“OUTCOME OF ENDOSCOPIC DECOMPRESSION OF OPTIC NERVE FOR TRAUMATIC OPTIC NEUROPATHY”

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In partial fulfilment of the Regulations

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MAY 2022

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DESIGNATION OF PRINCIPAL INVESTIGATOR: POST GRADUATE STUDENT
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THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1. TIREC Application Form ☐
2. Study Protocol ☐
3. Department Research Committee Approval ☐
4. Patient Information Document and Consent Form in English and Vernacular Language ☐
5. Investigator's Brochure ☐
6. Proposed Methods for Patient Accrual Proposed ☐
7. Curriculum Vitae of the Principal Investigator ☐
8. Insurance /Compensation Policy ☐
9. Investigator's Agreement with Sponsor ☐
10. Investigator's Undertaking ☐
11. DCGI/DGFT approval ☐
12. Clinical Trial Agreement (CTA) ☐
13. Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA) ☐
14. Clinical Trials Registry-India (CTRI) Registration ☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted.
5. The TIREC will monitor the study
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7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should Receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear Terms as follows:
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   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and the revised budget form should be submitted.
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TABLE OF CONTENTS

ABBREVIATION

TON	-	Traumatic optic neuropathy
RTA	-	Road traffic accident
FE	-	Fovea ethmoidalis
LP	-	Lamina papyracea
ON	-	Optic nerve;
CCA	-	Anterior genu of the intracavernous carotid artery;
L. OCR	-	Lateral opticocarotid recess;
ISS	-	Sphenoid intersinus septum;
SS	-	Sphenoid sinus;
MS	-	Maxillary sinus;
MT	-	Middle turbinate
MR	-	Medial rectus muscle
VA	-	Visual acuity
#	-	Fracture
SAH	-	Subarachnoid hemorrhage
EDH	-	Extradural hemorrhage
SDH	-	Subdural hemorrhage
PL	-	Perception of light
HM	-	Hand movements
RTL	-	Reacting to light
NRTL	-	Not reacting to light
RAPD	-	Rapid afferent pupillary deficit

INTRODUCTION

Traumatic optic neuropathy is a serious vision threatening complication of ocular or head injuries. Traumatic loss of visual function, which can present as subnormal visual acuity, visual field loss, or colour vision dysfunction, is the characteristic of optic neuropathy. The presence of an afferent pupillary defect strongly shows that the injury occurred prechiasmally and is required to confirm the diagnosis of traumatic optic neuropathy. Traumatic optic neuropathy can cause partial or complete vision loss, which can be transient or permanent.

The patient may benefit more from early identification and treatment of traumatic optic neuropathy. The presence of an absolute or relative afferent pupillary defect, as well as disc edema and vascular congestion, aid in diagnosing an optic nerve deficit. These findings, when combined with the computed tomography (CT) scan, perhaps an MRI scan, and visual evoked potentials, may be sufficient to proceed with optic nerve decompression.

HISTORY

Hippocrates first observed visual loss following a craniofacial injury. As this is a very devastating condition for the patient, and also this is potentially reversible, timely intervention is crucial. But in 40-72% of cases, treatment of coexisting head injury takes precedence thus leading to delay in diagnosis and management of this condition.

Since 1916 Optic nerve decompression has been practised for several disorders with visual loss. Traumatic optic neuropathy is one of the common indication. In early times, the common method for decompressing optic nerve is fronto temporal craniotomy but with high rate of mortality. In 1926, Sewall approached medial optic canal via external ethmoidectomy and did extracranial optic nerve decompression with less morbidity than the previous craniotomy technique. Others forms of extracranial decompression were combined lateral and medial orbitotmy, lateral facial approach, transantral ethmoidal approach, sublabial transnasal approach ,and intranasal microscopic technique.

Recently, endoscopic endonasal optic nerve decompression has been widely applied for many disorders. Aurbach reported the use of endoscopic endonasal optic nerve decompression in German literature in 1992. Many concluded that this technique offers advantages of decreased morbidity, no external scars and rapid recovery time.

AIM OF THE STUDY

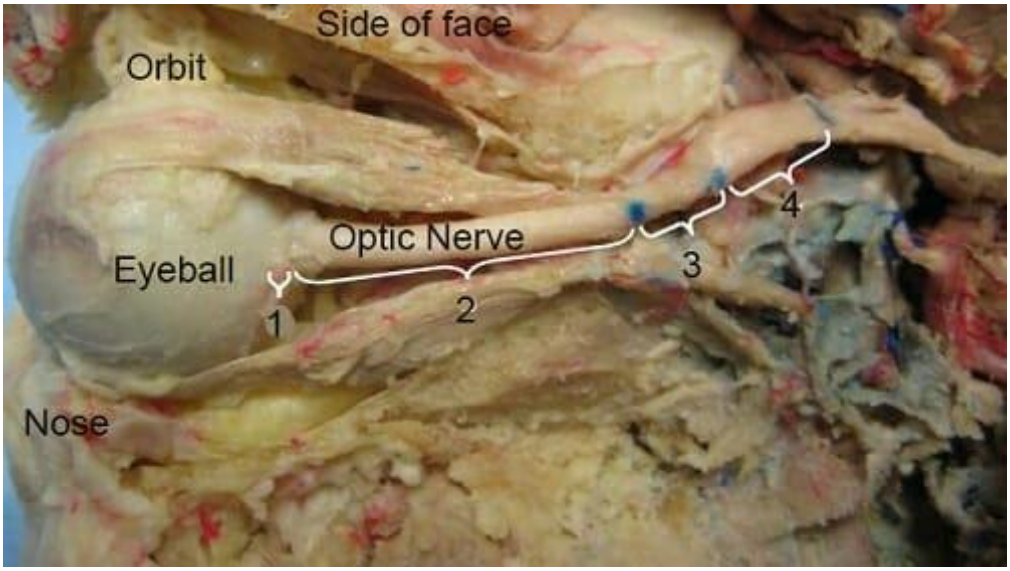
To analyse the outcome of endoscopic optic nerve decompression and prognosis in traumatic optic neuropathy patients attending Tirunelveli Medical College Hospital

REVIEW OF LITERATURE

OPTIC NERVE ANATOMY

OPTIC NERVE- PARTS

- Intraocular (1mm)
- Intraorbital (25mm)
- Intracanalicular(5mm)
- Intracranial(10mm)



INTRAOCULAR PORTION

- It includes the optic disc and that part of nerve lying within the sclera.
- The optic disc lies 3mm medial to macula lutea and just above the posterior pole of eyeball.
- It is about 1.5mm in diameter and is pale pin in color.

