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Principles and Practice of Ophthalmic Anaesthesia



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Foreword
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CHAPTER

3

Systemic Effects of Ocular Medications

Sujatha R

ABSTRACT

Topical eye medications and systemic medications prescribed to diagnose or treat ocular disease may have effect on other organ systems. Drugs instilled in conjunctiva enter blood stream and there are case reports of serious systemic reactions resulting from systemic absorption. Systemic adverse effects can be reduced by practicing simple manoeuvres. Ocular drugs with significant systemic effects are sympathomimetic eye drops (mydriatic), parasympathomimetic eye drops (mydriatic and cycloplegic) and anti-glaucoma medications (topical and systemic). This chapter reviews the basic pharmacology, potential side effects, and contraindications of these drugs.

INTRODUCTION

Topical mydriatic, cycloplegic and anti-glaucoma drugs (pilocarpine and beta-blockers) act on the autonomic receptors. Systemic anti-glaucoma medicines are carbonic anhydrase inhibitors and hyperosmolar agents. It is important for anaesthesiologists who are also peri-operative physicians to have a thorough knowledge on systemic effects of these ocular drugs. Premedication, general anaesthetics and surgical stress all potentiate the adverse effects of dilation drops used preoperatively. Patients who come for glaucoma surgery might be on one or more of anti-glaucoma medicines which may have systemic effects and interaction with anaesthetic drugs.

In this chapter, both pharmacokinetics and pharmacodynamics of topical and systemically used ocular drugs, their systemic effects, relative and absolute contraindications of these drugs are discussed.

PHARMACOKINETICS OF OCULAR DRUGS

Topical ocular drugs are instilled in the conjunctival sac. The average volume of conjunctival sac is 7 to 10 μL and volume of commercially available eye drops ranges 30 to 75 μL. When eye drop is instilled in

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conjunctival sac it transiently enlarges to hold 25 to 30 μL. The excess flows out or drains through nasolacrimal duct. Tearing dilutes the drug in conjunctival sac. The average tear turnover is 15%, the reflex tearing after eye drop instillation increases the turnover approximately to 30%. Blinking and squeezing of eye contracts orbicularis which channels conjunctival contents into nasolacrimal duct.[1] Topical medications enter systemic circulation through the conjunctival and nasal mucosal vessels. Drugs absorbed from conjunctiva and mucosa escape first pass metabolism in the liver and the pharmacokinetics are similar to intravenous route of administration.[2] Previous animal study have shown that only about 1–7% of the topically applied drugs enters aqueous humour whereas about 50–80% of the drug enters the systemic circulation.[3] Eyelid closure and punctal occlusion for at least two minutes after eye drop instillation reduces systemic absorption of drug by 2/3rd.[2,4]

PHARMACODYNAMICS OF OCULAR DRUGS

Infants and children are at a higher risk for systemic adverse reactions of eye drops for several reasons. Ocular drug doses are not calculated to the weight of the patient. Systemic absorption may result in higher plasma concentrations of drug in children, since they have lesser blood volume compared to adults. Immature metabolic system and poorly developed blood-brain barrier predispose infants to adverse reactions. Children may end up receiving multiple doses if they are uncooperative.

Adults with systemic diseases and medications that may interact with ocular drugs are at higher risk for systemic effects of ocular drugs (Table 1).

CLINICAL AND SYSTEMIC EFFECTS OF MYDRIATICS AND CYCLOPLEGICS

Phenylephrine

Clinical Effects

Phenylephrine, a sympathomimetic agent acts on the alpha-1 adrenergic receptors in the pupillary dilator muscle and the conjunctival blood vessels resulting in pupillary dilation due to contraction of the dilator muscle and blanching of conjunctiva due to contraction of the smooth muscle in the arterioles of the conjunctiva. Phenylephrine 2.5% and 10% eye drops are used as dilating agent for both therapeutic and diagnostic purpose. Phenylephrine does not have cycloplegic effect.

