult to intubate their tracheas. Karabiyik reported a case of WMS who was difficult to ventilate his lung and to intubate his trachea [6]. His patient had joint stiffness and limited mouth opening. Tracheal intubation was performed with the help of an intubating laryngeal mask. Dal et al in another case report used the laryngeal mask airway (LMA) to secure the patient's airway during surgery while he was breathing spontaneously without neuromuscular blockade[7]. McKusick reported that patients with WMS can have hypoplatic maxilla, joint stiffness and arthritis.[8] Giordano et al found a case of WM syndrome showing progressive joint stiffness; an unusual feature was the presence of "primary" osteoporosis in the 28-year-old affected man who was 130 cm tall [9]. One feature of this syndrome is malformed and malaligned teeth [7]. Karabiyik performed magnetic resonance imaging postoperatively which revealed laryngeal stenosis [6]. All of these factors could attribute to intubation difficulty.

On the contrary, we had no difficulty to ventilate any of our patients. Although joint stiffness is one of the fibrous tissue manifestations of WMS, preoperative airway assessment was normal for all our patients; anaesthesiologists who evaluated patients in the perioperative period failed to suspect and/or detect any airway abnormality. Despite the well documented difficulty of intubation, it was hard to differentiate in case no. 2 whether the difficulty to intubate could be related to the syndrome itself or to the previous facial trauma, surgical correction and tracheostomy, or to a combination of all these factors. It was mentioned that joint stiffness and joint prominence are due to dystrophia mesodermalis hyperplasia, and this may lead to difficulty of airway control and intubation [6].

Occasionally, cardiac abnormalities can complicate the clinical course of WMS. Ferrier et al reported an affected 11-year-old girl who also had subvalvular fibromuscular aortic stenosis [10]. Dal et al reported a systolic murmur during patient's examination but paediatric cardiology consultation did not reveal any pathological finding [7]. Although none of our patients had any cardiac abnormalities, comprehensive cardiac evaluation should be carried out with this kind of patients.
Weill-Marchesani syndrome is also associated with multiple skeletal abnormalities such as joint stiffness [5], joint arthritis and limited movement of fingers and modelling defect [8]. Therefore, special attention should be directed to patient positioning during surgery and in the recovery room until the patient is fully awake.

Conclusion

WMS is a rare condition which could present for surgery as a routine ophthalmic procedure or an emergency procedure. Special consideration should be paid for difficult intubation, cardiac abnormalities and patient positioning.

References

1. Weill G. Ectopie du cristallin et malformations générales. Ann Ocul 1932; 169:21-44.
2. Marchesani O. Brachydaktylie und angeborene kugellines als systemerkrankung. Klin Mibl Augenheilkd 1939;103:392-406.
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4. Faivre, L, Megarbane A, Alswaid A, Zylberberg L, et al. Homozygosity mapping of a Weill-Marchesani syndrome locus to chromosome 19p13.3-p13.2. Hum Genet 2002; 110:366-70.
5. Rennert OM. The Marchesani syndrome: a brief review. Am J Dis Child 1969; 117:703-5.

A simple model for practising sub-Tenon's block

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Sub-Tenon's block has gained widespread acceptance for ocular surgery[1]. The ability for trainees to learn to perform this block is , however, relatively limited for a number of reasons . The European Working Time Directive has cut the number of hours trainees spend in theatre [2]. Ophthalmic surgeons often prefer to perform the block themselves. Furthermore senior subspecialty trainees may get a preference on eye lists.

This block requires a certain degree of manual dexterity. Most trainees are unfamiliar with handling the surgical instruments required for this block.

We describe a simple model to help train anaesthetists new to this technique. A latex rubber disposable glove is placed tightly over a model of an orbit as shown in figure 1.

Figure 1

The outer latex cover corresponds to the conjunctiva which can be picked up and incised (figure 2).

Figure 2

Blunt dissection can be performed between this layer superiorly and the surface of the globe inferiorly corresponding to subtenon's space. This arrangement allows trainees to practise handling surgical instruments safely. Trainees can also acquire the skill of positioning the subtenons cannula along the contour of the orbit (figure 3). Moving the cannula along this saggital plane is a manoeuvre that in our experience trainees find awkward.

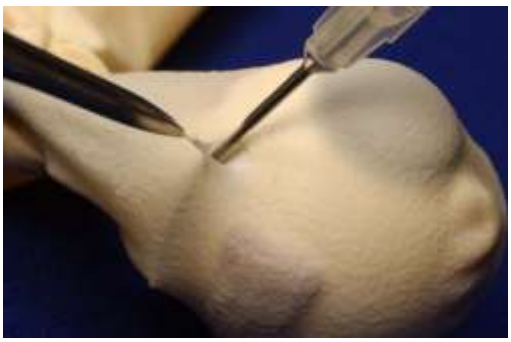

Figure 3

We hope this simple model helps trainees gain confidence in the skills required to perform subtenons block before performing them on patients.