- It is paler than the rest of the retina.
- Its central part has a depression through which the central retinal vessels enter and leave the eye.
- Optic disc is otherwise known as optic nerve head.
- Rods and cones are completely absent at the optic disc and thus it is insensitive to light and hence referred to as blind spot.
- Optic nerve fibres exit from the eyeball via orifices of lamina cribrosa

INTRAORBITAL PORTION

- After exiting via lamina cribrosa, optic nerve acquires myelin sheaths formed by oligodendrocytes.
- This part of optic nerve has a sinuous course permitting the freedom of movement of the eyeball.
- Optic nerve is surrounded by dura mater, arachnoid mater and pia mater.
- These sheaths with an extension of subarachnoid space extend to the eyeball anteriorly.
- Posteriorly these are continuous with the meningeal coverings of the brain via the optic canal.
- Central artery and vein of the retina pierces the inferomedial surface of dural sheath about 12mm behind the eyeball.
- Before entering the optic nerve, the artery crosses the subarachnoid space obliquely.the optic nerve lies within the muscular cone formed of four

recti muscles and is surrounded by their tendinous origin at the orbital apex

INTRACANALICULAR PORTION

- Optic canal is about 5mm long and it lies within the lesser wing of sphenoid.
- Optic nerve with its three meningeal sheaths passes through optic canal.
- The dural sheath fuses with the periorbita of the optic canal and this fixes the nerve. The subarachnoid space filled with CSF covering the optic nerve is continuous with the intracranial subarachnoid space.
- ophthalmic artery and the accompanying post ganglionic sympathetic nerves passes through the optic canal on the inferolateral part of the optic nerve.
- As the nerve passes through the canal, it is separated from the sphenoidal and posterior ethmoidal sinuses on the medial side by a thin layer of bone.

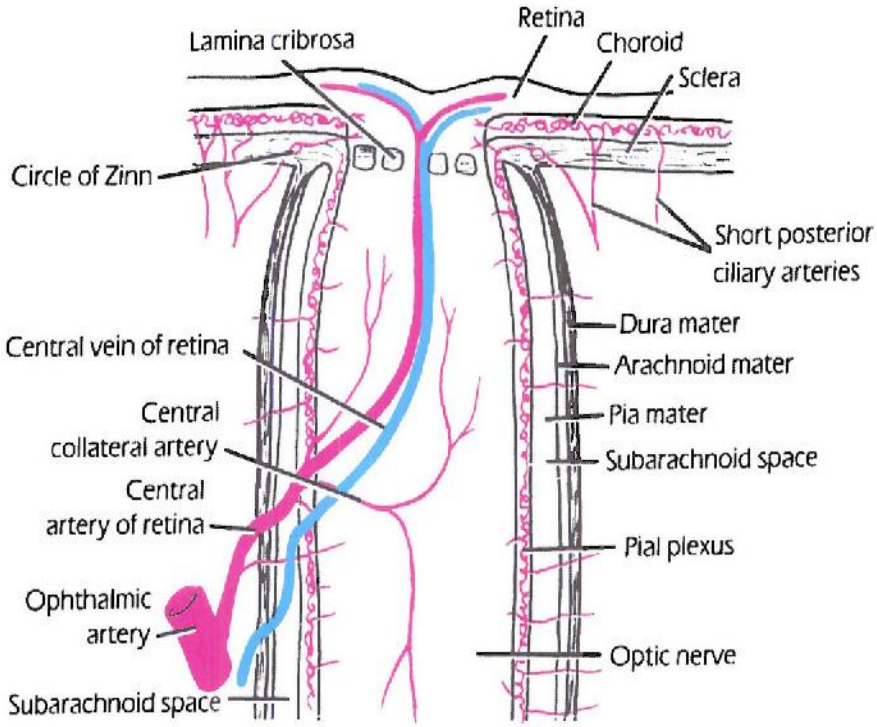
INTRACRANIAL PORTION

- After leaving the optic canal, the optic nerve passes upwards, backwards and medially in the subarachnoid space to reach the optic chiasma at the floor of the third ventricle.

- Above it is related to optic tract, anterior cerebellar artery and the gyrus rectus.
- Laterally it is related to internal carotid artery.

STRUCTURE OF OPTIC NERVE

- About 1,200,000 myelinated axons make up the optic nerve, with 90% of them having a tiny diameter (1 micrometre) and the rest having a diameter of 2 to 10 micrometres.
- The smaller axons come from the ganglion cells associated with the cones in the midbrain, whereas the larger axons come from the ganglion cells associated with the rods in the peripheral retinal areas.



Blood Supply

Intraocular Portion

- The optic nerve is nourished by branches of the Zinn anastomotic circle in the sclera.
- The blood supply for this incomplete circle comes from the short posterior ciliary arteries.
- The optic nerve's intraocular portion is not supplied by the central artery of retina.

Orbital Portion

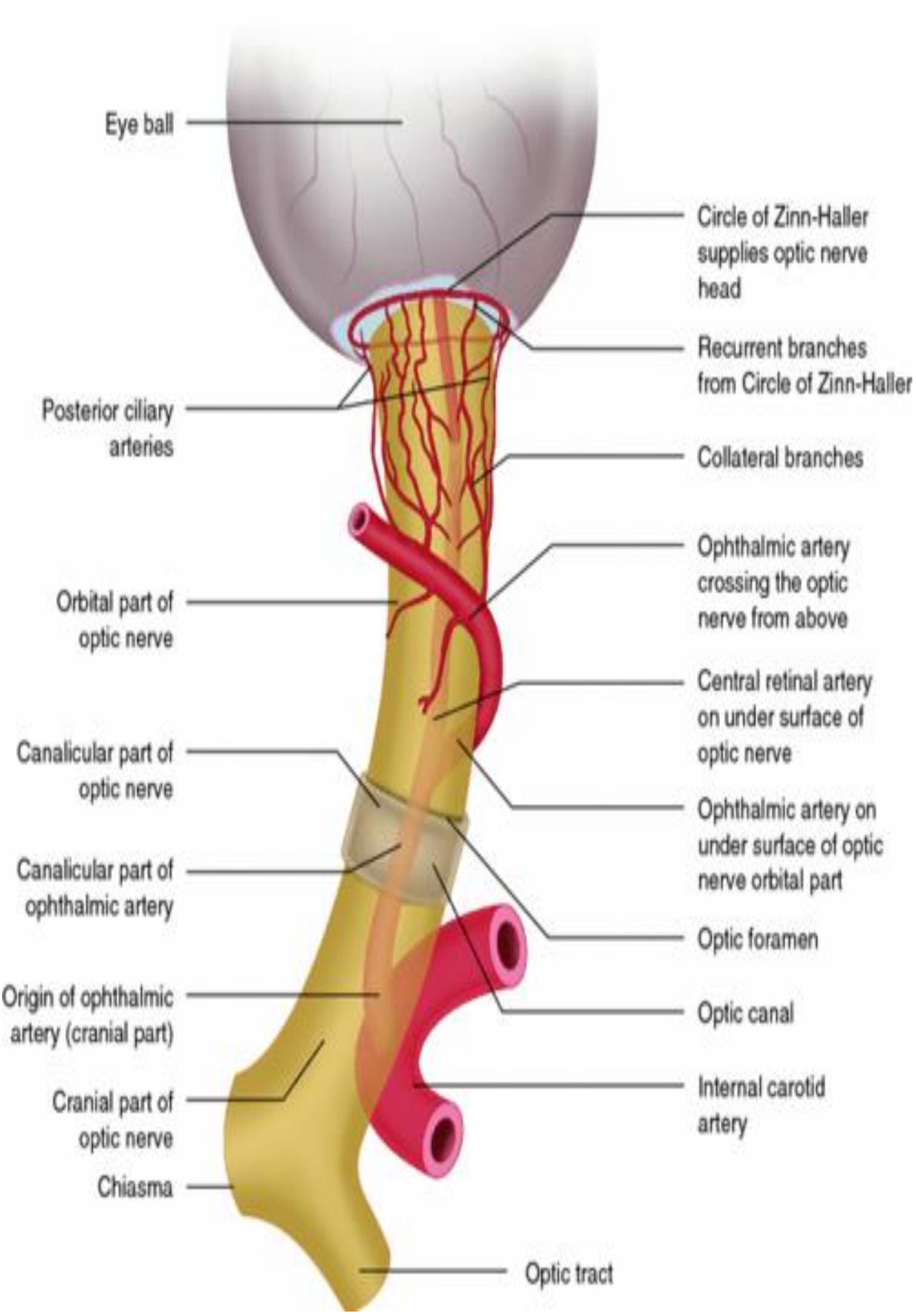
- The pial plexus of vessels supplies blood to this nerve; branches of the plexus enter into the nerve along the pial septa.
- The arterial supply for the pial plexus comes from nearby branches of the ophthalmic artery.
- A few branches come from the extra neural portion of the central artery of retina. Only a few minor branches emerge from the intraneural part of the central artery.