Conjunctival instillation of phenylephrine hydrochloride results in maximal dilatation between 45–60 minutes and recovery occurs in about 6 hours. Darker iris tends to dilate slower than lighter iris.[5,6] Dilatation of pupil with concentrations varying from 1–10% has been studied. The results indicate that the dose response begins to plateau at 5% phenylephrine and a complete plateau is established at 10% phenylephrine.[5]

TABLE 1: Drugs with significant systemic effects

Class of drugs	*Drugs*
Mydriatic	Sympathomimetic (Phenylephrine)
Mydriatic and cycloplegic	Parasympatholytics (atropine, homatropine, cyclopentolate, tropicamide)
Anti-glaucoma medications	Alpha-adrenergic agonist (brimonidine) Parasympathomimetic drugs (pilocarpine) Beta blockers (timolol, levobunolol and betaxolol) Carbonic-anhydrase inhibitors (acetazolamide) Hyperosmolar drug (Mannitol)

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Systemic Effects

Phenylephrine acts on alpha-1 adrenergic receptors present in smooth muscles of blood vessels and causes a rise in systolic and diastolic pressures and reflex bradycardia. Most vascular beds are constricted. Renal, splanchnic, cutaneous, and limb blood flows are reduced but coronary blood flow is increased. Pulmonary vessels are constricted and pulmonary arterial pressure is raised. Phenylephrine has no direct action on the heart. Increase in blood pressure is the most common adverse effect of phenylephrine (Table 2). A marked increase in blood pressure has been reported in low weight premature neonates, infants and hypertensive patients (Table 3). Plasma phenylephrine increases the sensitivity to endogenous catecholamines. This probably explains the increased sensitivity to the drug in longstanding diabetics with autonomic dysfunction (sympathetic denervation).[10]

TABLE 2: Systemic adverse effects of phenylephrine

- Increased blood pressure
- Cerebrovascular accidents
- Pulmonary oedema
- Ventricular arrhythmias
- Tachycardia
- Reflex bradycardia

TABLE 3: Increased risk of side effects of phenylephrine[9]

- Insulin dependent diabetes
- Idiopathic orthostatic hypotension
- Cardiac disease
- Hypertension
- Thyrotoxicosis
- Aneurysms
- Advanced Arteriosclerosis
- Atropine premedication
- Extremes of age (very young and old)[7,8]
- Disturbed corneal epithelial barrier (Intraoperative/trauma/inflammation)
- Decreased lacrimation (under general anaesthesia)
- Monoamine oxidase inhibitors (up to three weeks after their discontinuation) and tricyclic antidepressants potentiate vasopressor effect of phenylephrine.[9]

Parasympatholytics (Anticholinergics, Muscarinic Receptor Antagonist)

Atropine, homatropine, cyclopentolate, and tropicamide are parasympatholytic agents. They block the muscarinic effects of acetylcholine. Parasympatholytic eye drops block the muscarinic receptors of the pupillary sphincter muscle and ciliary muscle, resulting in pupil dilation (mydriasis) and paralysis of accommodation (cycloplegia).

Anticholinergic Syndrome

Anticholinergic syndrome refers to central and peripheral effects resulting from over dosage or abnormal reaction (idiosyncratic response) to clinical dosage of parasympatholytic (anticholinergic) drugs. Patient may present with signs of CNS stimulation, such as agitation, seizures, restlessness, hallucinations, disorientation or signs of depression, such as stupor, coma and respiratory depression. Peripheral signs include tachycardia, hypertension and hyperthermia. They may also have urinary retention and decreased bowel activity.

Signs and symptoms of the anticholinergic syndrome are memorised using mnemonics: *Red* as a beet (cutaneous vasodilation); *Dry* as a bone (anhidrosis); *Hot* as a hare (hyperthermia); *Mad* as a hatter (psychosis/agitation); *Blind* as a bat (nonreactive mydriasis) and *Stuffed* as a pipe (ileus and urinary retention).

