Perioperative Visual Loss

An Essay

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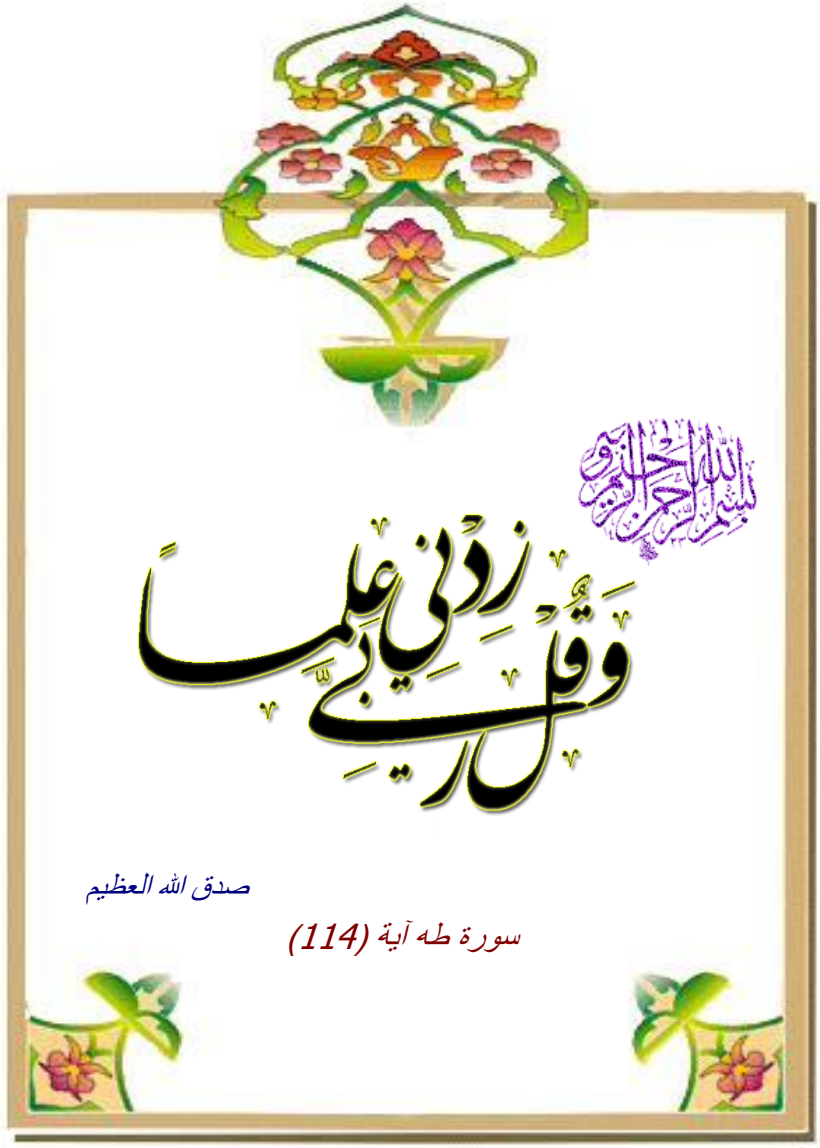

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List of Abbreviations

AION	:	Anterior ischemic optic neuropathy
ASA	:	American Society of Anesthesiologist
BRAO	:	Branch retinal artery occlusion
CNS	:	Central nervous system
CPB	:	Cardiopulmonary bypass
CRA	:	Central retinal artery
CRAO	:	Central retinal artery occlusion
CRV	:	Central retinal vein
CT	:	Computerized tomography
ERG	:	Electroretinography
ION	:	Ischemic optic neuropathy
IOP	:	Intraocular pressure
IOV	:	Inferior ophthalmic vein
LGN	:	Lateral geneculate nucleus
MAC	:	Minimum alveolar concentrartion
MRI	:	Magnetic resonance imaging
NIS	:	Nationwide Inpatient Sample

List of Abbreviations (Cont.)

OA	:	Ophthalmic artery
ON	:	Optic nerve
ONH	:	Optic nerve head
PCA	:	Posterior cerebral arteries
PION	:	Posterior ischemic optic neuropathy
RAO	:	Retinal artery occlusion
RPE	:	Retinal pigmented epithelium
SOV	:	Superior ophthalmic vein
TURP	:	Transurethral resection of the prostate
VEP	:	Visiual evoked potential

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Introduction

Patients assume a certain risk of vision loss when undergoing ophthalmic surgery, but awaking blind after elective nonocular surgery is a catastrophic event for the patient, the surgeon and the anesthesiologist. Although relatively rare, perioperative visual loss (POVL) has warranted a substantial number of publications in the medical literature and has become an important medical-legal issue.

POVL is an uncommon complication primarily associated with cardiac, spine, and head and neck surgery that can have a potentially severe impact on quality of life.

It has long been recognized that visual damage may follow the administration of anesthesia. In 1937, Guedel noted “We still see too much conjunctivitis and traumatic keratitis following anesthesia”. The injuries at that time were attributed to open drop ether through a gauze covered mask in hard to handle subjects who half opened their eyes during light anesthesia (the patient was deemed in part responsible). A towel was customarily placed over the eyes and often became saturated quickly. Given that about 90% of anesthetics were produced by ether, mostly by an open or simple mask system throughout the first half of the 20th

century, it is not surprising that serious corneal injuries were common but considered a small price to pay for a pain free surgical experience.

The first report of perioperative visual loss was in 1948 by Slocum and colleges involving a patient in the prone position during spinal surgery. The suspected etiology was improper positioning of the head on a headrest with direct pressure on the globe. The incidence has varied significantly from study to study depending on the methods used for detection, the patient population involved, and surgical procedures.