Intracanalicular Portion

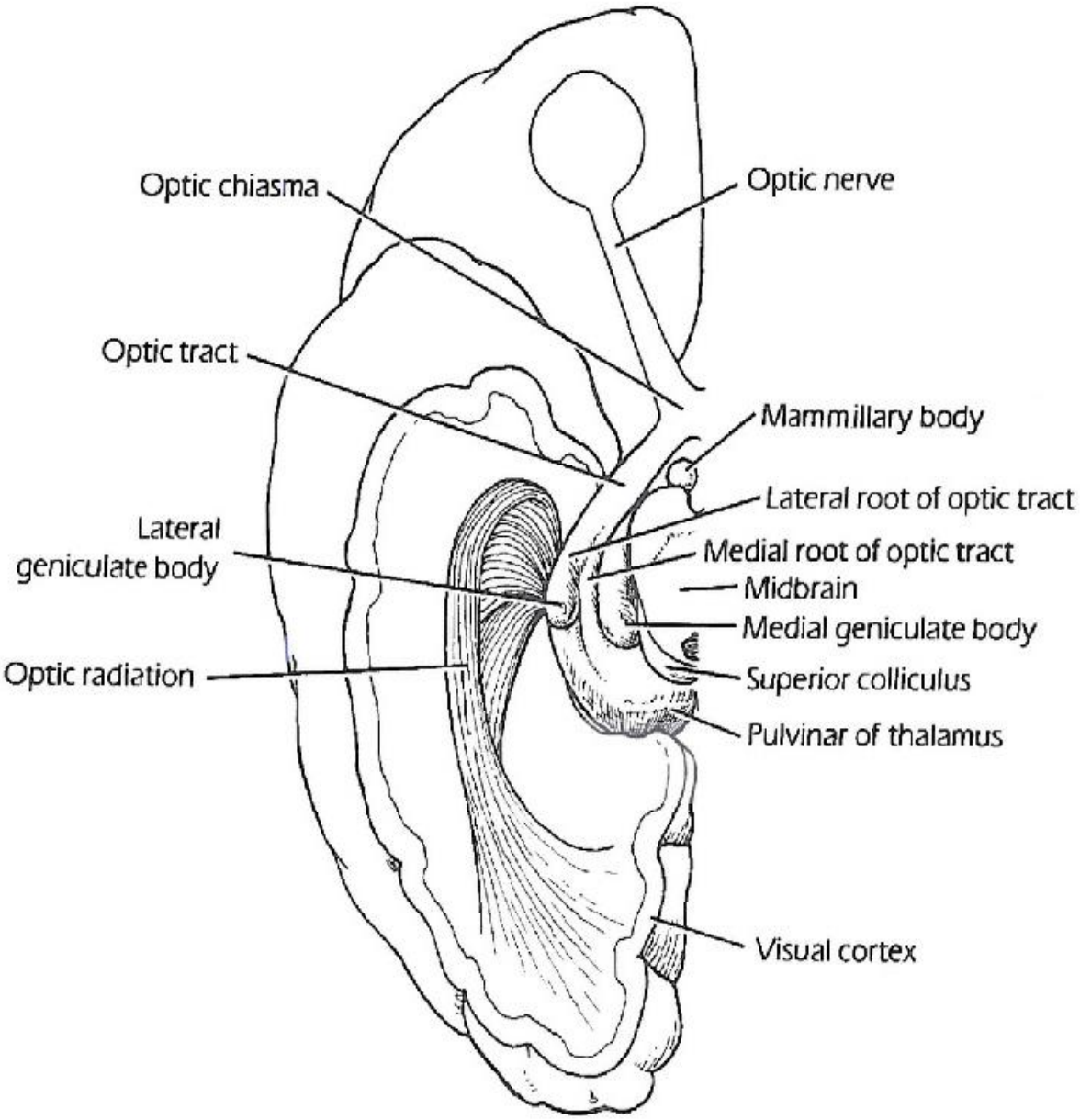
- Branches from the pial plexus supply this portion. Recurrent branches from the ocular artery join the plexus here.

Intracranial Portion

- The pial plexus also supplies blood to this region.
- The superior hypophyseal artery, branch from the internal carotid and the ophthalmic artery, supply this region of the plexus.
- The pial plexus also supplies blood to this region. The superior hypophyseal artery, branch from the internal carotid and the ophthalmic artery, supply this region of the plexus.


Eye ball
Circle of Zinn-Haller supplies optic nerve head
Recurrent branches from Circle of Zinn-Haller
Posterior ciliary arteries
Collateral branches
Ophthalmic artery crossing the optic nerve from above
Orbital part of optic nerve
Central retinal artery on under surface of optic nerve
Canalicular part of optic nerve
Ophthalmic artery on under surface of optic nerve orbital part
Canalicular part of ophthalmic artery
Optic foramen
Origin of ophthalmic artery (cranial part)
Optic canal
Internal carotid artery
Cranial part of optic nerve
Chiasma
Optic tract

VISUAL TRACT



OPTIC CHIASMA:

- It is situated at the junction of the floor and the anterior wall of the third ventricle. It is covered with pia mater and projects into subarachnoid space.
- It anterolaterally continues with optic nerves and posterolaterally continues with optic tracts.
- The nervous tissue is supplied by small branches from the plexus of vessels in the covering pia mater

