Care of the eye during anaesthesia and intensive care

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Learning objectives

After reading this article, you should be able to:

- understand the different aetiology of the two major causes of postoperative visual loss
- identify patients at high risk of postoperative visual loss and take appropriate prevention strategies
- understand the aetiology and effective prevention of perioperative corneal abrasions

Abstract

Perioperative eye injuries and blindness are rare but important complications of anaesthesia. The three causes of postoperative blindness are ischaemic optic neuropathy, central retinal artery thrombosis (these can exist in tandem and have been described as ischaemic oculopathies) and cortical blindness. This article aims to improve anaesthetists' knowledge of orbital anatomy, ocular physiology and the mechanisms of perioperative eye injuries to help reduce their occurrence.

Keywords Anaesthesia; central retinal artery occlusion; corneal abrasion; intraocular pressure; ischaemic optic neuropathy

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Perioperative eye injuries and blindness are rare but important complications of anaesthesia. Eye injuries account for 2% of claims against anaesthetists. A better understanding of orbital anatomy, ocular physiology and the mechanisms of ocular injury may help to reduce their occurrence.

Arterial supply to the optic nerve and retina

The ophthalmic artery enters the orbit through the optic canal enclosed within the dural sheath of the optic nerve and its first branch within the orbit. The central retinal artery runs along the inferior aspect of the optic nerve exiting from the dural sheath of the nerve approximately 10 mm behind the globe. The vascular supply to this posterior part of the optic nerve is from pial branches of the ophthalmic artery and the central retinal artery.

The central retinal artery divides into four major vessels at the optic disc each supplying one quadrant of the retina. The retinal vessels are distributed within the inner two thirds of the retina, while the choroidal circulation supplies the outer layers of the retina.

Two to three posterior ciliary arteries arise from the ophthalmic artery, each of which divides into one long and 8–10 short posterior ciliary arteries. The short posterior ciliary arteries pierce the sclera and form the choriocapillaris, which supplies the anterior part of the optic nerve, the lamina cribrosa and the choroid posterior to the equator.

The long posterior ciliary arteries travel forward in the suprachoroidal space to the ciliary body where they combine with the anterior ciliary arteries to form the major arterial arcade. Recurrent branches of the long posterior ciliary arteries supply the choroid anterior to the equator and anastomose with the short posterior ciliary arteries.

Age-related arteriosclerotic changes in the orbital arteries are more severe in the most proximal vessels. In particular, arteriosclerotic changes are most marked at the following sites: where the ophthalmic artery enters the orbit and at the origins of the posterior ciliary arteries and central retinal artery.[1]

Ocular blood flow and perfusion

Ocular blood flow (OBF) is determined by the pressure difference between mean arterial pressure (Pa) and mean venous pressure (Pv) and the resistance to that flow (R).

$$\mathrm{OBF} = \frac{P\mathrm{a} - P\mathrm{v}}{R}$$

Retinal blood flow is approximately 170 ml/100 g/minute but between 60 and 90% of retinal oxygen supply comes from the very high choroidal circulation (2000 ml/100 g/minute). The retinal circulation autoregulates in response to changes in arterial PaO_2, $PaCO_2$, BP, (up to a 40% increase in) and perfusion pressure. However, the choroidal circulation autoregulates in response to changes in PaO_2, $PaCO_2$ and BP, but not to increases in intraocular pressure (i.e. perfusion pressure). Breathing 100% oxygen causes retinal vasoconstriction, reducing retinal blood flow by 60%, which is not sufficient to prevent an overall increase in retinal PO_2. Carbon dioxide causes retinal vasodilatation so that retinal blood flow increases by 3% for each 1 mmHg increase in $PaCO_2$. Nitric oxide (NO) is an important regulator of ocular blood flow as it mediates hypercapnia induced vasodilatation in the choroid and modulates pressure autoregulation of ocular blood vessels. Inhibition of NO by nitric oxide synthase inhibitors causes a 40% decrease in the choroidal blood flow with no effect on retinal blood flow indicating that NO is produced by retinal ganglion cells in addition to the retinal vascular endothelium. Pathological conditions like hypertension,

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hypercholesterolaemia, arteriosclerosis, diabetes and ischaemia impair nitric oxide production.