References

1. Eke T, Thompson JR. The National Survey of local Anaesthesia for Ocular Surgery: (1) Survey Methodology and Current practice. Eye 1999; 13:189-95
2. European Working Time Directive. Dept. of Health.2002. The Hospital at Night. MacDonald R. BMJ Career Focus 2004;328

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Dr Monica Hardwick
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CALL FOR PAPERS

There will be a free paper session on Thursday 29th June, and a poster exhibition on both days.
Original research, audit or case reports of interest to Ophthalmic Anaesthetists and Ophthalmologists will be considered. The best free paper will be awarded the Kumar Prize.

CLOSING DATE 19TH MAY 2006

CASE SCENARIO SESSION

Delegates are invited to present short cases with clinical management or medicolegal implications for debate and discussion.

For more details e-mail: boas06@aol.com

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BOAS 2006 Birmingham

Provisional Scientific Programme

Wednesday 28th June

Registration ***1200 hours onwards***

Lunch 1215 – 1300 hours

Introduction & Welcome ***1300-1310 hours Ken Barber – President BOAS***

Session 1 ***1310 – 1530 hours Back to Basics***

1310-1340	History of Ophthalmic Anaesthesia	TBC
1340-1410	Preoperative Preparation/Investigations	Steve Mather
1410-1440	General Anaesthesia for Ophthalmic Surgery	TBC

TEA 1445-1500 hours

Session 2 ***1500 – 1615*** ***Topics in Continuing Education***

1500-1520	The Eye Patient with a Pacemaker	Teri Millane
1520-1540	Anaphylaxis to Anaesthetic Drugs	K-L Kong
1540-1610	Suxamethonium and the Open Eye	Steve Gayer

Guest Lecture 1615 – 1700 hours

Oculopressure Devices Gary Fanning

Conference Dinner at the Burlington Hotel, 1930 hours for 2000 hours

Thursday 29th June

Session 3 ***0900 – 1030 hours Subspecialty Ophthalmic Surgery***

0900-0920	Demands of Modern Glaucoma Surgery	Peter Shah
0920- 0950	Patients with Inflammatory Eye Disease	Phil Murray/Saaeha Rauz
0950-1010	Challenges of Orbital Surgery	Aiden Murray
1010-1030	New Techniques in Corneal Surgery	Sunil Shah

COFFEE 1030 – 1100 hours

Session 4 ***1100 – 1200 hours - Case Scenarios***

Discussion of difficult ophthalmic cases (Contributions are invited from Conference Delegates)

BOAS Annual General Meeting ***1200 - 1245 hours***

LUNCH 1245 – 1345 hours

Session 5 ***1345 – 1445 hours - Free Papers***

TEA 1445 – 1515 hours

Session 6 ***1520 – 1630 hours – Ophthalmic Controversies***

1520-1540	Laser Safety in Ophthalmic Theatres	Marie Tsaloumas
1540-1600	Why worry about Biometry Measurements? TBC	
1600-1630	Political/Manpower Update	Paul Chell

16.30 hours ***Closing Remarks Ken Barber - President***

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BOAS Annual Meeting Report

Postgraduate Centre, General Hospital, Jersey, Channel Islands 27th and 28th October 2005

The Annual meeting of the British Society of Ophthalmic Anaesthesia was held on 27 and 28 October 2005 at the Postgraduate Centre of the General Hospital in Jersey, in the Channel Islands. Mr Bartley McNeela had organised the meeting, in this delightful autumn venue. The meeting was well attended, with over fifty delegates, many of whom brought their partners and extended their stay into a long weekend break. As such, the meeting was a highly successful combination of academic interaction and social conviviality! The weather was gorgeous as well!

The President, Prof Chris Dodds, opened the meeting on the Thursday afternoon with a short, but typically entertaining welcome. This was followed by the first session on case presentations of difficult problems in ophthalmic anaesthesia, chaired by Dr Tony Rubin and Prof Chandra Kumar. This was the first occasion that such a format had been programmed—short case presentations, followed by extended discussion from the floor. The organisers had been rather worried that the session would not work well, as few applications had been received. However, they need not have been concerned, as there were eight separate presentations, followed by extremely lively debate. A recurring theme was the changing approach to informed consent. The session was universally acknowledged to have been highly informative and thought provoking, so much so, that the format will be repeated at the 2006 meeting in Birmingham.

After tea, Mr Paul Rosen, a Consultant Ophthalmologist from Oxford, delivered the first guest lecture on "The implications for ophthalmic anaesthesia of Independent Diagnostic and Treatment Centres". He described the history of the development of such a centre in Oxford and the negative impact this had had on training and delivery of a complete ophthalmological service to the local population. The National implications were extremely concerning, but it does seem likely that most routine cataract surgery will move out of the acute hospitals.

The Annual dinner was held on the Thursday evening, where many of the faculty and delegates were staying. Fish was prevalent on the menu, as Jersey is justifiably proud of the excellent reputation of it's fish restaurants. This dinner was no exception and the lobster was a particular treat.