OPTIC TRACTS

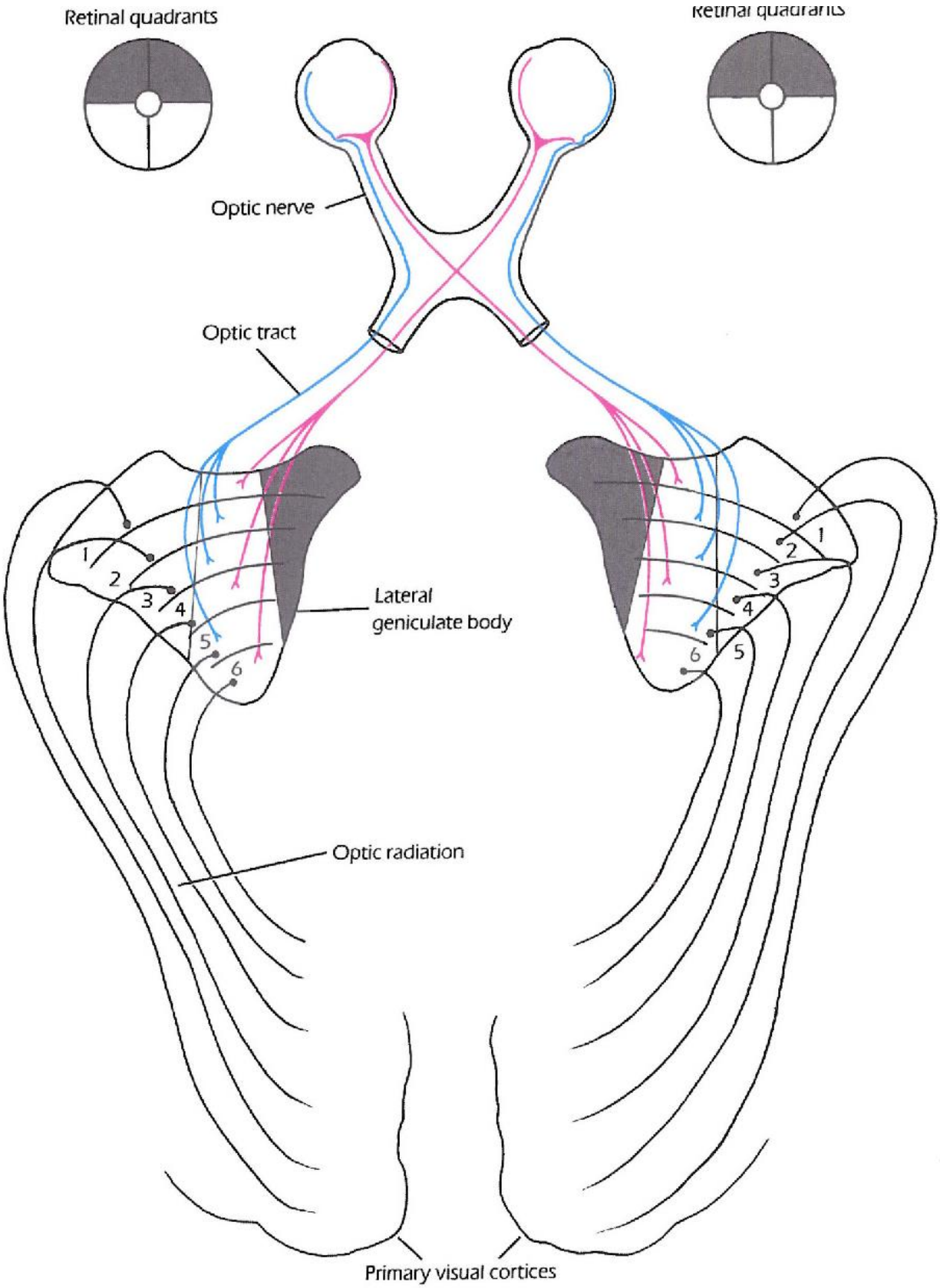
- The optic tracts develop as cylindrical bands from the posterolateral angle of optic chiasma.
- Each travels between the tubercinereum medially and the anterior perforated substance laterally.
- The tract flattens out and winds along the lateral margin of the upper half of the cerebral peduncle .
- It is attached to the midbrain here, and the uncus and parahippocampal gyrus overlap it.
- The lateral root of the optic tract is made up of nerve fibres that end in the lateral geniculate body and are concerned with conscious visual sensation.
- Fibers of unclear function are found in the smaller medial root.

LATERAL GENICULATE BODIES

- The lateral geniculate body is a tiny oval bulge on the undersurface of the thalamic pulvinar .
- The superior brachium, connects lateral geniculate body to the superior colliculus.
- Each lateral geniculate body has roughly 1 million nerve cells, about the same amount as the optic nerve and optic tract.

OPTIC RADIATIONS

- These are made up of nerve fibres that begin from the nerve cells of laminae of the lateral geniculate bodies.

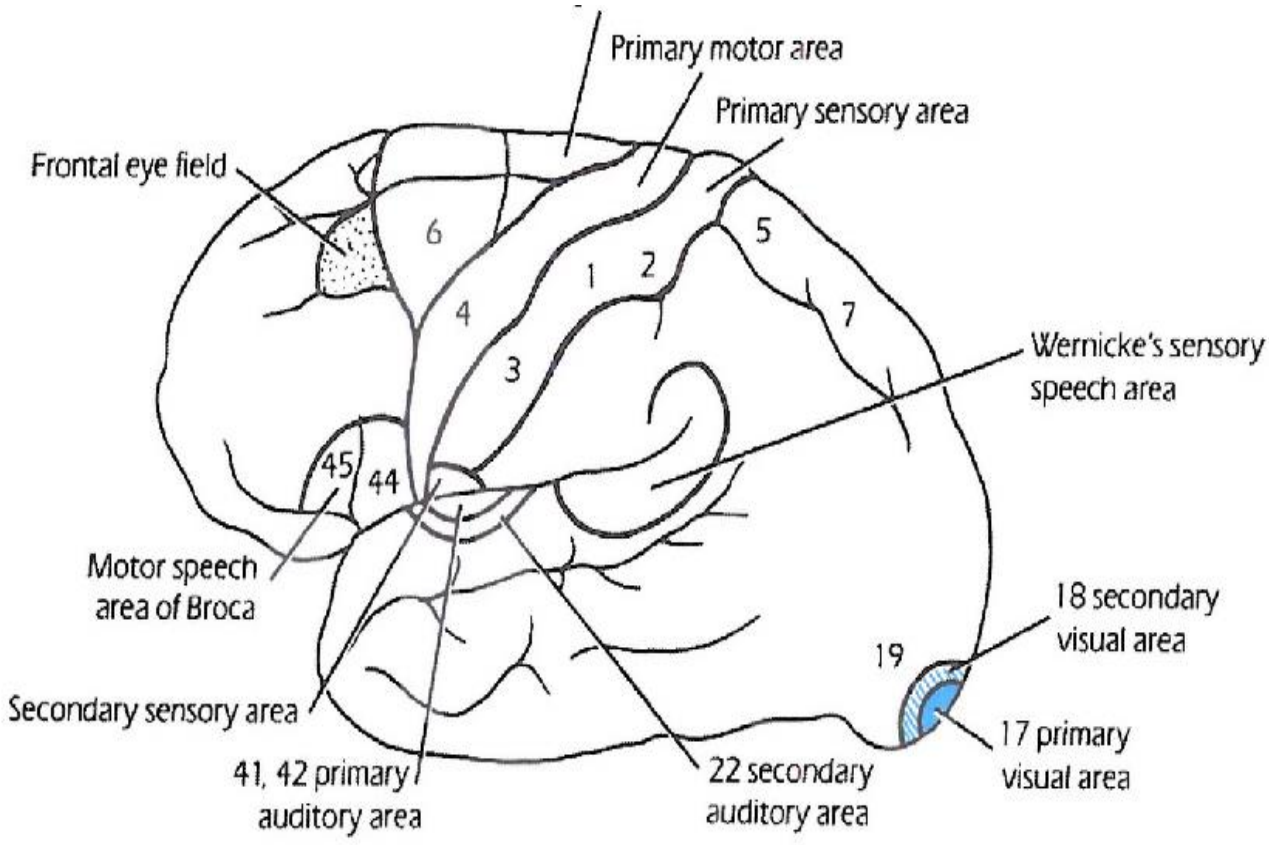

Retinal quadrants
Retinal quadrants
Optic nerve
Optic tract
Lateral
geniculate body
Optic radiation
Primary visual cortices
1
2
3
4
5
6