In the upright position the pressure within the artery is between 60 and 70 mmHg, while the intraocular pressure (IOP) is between 10 and 15 mmHg. Under normal conditions this provides a perfusion pressure of approximately 50 mmHg. The episcleral venous pressure is approximately 3–7 mmHg and increases by 3–4 mmHg in the supine position. If large increases in episcleral venous pressure occur, part of this pressure will be transmitted into the intraocular veins causing congestion with reduced perfusion pressure. Patients (particularly the obese) may have increased intraabdominal and central venous pressures in the prone position thereby causing increased venous pressure in the head.[2]

Intraocular pressure also varies with posture.[3] Intraocular pressure doubles when anaesthetized patients are positioned prone. Thereafter, IOP continues to increase with time and reaches a value of approximately 40 mmHg after 320 minutes in the prone position.[4]

The pressure exerted on the face in the prone position can be reduced by the use of Mayfield pins to secure the head or by using face contoured prone positioning devices. These reduce the average pressure on the face around the eyes by approximately 29% compared with non-contoured products. Pressures greater than 50 mmHg can occur just above the supraorbital ridge[5] and if the face is placed in an ill-fitting device this external pressure may be directly transmitted to the eye reducing ocular perfusion pressure and blood flow.

Retinal blood flow is maintained by autoregulation over a wide range of perfusion pressures thus maintaining PO_2 in the inner parts of the retina. In most people, when intraocular pressure increases retinal blood flow remains constant until the IOP reaches approximately 40 mmHg after which it falls with progressive increases in IOP. At an IOP of approximately 60 mmHg blood flow to the optic nerve at the disc ceases. Autoregulation fails to occur in a significant minority of individuals (approximately 20%), which results in a progressive reduction in retinal blood flow at the onset of an increase in IOP. The lack of choroidal autoregulation during increases in IOP, means the PO_2 in the choroid and outer retina decreases. In primates, irreversible damage occurs if ocular ischaemia exceeds 100 minutes, but in humans there is little correlation between occlusion time and visual outcome.

Causes of ischaemia of the optic nerve

- Arterial hypotension
- Elevated venous or intraocular pressure (prone/head down position)
- Increased resistance to flow: (a) decreased endothelial derived vasodilators (prostanoids and nitric oxide) in atherosclerosis, diabetes mellitus, hypertension, hypercholesterolaemia, cigarette smoking and (b) increased endothelial derived vasoconstrictors (endothelins 1–3, angiotensins)
- Decreased oxygen delivery (anaemia)

Postoperative visual loss

Ischaemia of the optic nerve is classified into either anterior or posterior ischaemic optic neuropathy (ION).

Anterior ischaemic optic neuropathy (AION) is caused by infarction at the watershed zones listed above and results in a visual field defect, a pale oedematous optic disc and oedema of the optic nerve in the posterior scleral foramen.

Posterior ischaemic optic neuropathy (PION) occurs when the pial branches of the ophthalmic artery become occluded. Blood flow in the posterior part of the optic nerve is significantly less than that in the anterior part of the optic nerve. These pial vessels are end arteries that are not capable of autoregulatory control and therefore this part of the optic nerve is more vulnerable to ischaemia in the event of a fall in perfusion pressure or anaemia. PION is characterized by a slower onset of visual field defect and mild optic disc oedema.

The incidence of ION varies between 1 in 30,000 and 60,000 operations. High-risk procedures are spinal surgery, cardiopulmonary bypass and bilateral neck dissection.

The primary mechanism of ocular ischaemia following bilateral neck dissection is a reduction of ocular perfusion pressure. This is due to the increase in venous pressure when the normally adequate venous collateral circulation is sacrificed to ensure adequate tumour clearance.

There are multiple reasons for postoperative visual loss after cardiac surgery; embolic, changes in oncotic pressure, ischaemic, thrombotic and surgical technique.

In an analysis of 93 cases of postoperative visual loss after spinal surgery ION occurred in 83 of the 93 patients. PION was diagnosed in 56 cases[6] of the cases and of those with ION, 55 suffered visual loss affected both eyes. In contrast, central retinal artery occlusion was the cause of visual loss in 11, all of which were unilateral. Headrests (including horseshoe headrest) were used in all cases. Stigmata of periocular trauma were present in 70% of patients with central retinal artery occlusion:

- decreased supraorbital sensation
- unilateral erythema
- periorbital oedema
- ptosis
- corneal abrasion
- ophthalmoplegia
- proptosis.

These findings suggest that central retinal artery occlusion was caused by globe compression in the prone position.

In contrast, the causes of ION in prone surgery appear to be multifactorial and might be related to an acute increased venous pressure in the head and neck. Risk factors for ION are: male sex, obesity, prolonged procedures extensive blood loss and use of the Wilson frame which places the patient's head much lower than the heart. There is no safe lower limit for either arterial BP or haematocrit that avoid postoperative visual loss.

When patients are anaesthetized in the prone position, the head should be positioned so that it is level or higher than the heart and in a neutral forward position, without significant neck flexion, extension, lateral flexion or rotation.[7] It is imperative that anaesthetists regularly check for globe compression and that only headrests specifically designed for the prone positions are used.

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