The first session on Friday morning started with a presentation by Prof Chandra Kumar on chronic orbital pain. We were reminded that we have much to offer these unfortunate patients and that the therapeutic approaches available are within the remit of most practicing ophthalmic anaesthetists. Idse Herrema then described his experiences of paediatric ophthalmic anaesthesia in Newcastle while Prof Chris Dodds covered the management of ophthalmic trauma. Mr Richard Downes from Jersey followed by describing the local anaesthetic techniques he uses for ocular adnexal surgery. I was left hoping that the cosmetic surgeons I work with in the private sector will not catch on to this, as I will be seriously short of work!

After a coffee break, the free papers were presented. All the presentations were of very interesting interesting, none were judged to be of sufficiently high standard to merit publication as an abstract in the Journal Anaesthesia. The Kumar prize, kindly provided by Chandra Kumar is awarded to a junior for best abstract. As there was no abstract presented by a junior, no prize was awarded this year. The Annual General Meeting followed the free papers.

After lunch, Hamish MacLure updated us about new drugs and some old ones, used in ophthalmic anaesthesia . Chris Dodds then spoke about the effects of MMC on ophthalmic anaesthesia and Tom Eke, from Norwich, spoke about topical anaesthesia and its safety. The session finished with a spirited debate on the motion "an anaesthetist is required in the theatre suite for all cataract surgery". Mr David Smerdon, Middlesbrough spoke for the motion and Mr Tom Eke, Norwich, spoke against it. Much hung on the exact definition of "in the theatre suite", but the motion was carried by a substantial majority-

hardly surprising as most of the delegates were anaesthetists!

After tea, Professor Jacques Ripart, from Montpellier, France gave the Abbott lecture on "new perspectives in ophthalmic regional anaesthesia". This session was kindly sponsored by Abbott UK Ltd. The new President, Mr Ken Barber, presented Jacques with a token of the society's appreciation and closed the meeting. Mt Bartley MacNeela and his team were thanked for having organised an excellent meeting in a delightful venue and all were reminded of the next BOAS meeting to be held in Birmingham in June 2006.

News and information

Newsletter
BOAS Newsletter will be available on the BOAS website (www.boas.org). It may be possible to send the newsletter in PDF format provided we have your current email address. To make this facility more effective, please make sure to update your email address and inform the secretary of BOAS.

OASIS (OAS Newsletter)
Printed version of OASIS is not available. Please visit OAS website (www.eyeanesthesia.org)

No subscription for retired members
Retired members do not need to pay the annual subscription fee.

Income Tax Rebate to Society Members
BOAS is registered with Her Majesty's Inland Revenue for the purposes of Corporation Tax. Members can claim income tax allowance against the BOAS subscription.

Contribution for the 13th issue
The next Newsletter will be published in October 2006. Please send your articles or any contributions for inclusion in the Newsletter by 30th March 2005 to Professor Chandra Kumar, Editor Ophthalmic Anaesthesia News, The James Cook University Hospital, Middlesbrough TS4 3BW, UK or email chandra.kumar@stees.nhs.uk

Subscription to Journal of Cataract and Refractive Surgery
Anaesthetic members of BOAS can receive the journal at a discounted rate of £65 by writing to Andre Welsh, UKISCRS, PO Box 598, Stockton on Tees TS20 1WY. Tel 01642651208, Fax 01642651208, Email: ukiscrs@onyxnet.co.uk, Website: www.ukiscrs.org.uk

BOAS database management
Mrs Pat McSorley maintains the BOAS membership database since 1998 but has now retired. She continues to administer and maintain the membership database from her home.

Reasons for joining BOAS
BOAS was formed in 1998 to provide a forum for anaesthetists, ophthalmologists and other professionals with an interest in ophthalmic anaesthesia to facilitate co-operation on all matters concerned with the safety, efficacy and efficiency of anaesthesia for ophthalmic surgery. It is concerned with education, achievement of high standards, audit and research. BOAS will organise annual scientific meetings, produce a newsletter and maintain a web page.

Membership
Membership of BOAS includes anaesthetists, ophthalmologists and other professionals with an interest in ophthalmic anaesthesia.

Membership subscription
Membership runs from January each year. The current subscription is £25.00 payable by banker's standing order.

Liaison and specialist professional advice
With the Association of Anaesthetists of Great Britain and Ireland and the Ophthalmic Anesthesia Society of the USA.

Benefits of Membership
- Opportunity to participate in BOAS annual scientific meetings

- Reduced registration fee for BOAS annual scientific meetings
- Reduced registration fee for other ophthalmic anaesthesia meetings and courses in UK
- Free advice from experts on matters related to ophthalmic anaesthesia
- BOAS newsletter and Directory of Members
- Opportunity to contribute towards development and improvement of ophthalmic anaesthesia
- Access to BOAS web page and scientific literature database
- Eligibility for election to Council of BOAS

Administrative Office and Membership information from

Web address

http://www.boas.org

Change of address

Members are advised to inform the secretary if there is a change of email or postal address.

Dr. Sean Tighe
Secretary, BOAS
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