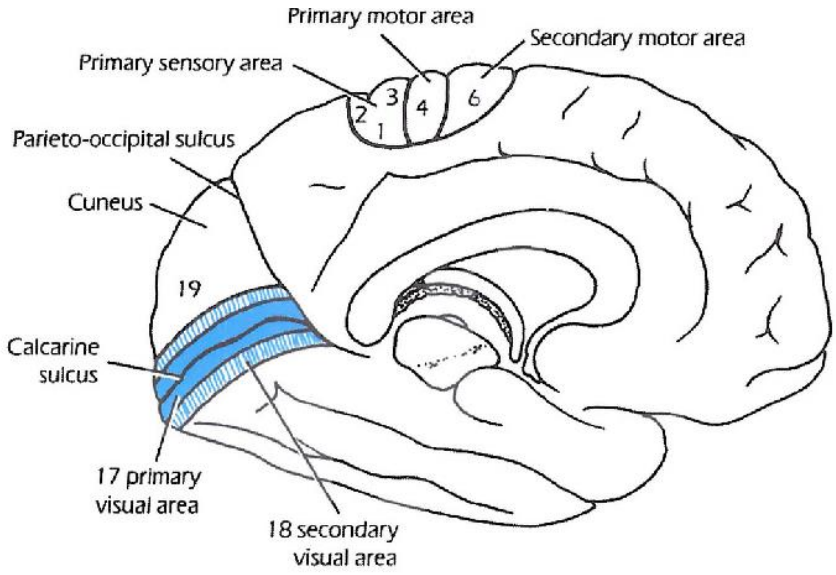
VISUAL CORTICAL AREAS

1. the primary visual area (Brodmann's area 17)
2. the secondary visual area (Brodmann's areas 18 and 19)

THE PRIMARY VISUAL AREA (BRODMANN'S AREA 17)

- The primary visual area is located on the walls of the deep calcarine sulcus on medial surface of the hemisphere and extends onto the cortex above and below the deep calcarine sulcus.
- The optic radiation carries afferent fibres from the lateral geniculate body to the primary visual cortex.





SECONDARY VISUAL AREA (AREAS 18 AND 19)

- On the medial and lateral surfaces of the hemisphere, secondary visual areas surround the primary visual area.
- The primary visual area (area 17) and other cortical areas, as well as the thalamus, send afferent fibres to the secondary association areas.
- The secondary visual area function is to connect the visual information acquired by the primary visual area to previous visual experiences, allowing the person to recognise and appreciate what he or she is seeing.
- Area 18 also connects the two parts of the visual fields via commissural fibres that cross the midline in the splenium of the corpus callosum

OTHER AREAS OF THE CEREBRAL CORTEX ASSOCIATED WITH SIGHT

OCCIPITAL EYE FIELD

- In humans, the occipital eye field is assumed to be located in the secondary visual area.
- Conjugate deviation of the eyes occurs as a result of stimulation, particularly to the opposite side.
- The eye field's function is thought to be reflex and connected with smooth eye movements when following an object (pursuit movement).
- Nervous pathways connect the occipital eye fields of both hemispheres through the corpus callosum, and they are also thought to be connected to the superior colliculus.
- The frontal eye field, on the other hand, is independent of visual stimuli and regulates voluntary fast eye movements (saccades).

FRONTAL EYE FIELD

- The frontal eye field (parts of Brodmann's areas 6, 8, and 9) extends forward from the facial area precentral gyrus into the middle frontal gyrus.
- Conjugate movements of the eyes, usually to the other side, are caused by electrical stimulation of this area.

- Nerve fibres from this location are thought to pass through the superior colliculus of the midbrain, but the exact path is unknown.
- The tectobulbar tract and the reticular formation connect the superior colliculus to the nuclei of the extraocular muscles.
- The frontal eye field, as previously indicated, is thought to control voluntary saccades and is unaffected by visual stimuli.

SENSORY SPEECH AREA OF WERNICKE

- This speech area is located in the superior temporal gyrus of the left dominant hemisphere, with extensions into the parietal region near the posterior end of the lateral sulcus.
- The arcuate fasciculus connects Wernicke's and Broca's areas via a bundle of nerve fibres.
- It gets fibres from the occipital lobe's visual cortex and the superior temporal gyrus's auditory cortex.
- Wernicke's area allows for the comprehension of written and spoken language, allowing a person to read a sentence, comprehend it, and repeat it out.

DIRECT AND CONSENSUAL LIGHT REFLEXES

- afferent neural impulses from the retina travel through the optic nerve, optic chiasma, and optic tract carry.
- A few fibres exit the optic tract and synapse with nerve cells in the pretectal nucleus, which is located near the superior colliculus.
- Axons of pretectal nerve cells transmit impulses to the parasympathetic nuclei (Edinger-Westphal nuclei) of the oculomotor nerve on both sides
- . The fibres synapse here and go to the ciliary ganglion in the orbit through the oculomotor nerve.
- Finally, postganglionic parasympathetic fibres get to the eyeball and the constrictor pupillae muscle via the short ciliary nerves.
- Because the pretectal nucleus sends fibres to the parasympathetic nuclei on both sides of the midbrain, both pupils contract in the consensual light reflex.

ACCOMMODATION REFLEX

- When the eyes are directed from a far to a near object, the ocular axis are brought together by contraction of the medial recti, the lens thickens due to contraction of the ciliary muscle, and the pupils constrict to limit light waves to the thickest central area of the lens.
- Afferent impulses travel to the visual cortex via the optic nerve, optic chiasma, optic tract, lateral geniculate body, and optic radiation.

- The eyefield of the frontal cortex is related to the visual cortex.
- Cortical fibres descend to the oculomotor nuclei in the midbrain via the internal capsule.
- The medial rectus muscles are innervated by the oculomotor nerve.

