Care of the eye during anaesthesia and intensive care

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Learning objectives

After reading this article, you should be able to:

- describe the mechanisms by which anaesthesia and intensive care treatment can impact on the health of the eye
- identify risk factors for eye damage during anaesthesia and critical care
- discuss measures to minimize the risk of injury to the eye
- recognize and discuss the management of iatrogenic eye injury

Abstract

This article describes care of the eye during a period of vulnerability in anaesthesia and intensive care. Risk factors, mechanisms of injury, recognition and management of common and important eye injuries will be covered as well as good practice points and preventative measures pertinent to all anaesthetists and critical care practitioners.

Keywords Chemosis; conjunctivitis; corneal abrasion; exposure keratopathy; eye care; intra-ocular pressure; ischaemic optic neuropathy; keratitis; postoperative visual loss; prone position

Royal College of Anaesthetists CPD Matrix: 1A01, 1A02, 3A12

Eye injury is an iatrogenic complication with potentially devastating consequences. Anaesthesia and critical care treatment attenuate or remove normal protective mechanisms and contribute to altered physiology during a period of increased risk to the eye. Simple preventative measures can reduce the risk of eye injury and adverse impact may be minimized by early recognition and treatment.

The spectrum of complications ranges from temporary and relatively mild, to permanent visual loss and can be broadly categorized as originating from damage to the front of the eye, or those secondary to ischaemia. The most common eye injury associated with anaesthesia is corneal abrasion. Subclinical corneal abrasion may occur in up to 1 in 25 cases of general anaesthesia; however, only 1 in 2800 become clinically significant.[1] Postoperative visual loss is in the range of 1 in 60,000–1 in 125,000 general anaesthetics. Significantly higher rates have been reported in subsets such as patients undergoing spinal surgery in prone position (about 1 in 3300) or cardiopulmonary bypass (about 1 in 1100).[1] The mechanism of visual loss is ischaemic optic neuropathy or retinal ischaemia. Patient, surgical and anaesthetic factors contributing to reduced oxygen delivery to these structures will be discussed within this article.

The risks are not exclusive to anaesthesia. In critical care, eyes are at risk of the above injuries and are additionally vulnerable to exposure keratopathy, chemosis and eye infections – conjunctivitis and keratitis – due to increased average time at risk. This is largely due to attenuation or complete removal of normal eye protective mechanisms such as tear production, blinking and the ability to close eyelids, by disease such as altered conscious level, oedema or neurological injury, or treatments such as sedation, muscle relaxants or the drying effects of oxygen masks or non-invasive ventilation.[2]

Normal safety mechanisms

The most fundamental safety mechanism of the eye is the ability to blink and close the eyelid as a physical barrier to injury and prevent drying of the surface of the eye. This may be impaired by altered conscious level produced by pathological processes, or pharmacologically induced with anaesthetic or sedative agents. Oedema may impair the ability to completely close the eyelid as may neurological disease. Treatment with oxygen and non-invasive ventilation also increase the risk of damage by drying the eye. Risk is markedly increased with invasive ventilation due to its association with longer critical care admission, procedures and sedation over and above the effects of positive pressure ventilation.[2] This exposes the eye to the risks of corneal abrasion from direct injury, exposure keratopathy through inability to maintain moisture and lubrication and super-imposed infection such as microbial conjunctivitis and keratitis. Up to 42% of patients treated in intensive care develop exposure keratopathy.[3]

Physiology and the effects of anaesthesia

Retinal blood supply is from choroidal circulation (via branches of the ophthalmic artery) and the central retinal artery. Blood flow through choroidal circulation is greater than tenfold higher than blood flow through the central retinal artery and accounts for the majority of retinal oxygen supply. Both circulations autoregulate in response to changes in PaO_2, $PaCO_2$ and mean arterial pressure (MAP). In addition, retinal vessels (but not choroidal circulation) exhibit autoregulation in response to changes in intraocular pressure (IOP). However, up to 20% of the population do not exhibit this autoregulation of ocular blood flow. Pathological conditions such as hypertension, hypercholesterolaemia, atherosclerosis, diabetes and ischaemia impair nitric oxide production, which mediates alterations in blood flow