Atropine

Clinical effects (Table 4)[9]

Atropine sulfate 1% ophthalmic preparation is the most potent mydriatic and cycloplegic agent available. It is generally preferred as a therapeutic mydriatic agent to prevent postoperative synechiae and treatment of uveitis. It is used as a cycloplegic agent before refraction.

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TABLE 4: Dosage and efficiency of anticholinergics

Drugs	*Time to maximal mydriasis (Minutes)*	*Recovery (Hours)*	*Time to maximal cycloplegia*	*Recovery*	*Dose*	
Atropine	30–40	7–10	3–6 hours	7–14 days	1%	1 drop
Cyclopentone	30–60	1	25–75 min	6–24 Hours	0.5%, 1%	1 drop
Homatropine	20–30	1–4	30–60 min	1–2 days	2%, 5%	1 drop
Tropicamide	20–40	6	30–40 min	2–6 hours	0.5%, 1%	1 drop

Systemic effects

Atropine is a competitive antagonist of muscarinic receptors. At lower dose predominant action is seen at salivary, bronchial, sweat glands and sinoatrial node. It is vagolytic at moderate dose and a CNS stimulant at very high dose. Systemic absorption of atropine from conjunctiva after topical instillation is significant reaching peak levels around 10 minutes.[11] Atropine toxicity could either be due to idiosyncratic reaction or overdose following multiple instillations of the drug.[12]

Atropine toxicity (Table 5).

Signs of atropine poisoning may occur within few minutes to few hours after the conjunctival instillation of drug. Dry mouth and cutaneous flushing are early signs of toxicity while tachycardia may occur at later stages.[13,14]

Incidence of atropine toxicity is particularly higher in lightly pigmented individuals and in children with motor and mental retardation.[9] (Table 6).

Midazolam may be required to control excitement and convulsions of atropine poisoning, but large doses preferably avoided because they mask the CNS depression occurring later stages. Physostigmine, is the specific antidote for atropine given intravenously (1–4 mg in adults, 0.5–1 mg in children) to reverse the coma.

TABLE 5: Peripheral and central symptoms of atropine toxicity

- Dry mouth
- Diffuse cutaneous flush
- Fever
- Urinary retention
- Tachycardia
- Excitement and hallucinations
- Somnolence
- Coma

TABLE 6: Increased risk for atropine toxicity

- Infants
- Blond children
- Children with spastic paralysis or brain damage[14]
- Children with akinetic seizures[14]
- Down's syndrome[15]

Homatropine Bromide

Homatropine 1% is a weaker cycloplegic agent than atropine. It is primarily used in management of iridocyclitis. Systemic toxic reactions following ocular instillation are similar to those for atropine.

Cyclopentolate

Clinical effects

Cyclopentolate hydrochloride ocular preparation has a rapid onset and shorter duration of action than atropine or homatropine (Table 4). Onset of cycloplegia generally approximates onset of maximum mydriasis.[9] The cycloplegic effect is superior to that of homatropine. Cyclopentolate is an effective cycloplegic before refraction and preferred mydriatic before retinal examination.

Systemic effects

Systemic adverse effects of cyclopentolate ocular preparation may occur within 20–30 minutes of conjunctival instillation and the

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symptoms can last 6–24 hours. CNS symptoms such as slurred speech, ataxia, agitation, visual and tactile hallucinations are more common in cyclopentolate toxicity (Table 7).[16-19] Cyclopentolate contains a dimethylated side group [–N–$(CH_3)_2$] also found in hallucinogenic drugs. This explains hallucinations occurring with cyclopentolate overdose and there are case reports of addiction to cyclopentolate eye drops. Children and elderly are at high risk.[19,20] CNS immaturity or aging predisposes to psychotomimetic action to become manifest.[20] Studies on preterm infants has shown that cyclopentolate 0.5% eye drops significantly decreases gastric secretion and this may lead to gastroenteritis and necrotising enterocolitis.[21,22]

TABLE 7: Systemic adverse effects of cyclopentolate

- Drowsiness
- Disorientation
- Agitation
- Cerebellar dysfunction (Incoherent speech, ataxia)
- Visual and tactile hallucinations
- Gastrointestinal toxicity

Adverse reaction due to cyclopentolate ophthalmic preparation in children and adolescents are much less common with the 1% compared with the 2% solution[24] and rarely encountered with the use of the 0.5% concentration in children.