ANATOMY OF ORBIT

- It is pyramid shaped with a volume of 30 ml.
- The medial walls of two orbits are parallel to each other and the lateral walls are 90 degrees to each other and 45 degrees with the lateral wall.
- The orbits are lined by periosteum

BONY COMPOSITION OF ORBITAL WALLS

Floor of the orbit

- Maxillary bone
- Palatine bone
- Zygomatic bone

Medial wall of the orbit

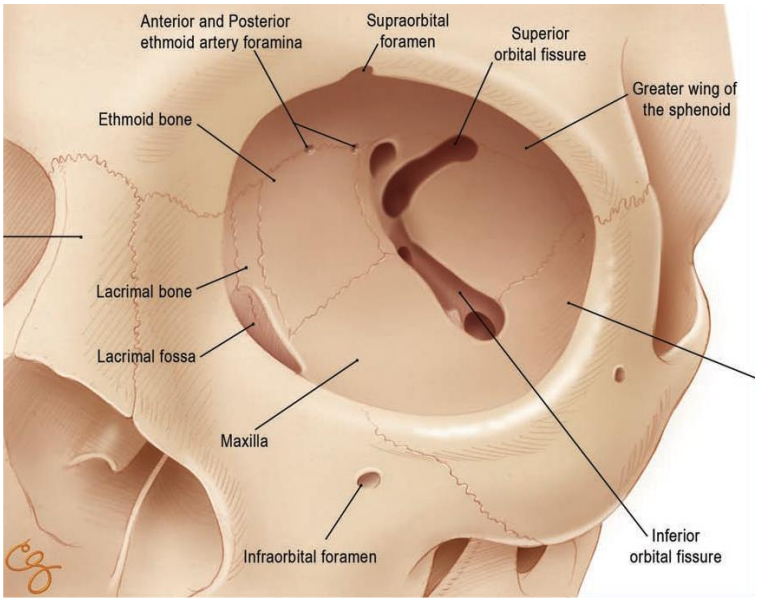
- Maxillary bone
- Sphenoid bone
- Ethmoid bone
- Lacrimal bone

Roof of the orbit

- Lesser wing of the sphenoid
- Frontal bone

Lateral wall of the orbit

- Greater wing of the sphenoid
- Zygomatic bone



OPTIC FORAMEN

- The optic foramen contains the optic nerve, the ophthalmic artery, and sympathetic fibers from the carotid plexus.

SUPERIOR ORBITAL FISSURE

- The greater and lesser wing of the sphenoid form the superior orbital fissure.

- The origin of the lateral rectus divides the superior orbital fissure into superior and inferior sections.
- The superior section transmits the cranial nerves ophthalmic and frontal and lacrimal branches of trochlear nerve.
- The superior and inferior divisions of cranial nerve III, the nasociliary branch of cranial nerve ophthalmic, abduscent nerve, the superior ophthalmic vein, and the sympathetic nerve plexus are all transmitted via the inferior half.

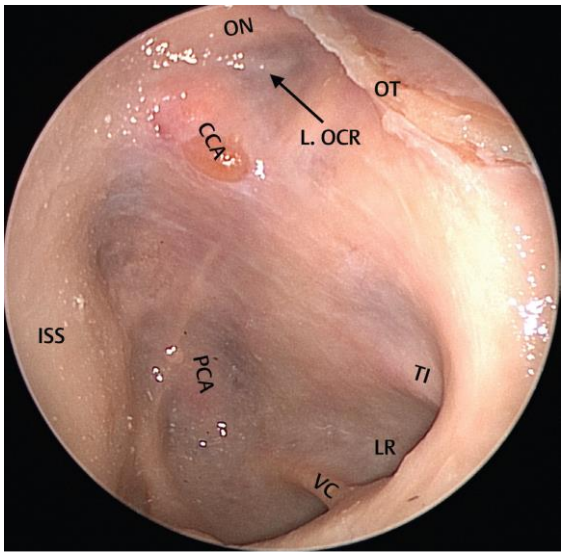
INFERIOR ORBITAL FISSURE

- The inferior orbital fissure is located between the lateral wall and floor of the orbit. It carries branches of the maxillary nerve as well as the inferior ophthalmic vein.

SURGICAL ANATOMY

- The optic canal is formed by the two struts of the lesser wing of sphenoid, and it contains the optic nerve as well as the ophthalmic artery.
- Unlike a peripheral nerve, the optic nerve continues directly from the brain and contains all three meningeal layers: pia, arachnoid, and dura.
- If the sheath is incised, a CSF leak is possible, and this must be carefully considered. The carotid canal is located just below the optic canal.
- The anatomic proximity of these two essential structures along the lateral wall of the sphenoid sinus is determined by the degree of sphenoid sinus/lesser sphenoid wing pneumatization.
- The optic nerve passes through a posterior ethmoid cell, also known as sphenoethmoid or Onodi cell, in some cases.
- This crucial relationship must be understood by the surgeon in order to ensure correct optic nerve identification and avoidance of injury.
- Although the ophthalmic artery usually runs superolateral to the optic nerve and out of the surgical field, in about 20% of orbits, it runs inferior and more medial to the optic nerve.

LEFT SPHENOID SINUS



- The vidian canal (VC) and trigeminal impression (TI) for the trigeminal nerve's maxillary division are clearly visible.
- The lateral recess (LR), a dip in the lateral sphenoid wall between the VC and the TI, can be visible if the sphenoid sinus is strongly pneumatized. It is possible to see the lateral opticocarotid recess.
- The pneumatization of the optic strut corresponds to this depression (the bony bridge that separates the optic canal from the superior orbital fissure).
- Further pneumatization of the optic strut could lead to a pneumatized anterior clinoid process, putting the optic nerve on a mesentery.
- The optic tubercle (OT) is a bone that connects the orbital apex with the sphenoid sinus.

EPIDEMIOLOGY

- overall incidence of TON after blunt or penetrating trauma is 0.7- 2.5%.
- indirect TON has a higher prevalence than direct TON
- The intracanalicular region (71.4 percent) is the most prevalent site of indirect TON, followed by the orbital apex (16.7 percent).
- In 11.9 percent of the cases, both the intracanalicular segment and the orbital apex were involved.
- Another common site for optic nerve traumatic injury is the intracranial part of the optic nerve next to the falciform ligament.
- Up to 80% of TON patients have been recorded to be male, with a median age of 31 years, while 21% are under the age of 18.
- Fall (26%) is the most common cause of TON among the general population, followed by motor vehicle accidents (21%), and assaults (21%).
- there is a strong association between TON and head injury, with all TON patients suffering from head injuries (two-thirds of them have a significant head injury)
- In the paediatric group, the most common causes of TON are falls (50 percent) and motor vehicle accidents (40 percent).

Author	Cases	Motor Vehicle	Bicycle	Fall	Assault	Others
Lubben(28)	65	15(23%)	10(15%)	-	7 (11%)	33(51%)
Millesi(26)	29	18(62%)	-	6(21%)	5(17%)	-
Seiff(30)	36	15(42%)	-	13(36%)	4(11%)	4(11%)
Matsuzaki(29)	33	20(60%)	-	7(21%)	4(12%)	2(6%)
Kountakis(27)	34	18(53%)	1(3%)	9(26%)	6(18%)	-

- Following traumatic optic neuropathy, the percentage of patients with light perception and no light perception vision ranges from 43 percent to 78 percent.

TYPES OF TON (MODE OF INJURY)

There are two types of TON:

- indirect
- direct.

INDIRECT TON

- Trauma to the head or face causes energy from the force of impact to be passed to the bone structures that carry the optic nerve, resulting in indirect TON.
- Sheering forces can impair the nerve or the pial vascular system

DIRECT TON

- Damage induced by mechanical forces delivered directly to the nerve through avulsion or laceration, or impingement of the nerve from numerous sources, such as a penetrating foreign body, displaced fracture fragment, or optic canal fracture, is referred to as direct TON.