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related to changes in $PaCO_2$, and may therefore impair ocular autoregulation making the eye more vulnerable to ischaemic insults.[4]

IOP varies with position but is normally 10–15 mmHg in the upright stance. Retinal perfusion pressure (retinal vessel arterial pressure − intra-ocular pressure) is therefore normally in the region of 50 mmHg. Factors that increase IOP will consequently reduce perfusion pressure, assuming arterial pressure remains constant. Blood flow to the retina remains constant until IOP reaches approximately 40 mmHg, falling progressively thereafter and ceasing at about 60 mmHg. Moving an anaesthetized patient from supine to prone position will double IOP. This is not a static process. *IOP increases with time spent in prone position, reaching approximately 40 mmHg after 320 minutes*.[5]

Likewise, any increase in central venous pressure can impair venous drainage of the eye, such as may be caused by positional changes, raised intrathoracic or intra-abdominal pressures, or in bilateral neck dissection which is a significant surgical risk factor. The resultant increased volume of the vascular bed will lead to increased IOP, again reducing perfusion pressure. Raised IOP may be caused by direct pressure on the eye, hypercarbia, hypoxia, coughing, straining or alterations in the production or drainage of aqueous humour. Volatile and intravenous anaesthetic agents (with the exception of ketamine) reduce IOP as a result of reduced choroidal blood volume and relaxation of extra-ocular muscles. Non-depolarizing muscle relaxants have minimal effect and suxamethonium increases IOP by around 5–10 mmHg for 5–10 minutes.

The optic nerve is supplied by branches of the ophthalmic and central retinal artery. The posterior section of the nerve is at the highest risk of ischaemia as the feeding vessels are not capable of autoregulation and blood flow is significantly less. This is the most widely reported cause of postoperative visual loss. Optic nerve ischaemia is caused by reduced perfusion pressure (prolonged systemic hypotension, raised IOP), increased resistance to blood flow to the optic nerve (vascular comorbidities), or decreased oxygen delivery, for example, secondary to anaemia or haemodilution.

Hazards to the eye surface

Corneal abrasion and exposure keratopathy

A corneal abrasion is a superficial scratch, removing surface epithelium causing the eye to become red. Patients most at risk include those undergoing prolonged surgery, lateral or prone position and operations on the head and neck. Although these may be caused by direct trauma to the eye or chemical injury (Table 1) the majority result from exposure keratopathy which results from corneal drying as a result of lagophthalmos (failure of the eyelid to close completely). Corneal dryness due to incomplete lid closure leads to excessive tear evaporation and incomplete spread of tear across the eye surface. This occurs in 20–42% of ICU patients,[4] rising to 60% in those ventilated for greater than 48 hours.[6]

- The eye will often become red.
- On examination with fluorescein eye drops and a blue light − the epithelial defect glows bright yellow.
- A white light can also work, but the defect is less obvious.
- In exposure keratopathy, dryness of the cornea may be identifiable due to incomplete lid closure. The defects can appear identical to corneal abrasions.

Chemosis

This describes conjunctival oedema which causes the conjunctiva to bulge outwards. Impaired eyelid closure can either cause or be caused by chemosis, leading to exposure keratopathy. Risk factors include the following:

- compromised venous return from ocular structures caused by;
  - positive pressure ventilation
  - escalating PEEP
  - tight ETT taping
- generalized oedema caused by;
  - fluid overload
  - hypoalbuminaemia
- gravitational causes of increased hydrostatic pressure;
  - prolonged recumbence
  - prone ventilation
  - steep Trendelenburg
- or by increased capillary leak;
  - systemic inflammatory response syndromes.[2]

Treatment of corneal abrasion where it has occurred, is aimed at reducing pain, maintaining lubrication and preventing infection.[2] Often this will consist of topical treatment and ophthalmology opinion should be sought.