Tropicamide

Tropicamide is a rapid-acting mydriatic with mild transient cycloplegic action. Tropicamide has shown to have a lower affinity for systemic muscarinic receptors. Therefore, lower incidence of systemic side effects.

CLINICAL AND SYSTEMIC EFFECTS OF ANTI-GLAUCOMA DRUGS

Pilocarpine Hydrochloride

Clinical Effects

Pilocarpine is a direct acting cholinergic agonist (parasympathomimetic). It contracts the pupillary sphincter muscle (miosis) and ciliary body and the net result is increase in trabecular outflow.[23] It is available in various concentrations ranging from 0.5–4% and is indicated in acute and chronic narrow angle glaucoma, open angle glaucoma and in secondary glaucoma resulting from pupillary block. Its onset of action is rapid and peak effect occurs between 30–60 minutes and lasts for 4–8 hours.

Systemic Effects (Table 8)

Pilocarpine increases sweat and salivary secretion. Systemic adverse effects due to pilocarpine eye drop is rare but on repeated administration may increase parasympathetic tone.[9]

TABLE 8: Systemic adverse effects of pilocarpine

- Headache
- Brow ache
- Salivation
- Sweating
- Increased gastrointestinal motility (vomiting and diarrhea)
- Relaxation of urethral and anal sphincters (incontinence)
- Bronchospasm in susceptible patients

Brimonidine

Clinical Effects

Brimonidine is a selective alpha-2 adrenergic agonist. Brimonidine tartrate ophthalmic preparation (0.2%) reduces IOP most likely by a dual mechanism.[23,24] The immediate effect is due to local alpha-2 receptor mediated vasoconstriction which decreases aqueous humor production and the chronic effect is due to stimulation of prostaglandin secretion which relaxes ciliary muscle and increase aqueous humor outflow through the uveoscleral pathway.

Systemic Effects

Brimonidine that enters the systemic circulation after conjunctival instillation is

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rapidly metabolised and has a short plasma half-life of approximately 2 hours. Brimonidine is lipophilic and therefore readily crosses blood-brain barrier and this explains case reports of respiratory depression and coma in children (Table 9).[25,27-29]

Dry mouth probably due to the local action of drug at oropharynx is the most common side effect. In a multicentre regulatory trail approximately 10% of patients who participated complained of fatigue/drowsiness. In another study about 50% of patients experienced varying degrees of drowsiness, fatigue and uneasiness.[30] The same study reported that the frequency of side effects was more in patients above 60 years. Children are more prone to CNS depressive effects of the drug[31,33] and there have been reports of apnoea, bradycardia, hypothermia, hypotonia, lethargy, and unresponsiveness in children receiving brimonidine treatment.[32-34] Acute psychosis has been reported in one 68-year-old man.[35]

Brimonidine is contraindicated in patients who are on monoamine oxidase (MAO) inhibitors therapy, tricyclic antidepressants and in children less than 2 years of age.

TABLE 9: Adverse effects of brimonidine

- Dry mouth
- Fatigue
- Drowsiness[32]
- Shortness of breath
- Dizziness
- Headache
- Low mood

Beta Blockers

Clinical Effects

Ocular beta blockers (timolol, levobunolol and betaxolol) lower the intraocular pressure possibly by decreasing the production of aqueous humour. Timolol and levobunolol are non-selective beta blockers while betaxolol is beta 1-selective blocker.