ANATOMICAL CLASSIFICATION

ANTERIOR INJURY

- Visual loss associated with injuries that occur anterior to where the central retinal artery enters the optic nerve, resulting in abnormalities in the retinal circulation.

POSTERIOR INJURY

- Posterior optic nerve injuries are the injuries occurring posterior to the site of the entry of central retinal vessels to the optic nerve

PATHOLOGY

Pringle performed 174 autopsy on individuals who were rendered unconscious after a head injury and died. Blood was seen in the optic nerve sheath in 16 cases, leading to the conclusion that an indirect damage to the optic nerve was the cause i.e the optic nerve was compressed due to a bleed. Hughes' research backs up this assertion who looked at six nerves in five different individuals.

Crompton described the visual lesions found in 84 autopsies conducted shortly after closed head trauma. In 69 of the 84 cases, there were optic nerve

dural sheath haemorrhages. In 30 of the 84 cases, interstitial optic nerve haemorrhages were found. In 20 of the 30 cases, the interstitial haemorrhage was seen in the optic canal. In 37 of the 84 cases (44 percent), shearing lesions and ischemia necrosis were observed, and the intracanalicular optic nerve was implicated in 30 of the 37 cases. In 20 of the 37 cases, shearing lesions and ischemic necrosis were also found in the intracranial optic nerve. Skull fractures were found in 27 of the 46 cases of optic nerve damage in Turner's study. Only four individuals had an abnormality of the optic canal, indicating the rarity of canal fractures, at least based on plain film radiography.

Sphenoid fractures have been found to be 50 percent common in cases of traumatic optic neuropathy, according to investigations using computed tomography. These investigations also show that traumatic optic neuropathy can arise even when there is no evidence of an optic canal fracture.

Forces applied to the frontal bone are transferred and focused in the area of the optic canal, according to laser interferometry studies. Over milliseconds, the full force of deceleration can be loaded onto the face bones. The sphenoid's elastic deformation causes force to be transferred into the intracanalicular optic nerve, which is tightly bound, producing contusion necrosis of the nerve by damaging axons and vasculature (79). The elastic limit of the affected bone determines the location of a fracture. Bone that is thinner is more likely to deform. Thick bone, on the other hand, is inelastic and more prone to fracture. Although canal fractures sometimes occur, it appears that harm to the optic

nerve by displaced bone fragments is uncommon. The falciform dural fold that overlies the sphenoid plane or where the nerve becomes fixed entering the intracranial aperture of the optic foramen might also damage the intracranial optic nerve.

Walsh hypothesized that enlargement of the optic nerve within the bone canal could make the nerve's intracanalicular part vulnerable to ischemic injury.

PATHOGENESIS

Apoptotic degeneration occurs in the some of wounded and presumably intact neighbouring neurons. Ischemic optic neuropathy, experimental glaucoma, and optic nerve damage have all been associated with this form of degeneration Apoptosis, or planned cell death refers to a number of secondary injury mechanisms which leads to further axonal death after the initial injury. this apoptotic loss can be stopped by adequate treatment which preserves axons.

Perhaps the most important aspect of secondary injury after trauma is ischemia. The generation of oxygen free radicals by partial ischemia and reperfusion of transiently ischemic areas causes reperfusion injury.

Axons have significant polyunsaturated lipid contents in their cell membranes. Peroxidation of these lipids occurs as a result of the generation of oxygen free radicals as a result of trauma or ischemia, causing damage to the neural membrane. Cell death after ischemia or severe injury is hypothesised to be mediated by this mechanism.

Arachidonic acid is converted into thromboxane, PGE1, PGF2, hydroperoxides, and oxygen free radicals when it is liberated from injured cells. Platelet adhesion and microvascular sludging are induced by thromboxane. Following optic nerve injury, direct thrombin inhibition has been proven to be neuroprotective. Treatment with cyclooxygenase inhibitors or antioxidants can also help to reduce the degree of hypoperfusion after a brain or spinal cord injury.

The generation of xanthine oxidase and the release of elemental iron from bleeding are two other potential processes that can lead to oxygen free radical production after trauma or ischemia. The superoxide ion and hydrogen peroxide (H2O2) are linked to iron, which acts as a redox metal and produces the hydroxyl radical or feryl ion, which can oxidise a wide range of substances, including lipids.

Bradykinin and kallidin are proteins that are activated in response to injury and play a role in the formation of free radicals, changes in intracellular calcium regulation, and the release of arachidonic acid from neurons. Kallikrein enzymes catalyse the activation of kinins. When blood is exposed to collagen and basement membrane after tissue injury, the intrinsic coagulation cascade is activated. This cascade activates kallikrein, which converts kininogens of different molecular weights to bradykinin and kallidin, respectively.

Early inflammation is mediated by kinins, which causes smooth muscle contraction, venous constriction, arteriole dilation, and edoema, as well as stimulation of nociceptive receptors. Bradykinin activates phospholipid metabolism by binding to cell membrane receptors. Arachidonic acid is released from the neuron as a result of these cellular modifications. The release of arachidonic acid from neurons is triggered by bradykinin, and the resultant prostaglandins, oxygen-containing free radicals, and lipid peroxides cause a loss of cerebrovascular autoregulation.

CLINICAL ASSESSMENT

- Traumatic optic neuropathy is still a medical condition.
- A history of trauma is required by definition.
- It's not uncommon to experience a loss of consciousness as a result of the condition.
- In the presence of normal vision and pupillary function, an optic nerve injury diagnosis should be avoided.
- Respiratory and cardiovascular resuscitation are the first priorities after a trauma.
- Other clinical factors may limit the initial ocular examination. The eye examination should be completed as soon as possible.

HISTORY

- The clinical evaluation of a patient with sight loss as a result of trauma should begin with as much information as feasible about the patient's medical history.
- When the patient is unconscious, significant persons should be questioned about the patient's past.
- To rule out visual loss prior to the incident, an ocular history must be investigated.
- A thorough medical, pharmacological, and allergy history is also required.
- Tetanus is a risk of open injury, so the patient's tetanus immunisation status should be checked.

EXAMINATION

VISUAL ACUITY

Following indirect optic nerve injuries, visual acuity is frequently diminished. In Hughes' set of 56 instances, none of the patients had any light perception. Turner's 46 patients all had no light perception on the side where the optic nerve had been injured. In Hooper's study, 14 of the 21 individuals had no light perception. Edmund observed that 17 of 22 patients in his series had no light perception. While vision better than 20/400 is not impossible in traumatic optic neuropathy, it is uncommon. The rate of delayed visual loss has been estimated to be as high as 10%.

PUPILLARY REFLEXES

The existence of an afferent pupillary deficit is required for the diagnosis of traumatic optic neuropathy in cases of unilateral traumatic optic neuropathy.