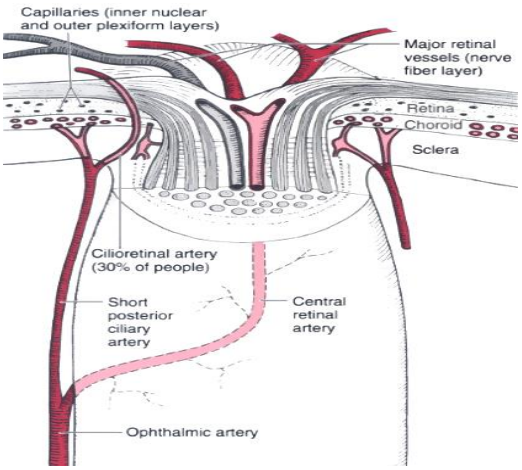
Perioperative visual loss is a dreaded complication for patients and healthcare providers because the most common causes of POVL have no-proven treatment and are associated with poor recovery. The disability and impact on quality of life for patients can be devastating. Research on this topic has barely progressed beyond the infancy stage because of its low incidence, lack of animal models, and technological deficiencies in monitoring the visual pathways under anesthesia.

This essay presents the specific mechanisms responsible for perioperative eye injury, differential

diagnosis including symptoms and commonly used diagnostic tests, causes of postoperative visual loss and ocular complications, diagnosis of visual loss and other ocular complications in the postoperative period, prognosis and the lines of management.

Blood Supply of the Eye

The blood supply to most of the eye is provided by the ophthalmic artery (OA), the first intracranial branch of the internal carotid artery. The intraorbital optic nerve (ON), retina and choroid are supplied by branches of the OA, specifically, the posterior ciliary arteries (PCAs), central retinal artery (CRA), and the pial vessels along the ON (Fig 1). Anastomoses may be present between the internal carotid system from which the OA arises and the external carotid artery. The lacrimal artery anastomoses to the middle meningeal artery. In the eyelids and nose, distal branches of the OA and the external carotid artery anastomose ***(Remington, 2012).***



(Fig.1)**The ophthalmic artery branches into the posterior ciliary arteries and the central retinal artery.** The central retinal artery enters the optic nerve behind the globe and sends branches to supply the optic nerve ***(Federman et al., 1994).***

Under normal conditions, these connections may not be significant in the ocular blood supply, but they may become potential pathways for iatrogenic particle embolization after intra-arterial injection into the external carotid system, or conversely, they could compensate for obstruction in the OA. The ocular branches of the OA are the CRA and PCA trunks, which branch into the main PCAs. Typically two to three PCA trunks branch into the medial and lateral PCAs (Fig 1). The main PCAs divide into short PCAs (sPCAs) ***(Mames et al., 1991)***.

sPCAs supply the posterior choroid and the anterior portion of the ON. The medial and lateral sPCAs form the circle of Zinn-Haller around the ON (Fig 2). From the circle of Zinn-Haller are derived the pial branches, choroidal branches, and other vessels that supply the ON ***(Cioffi, 1999)***.

Anatomic variation in the PCA circulation has important implications in the etiology of ischemic optic neuropathy (ION). The OA gives rise to one to five PCAs. One PCA is found in 3%-7% of subjects, two in 25%-48%, three in 39%-50%, four in 8%-17%, and five in 2%-8% ***(Erdogmus and Govsa, 2008)***.

The PCAs are end arteries without anastomoses, and the location of the resulting watershed zones (area between two end arteries) in the ON varies anywhere between the fovea and the nasal border of the optic disk ***(Wray, 1993)***.

The watershed zones may be located temporal to the optic nerve head (ONH), pass through parts of the ONH, or even include the entire ONH. The location of the watershed zone determines the susceptibility of a portion of the ONH to ischemia ***(Hayreh, 1990)***.

The visual cortex is supplied by the posterior cerebral arteries, and the ON radiation is supplied by the middle cerebral arteries in the parietotemporal lobes. Occlusion of both posterior cerebral arteries does not uniformly produce blindness because of collateral circulation through the circle of Willis. Infarction in the visual cortex is instead believed to be embolic or the result of a bilateral "watershed" incident from hypoperfusion in the distal branches of the posterior cerebral circulation ***(Aldrich et al., 1987)***.

Venous drainage of the eye:

The orbital veins are devoid of valves. The superior ophthalmic vein (SOV) contains most of the ocular venous outflow. The retina and anterior ON are drained by the

central retinal vein (CRV), which empties into the SOV. The choroid is drained by four vortex veins located in each posterior quadrant of the eye; these veins drain into the SOV and the inferior ophthalmic vein (IOV) and have numerous anastomoses between them. Both the SOV and the IOV empty into the cavernous sinus ***(Remington, 2012).***

The Optic Nerve (ON) Microvasculture:

The anterior portion of the ON is proximal to the lamina cribrosa, an elastic, collagenous tissue through which the ON and the CRA and CRV pass as they enter the optic disk. The anterior portion of the ON includes the superficial nerve fiber layer and the prelaminar region. The prelaminar area is a thick tissue constituting the major part of the optic disk volume ***(Hayreh and Jonas, 2001)***.

Neural fibers transit the laminar region through fenestrations. The retrolaminar region is the posterior portion of the ON and consists of meningeal sheaths and myelinated axons. The ON diameter is enlarged in this area to about 3mm. The superficial nerve fiber layer derives its blood supply mainly from arterioles in the retina, although in the temporal regions, it may receive blood from the PCAs. The