Microbial conjunctivitis and keratitis

The longer patients are ventilated and sedated in intensive care, the more likely the eye will become colonized with one or multiple bacteria. *Pseudomonas aeruginosa*, *Acinetobacter* spp. and *Staphylococcus epidermidis* are the most commonly isolated organisms.[3]

A major source of these infections is thought to be from respiratory secretions. Two mechanisms of potential transmission include:

- Aerosols from respiratory suctioning.
- Direct contact from tracheal suction catheter with the eye.

Perioperative causes of corneal abrasion

Perioperative	Postoperative
Face mask	Face mask
Anaesthetist's hands	Patient's fingers
Watch strap	Bed linen
Name badge	
Laryngoscope blade	
Direct irritant effect of volatile gas	
Surgical drapes	
Surgical implements	
Skin preparation solutions	

Table 1

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Where corneal abrasion or exposure keratopathy exists, microbial keratitis can quickly progress to cause deep infection and potential for permanent damage or vision loss.

Conjunctivitis is a common condition which is very contagious. Great care should be taken with hand hygiene in order to prevent spread to staff and other patients. The eye will be sticky and usually red. Microbial keratitis occurs when a vulnerable defect in the damaged cornea becomes infected. The cause is usually bacterial but herpes simplex keratitis may present in immunosuppressed patients in intensive care. Again the eye is red and may be watery or sticky and a corneal ulcer may be present. The epithelial defect stains with fluorescein dye. If on examination the cornea is dull or contains a white patch, ophthalmology opinion should be sought urgently.

Treatment priorities include ensuring the prevention of spread of infection while helping to maintain the normal function of the eye and treating the infection. For this reason ointment is used as a first line as it lasts longer and helps with lubricating the surface of the eye. Chloramphenicol ointment is a common choice. The eye should always be swabbed for microbiological investigation and where an organism is found to be insensitive then addition of a second agent in the form of drop or ointment may be required. Where significant improvement is not observed over 24–48 hours, ophthalmology opinion should be sought.[1]

Ischaemic hazards and postoperative visual loss

Ischaemic optic neuropathy (ION) occurs when blood supply to the optic nerve is not sufficient to meet demands resulting in infarction or ischaemia. The causes include those factors mentioned above that lead to decreased perfusion pressure. The posterior part of the optic nerve is more vulnerable to ischaemia and posterior ischaemic optic neuropathy can be more insidious in onset. Surgeries known to carry an increased risk of ION include spinal surgery, cardiopulmonary bypass and bilateral radical neck dissection.[4] Surgery over 6 hours, blood loss greater than 1000 ml and comorbidities such as atherosclerosis, diabetes and hypertension along with male gender have all been shown to increase risk of visual loss during surgery in the prone position where there was no direct pressure on the eye. In one analysis, 83 of 93 cases of postoperative visual loss following spinal surgery in the prone position were caused by ION.[7] The use of a Wilson frame, where the patient's head is placed lower than the heart, is also associated with an increased risk of ION.[4]

Central retinal artery occlusion (CRAO) following surgery is thought to be a complication of patient positioning, where external pressure on the eye causes compression and cessation of flow in the central retinal artery. In the same analysis, all 10 cases of CRAO were unilateral and used some form of headrest rather than Mayfield pins. Signs of periocular trauma such as decreased supraorbital sensation, unilateral erythema or corneal abrasion were present in 7 out of 10 cases.[7]

ION or CRAO present as painless visual loss after surgery. Urgent ophthalmology referral is indicated. Sadly this is often permanent, bilateral and complete. It may alternatively present as a visual field defect. The optic disc may appear pale and oedematous. Where either condition is suspected, perfusion to the eye should be optimized by ensuring the patient's haemoglobin, haemodynamic parameters and arterial oxygenation are within the normal range. Magnetic resonance imaging may be used to rule out intracranial causes of visual loss. Treatment for CRAO aims to re-perfuse the organ as quickly and safely as possible and initiate secondary preventative measures. IOP lowering agents, steroids and anti-platelets may be used although no single treatment is superior. These therapies are not effective in the treatment of ION. Expert opinion should be sought early. The prognosis for both CRAO and ION is very poor.[4]

Strategies for eye protection

Sedative and muscle relaxing agents decrease the tonic contraction of the orbicularis muscle around the eye, which usually maintains lid closure. Lagophthalmos occurs in 59% of individuals under general anaesthesia.[4] For this reason, simple manual closure of the eyelids rarely provides sufficient protection for the eyes during anaesthesia. Patients undergoing certain operations, such as those performed on the head and neck and those in the lateral or prone position, require further eye protection.