Systemic Effects

Significant quantities of timolol enter systemic circulation after conjunctival instillation and there are reports of plasma levels reaching therapeutic levels in patients using timolol eye drops.[36] A dose of one drop of 0.5% timolol solution to each eye is equivalent to a 10 mg oral dose.[37,38] Oral timolol undergoes first pass metabolism in liver and its bioavailability is reduced whereas ocular timolol escapes first pass metabolism.This explains the reports where plasma levels after single drop of 0.5% timolol solution in each eye is equivalent to a 10 mg oral dose.[37-39] Timolol and levobunolol are non-selective, higher plasma levels may adversely affect cardiac and respiratory systems (Table 10), while betaxolol has advantage of not affecting respiratory system.

Children and patients with diabetes, mild to moderate chronic obstructive pulmonary disease (COPD), hyperthyroidism, cardiac failure, first degree heart block, peripheral arterial disorders (Raynaud's syndrome), and myasthenia gravis are at increased risk of side-effects with beta blockers. In diabetic patients, the beta-blockers may mask the typical systemic manifestations of hypoglycemia.

Use of timolol in patients with second and third degree heart blocks (who do not have pacemaker) is contraindicated. Timolol eye drops may potentiate the effects of the other beta blockers, calcium channel blocker, digoxin and antiarrhythmic (amiodarone) drugs.

Multiple studies have been undertaken to analyse the cardiovascular effects of timolol eye drops and the results found a

TABLE 10: Adverse effects of beta blockers eye drops

- Bradycardia[41]
- Hypotension
- Bronchospasm[42,43]
- Fatigue[44]
- Depression
- Decreased libido

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decrease in resting heart rate (5–10 beats/min) and decreased diastolic blood pressure (5–10 mmHg) and systolic (4–20 mmHg) pressures have been observed. In the first seven years of commercial production of ophthalmic timolol in the United States, 450 serious adverse cardiopulmonary events were reported and 32 deaths were attributed to the use of ophthalmic timolol. Pre-existing cardiovascular disease was reported in 31% of the 212 persons for whom medical history was provided.[41] Deaths secondary to bronchospasm and cardiac arrest have been reported in asthmatic and elderly patients.[41,45]

Carbonic Anhydrase Inhibitors

Carbonic anhydrase inhibitors reversibly block the enzyme carbonic anhydrase in ciliary body and decreases the rate of aqueous humor production.[46] Acetazolamide is an oral carbonic acid inhibitor used for reducing IOP. The onset of IOP reduction begins in 1 hour and maximum IOP reduction occurs 2 to 5 hours after oral administration. The action lasts for 6-8 hours. Recommended dosage[47,48] for reduction of IOP is 62.5 mg or 125 mg orally four times daily which can be increased to 250 mg four times daily, if necessary. Dosages are generally limited to a total of 1000 mg daily because a higher dosage is associated with severe side effects. The dosage in infants and children is 5 to 10 mg/kg administered every 4-6 hours.

Systemic adverse effects reported with oral acetazolamide are shown in Table 11.

TABLE 11: Systemic adverse effects of oral acetazolamide

- Numbness and tingling of extremities and perioral region.[50]
- Metallic taste.[50]
- Weakness, drowsiness, lethargy loss of libido[51]
- Gastrointestinal irritation[52]
- Metabolic acidosis[4]
- Relative hypokalaemia, hyponatraemia[52,53]
- Formation of renal stones[54]
- Blood dyscrasias[55]

Carbonic anhydrase enzyme catalyzes the conversion of hydrogen and bicarbonate ions to carbonic acid and then carbonic acid to carbon dioxide and water. In normal circumstances hydrogen ion is exchanged for sodium in proximal and distal renal tubules. Acetazolamide decrease the availability of hydrogen ions and therefore sodium and bicarbonate ions remain in renal tubule leading to diuresis. Systemic effects of acetazolamide are increased renal loss of sodium, potassium and water,[49] alkalisation of urine and metabolic acidosis.