The most common intervention to protect the eye during general anaesthesia is taping of the eyelids with a simple paper based tape such as 'Micopore'. This has been shown to reduce the risk of corneal abrasion.[4] The eye should be taped horizontally or from the upper lid to the lower, taking care to ensure that the lashes are clear of the corneal surface and that the lids are fully apposed. Care should be taken to avoid trauma to the eyelash or eyelid when removing tape from the eye. Tape should be removed from the upper lid before the lower lid.

Grading of incomplete eye closure

	Grade 0	Grade 1	Grade 2
Assessment	Lids completely closed	Conjunctival exposure but no corneal exposure	Any degree of corneal exposure
Treatment	None required Continue to assess regularly	Lubrication applied directly into the eye prior to eyelid closure Ointments are preferred as drops do not last long enough	Lubrication as for Grade 1 followed by taping of the lids with paper-based tape Ensure lashes are clear of the cornea and the outside of the eye is dry to allow the tape to stick

Table 2

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Appropriate protective strategies in intensive care are dependent on identifying those at the greatest risk of corneal injury. This is most effectively achieved by strict adherence to a clear protocol for assessment of the eye and intervention. Assessment should be carried out on admission to intensive care and regularly thereafter. Grade of severity of incomplete closure of the eyelid (lagophthalmos) is shown in Table 2.

As part of a clear protocol when treating the eye in intensive care it should always be examined with a bright light looking for redness, chemosis, corneal dullness or opacity. If found, this should prompt an increase in frequency of lubrication and consideration of referral for ophthalmology opinion.[2]

Eye protection for patients anaesthetized in the prone position, both for surgical procedures and in intensive care, is of the upmost importance as the incidence of complications ranging from corneal abrasions to complete visual loss are higher than in other positions.

While in the prone position, issues include increasing facial oedema, conjunctival swelling (chemosis) and direct pressure on the globe or orbit.[1] Direct pressure on the eye can lead to central retinal artery occlusion but visual loss can also occur in the absence of any pressure on the globe or orbit, as a result of ischaemic optic neuropathy.[8] It is imperative to check for direct compression on the globe at regular intervals. In intensive care, patients nursed in the prone position should have their eyes examined, re-lubricated and taped shut every four hours.

Regardless of equipment chosen to aid positioning, patients anaesthetized in the prone position should remain neutral or with their head higher than their heart. Any significant neck flexion, extension or rotation should be avoided where possible.[9] During surgical procedures the most effective way to reduce the risk of direct pressure on the eye involves the use of a three-pin head holder (Mayfield pins). This is commonly used during cranial and cervical spinal surgeries. Although it should prevent direct pressure on the eye it is an invasive technique which will cause lacerations to the scalp and requires specialist equipment. Other devices designed to aid care of the eyes and face during surgery in the prone position include preformed foam or moulded gel supports. These may be used for prolonged cases of up to 8 hours. Once positioned, it should be easy to view the eyes and check that the position is correct. If surgery is prolonged, or if the patient is left in either slight neck flexion or extension, then pressure areas can develop on the face or chin. A horseshoe, gel ring or pillows are used to aid protection of the eyes and face during shorter surgeries in the prone position. Although these are cheap and easily available, they are susceptible to changing position if the patient is moved and some necessitate having the head turned which increases risk of vessel, nerve or bony injury.[10]

Preventative measures are crucial to maintain health of the eye during anaesthesia and critical care. In contrast to corneal abrasion, where the vast majority make a full recovery with no adverse impact on vision long term, ischaemic visual loss is often permanent once it has occurred. Where concern exists that eye damage has developed, prompt ophthalmology opinion should be sought. ◆

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