Most common side effects are tingling and numbness, metallic taste in mouth, nausea and vomiting, which may be related to drug-induced metabolic acidosis. Blood dyscrasias may be idiosyncratic, occurring after as little as a single dose of medication, or they may be dose related.

Acetazolamide is used with caution in patients with chronic obstructive respiratory disease, compromised renal function and sickle cell trait. Patients with severe COPD may not be able to increase alveolar ventilation to compensate the drug-induced metabolic acidosis. Reduced plasma PH may aid sickling in susceptible patients. Hypokalemia is marked when acetazolamide is used with thiazide diuretics. Acetazolamide is contraindicated in patients with hypersensitivity to sulfa drugs, hepatic cirrhosis (due to reabsorption of ammonia) and pregnancy (teratogenic).

Mannitol

Mannitol is a hyperosmotic agent. It reduces IOP by reducing vitreous volume. Mannitol is available as 20% intravenous solution. Recommended dosage for reduction of IOP is 0.5–2 gm/Kg intravenously over a period of 30–60 min.

IV Mannitol causes initial volume expansion (increasing the risk of heart failure), subsequent hypovolemia and hypernatremia, due to diuresis. Mannitol in high doses (2 g/kg) is known to cause a significant

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TABLE 12: Systemic adverse effects of intravenous Mannitol[56]

- Dehydration (dry mouth, headache, fatigue, paresthesia, hypotension)
- Electrolyte imbalance particularly hypernatremia
- Metabolic acidosis
- Heart failure
- Pulmonary congestion
- Thrombophlebitis
- Skin necrosis if extravasation occurs
- Allergic reaction anaphylaxis[57]

increase in serum K+ levels, while low doses (1 g/kg) are known to cause hypokalemia.[58] Hyperkalemia may be due to an expansion in extracellular fluid (ECF) volume leading to dilution of bicarbonate and hence dilutional acidosis, RBC crenation leading to hemolysis, and solvent drag phenomena causing shift of intracellular K+-rich fluid to the ECF compartment to maintain the tonicity. In large doses, it can also cause renal failure because of intra-renal vasoconstriction and intravascular volume depletion.[56]

Mannitol should be used with caution in patients with congestive heart failure, compromised renal function

CONCLUSION

The ocular medications have varying systemic effects ranging from trivial to life threatening signs and symptoms. These systemic effects can be caused by the ocular medications per se or may due to interactions with oral medications taken for underlying systemic illness. In general, these medications should be used cautiously in extremes of ages.

NUGGETS OF WISDOM

1. Significant quantity of eye drop reaches blood circulation.
2. Instillation of multiple drops may result in plasma concentration of drug reaching therapeutic levels
3. Eyelid closure and occlusion of nasolacrimal duct for two minutes reduces systemic absorption.
4. Therapeutic levels of phenylephrine cause vasoconstriction and hypertension.
5. Phenylephrine 10% eye drops should be avoided in children and elderly.
6. Anticholinergic overdose can result in anticholinergic syndrome; symptoms may range from excitement to coma
7. Atropine toxicity most often presents as CNS excitation.
8. Systemic adverse effects of cyclopentolate eye drops are hallucination, behavioural changes and cerebellar symptoms (Ataxia, incoherent speech)
9. Brimonidine crosses blood-brain barrier and may cause severe depression of CNS, hence contraindicated in children
10. Serious adverse cardiopulmonary events are possible with long term use of non-selective betablocker. Cardioselective (betoxolo) does not affect respiratory system.
11. A symptom complex of malaise, fatigue, weight loss, depression, anorexia, and often loss of libido has been reported in up to 48% of patients taking diamox. Paresthesias in the proximal and distal extremities have been reported in up to 75% of patients.
12. IV mannitol should be used judiciously in patients with compromised renal or cardiac function for the fear of pulmonary edema. Patients with renal disease are at higher risk for electrolyte imbalances.

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