FUNDOSCOPY

It's important to look for signs of a penetrating ocular injury. A hyphema or angle recession can arise from a blunt lesion to the iris. Trauma can cause the lens to dislocate. Blood in the vitreous may obscure the fundus later. Before dilating the eyes, consult the treating neurosurgeon or trauma surgeon if the patient is neurologically unstable. A dilated fundus exam to rule out retinal tears will have to wait until the patient is neurologically stable.

An adequate fundus examination will involve an assessment of retinal circulation anomalies. A ring of bleeding or the appearance of a deep circular pit appears at the site of partial or total avulsion of the optic nerve head. Venous blockage and traumatic anterior ischemic optic neuropathy are caused by anterior injuries between the globe and the site where the central retinal arteries enter the optic nerve, causing problems in the retinal circulation. The circulation of the retina may be unaffected by haemorrhages in the optic nerve sheath posterior to the origin of the central retinal arteries, although the optic nerve head may swell. Despite the existence of traumatic optic neuropathy, Frank papilledema can occur in the presence of elevated intracranial pressure. Visual loss could be caused by choroidal rupture or commotio retinae. In the absence

of intraocular pathology, lower visual acuity and an afferent pupillary defect should indicate a posterior orbital, intracanalicular or intracranial optic nerve injury.

VISUAL EVOKED POTENTIAL

In an unresponsive patient suspected of having traumatic optic neuropathy, the visual evoked potential (VEP) may be useful. This is particularly true in bilateral situations where an afferent pupillary deficiency may not be seen. The VEP is only useful in cases where it is not recordable, in which case visual recovery is extremely unlikely. In an amaurotic eye immediately after optic nerve trauma with eventual extinction, the VEP is usually abnormal but recordable . The false-positive VEP is thought to be caused by a small number of still undamaged axons that can conduct.

VISUAL FIELD

Only when sufficient vision is present following optic nerve injuries can visual fields be obtained. The visual field gives a knowledge of the localisation of optic nerve damage due to the retinotopic organisation of the optic nerve. The pial penetrating vessels that supply blood to the optic nerve within the canal are subjected to shearing stresses at the time of injury. These pial vessels are regarded to be the most vulnerable to shearing forces because the superior segment of the optic nerve is most closely bound within the canal. Visual field deficits are caused by partial avulsion of the optic nerve at the globe, which

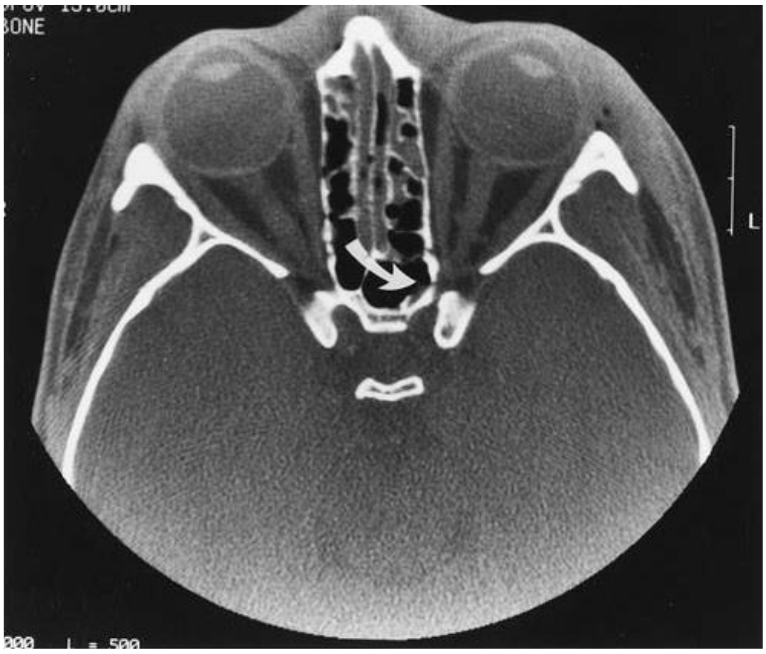
correlate to the avulsion location. There is, however, no pathognomonic visual field loss that can be used to diagnose optic nerve injuries. Visual field testing can be used to document the return of vision after an injury.

IMAGING

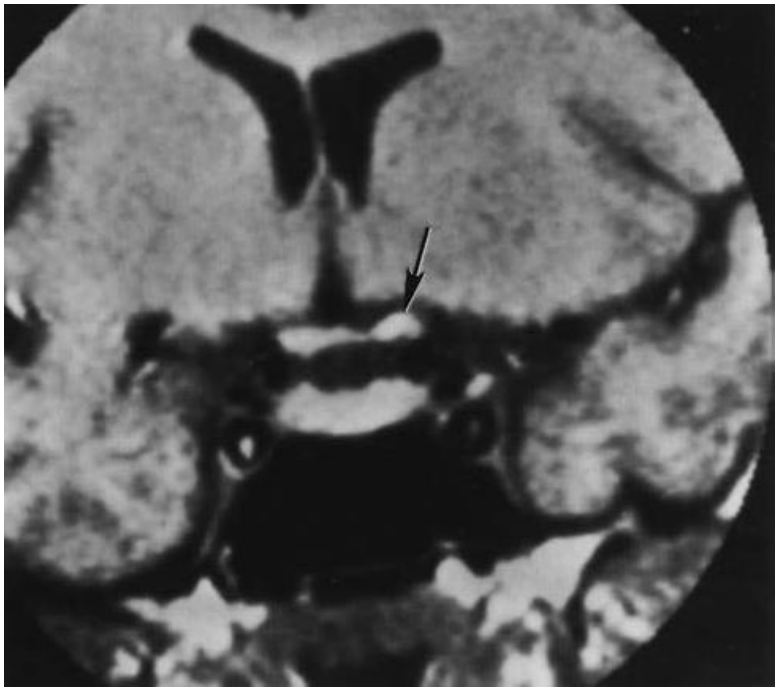
CT scanning has demonstrated specific pathologies implicated in the disruption of optic nerve nerve function, including an optic nerve sheath haemorrhage and a suspected arachnoid cyst. While CT scanning definitely outperforms magnetic resonance imaging (MRI) in identifying bone fractures, MRI outperforms CT scanning in imaging soft tissue. A comprehensive evaluation of a particular clinical issue often necessitates the use of both CT and MRI.

A word of caution: MRI should only be used after a CT scan or conventional x-ray has ruled out the presence of a metallic orbital or intraocular foreign body. Chiasmal trauma can be assessed with MR imaging.

CT ORBIT SHOWING LEFT OPTIC CANAL FRACTURE IN LATERAL WALL OF LEFT SPHENOID:



CORONAL MRI BRAIN AT THE LEVEL OF OPTIC CHIASMA DEMONSTRATING HEMORRHAGE IN OPTIC NERVE SHEATH :



DIFFERENTIAL DIAGNOSIS:

Apart from acute optic neuropathy, other causes of visual loss after trauma must be explored. The differential diagnosis of visual loss with disrupted pupillary function can be limited with a detailed history and thorough ocular examination. Other ocular injuries may be accompanied by an optic nerve injury. If the loss of vision and abnormal pupillary function cannot be explained by evident ocular injury, traumatic optic neuropathy should be suspected.

Minor head injuries might also result in acute optic neuropathy. It's equally important to be aware of visual loss that isn't caused by trauma. It's possible that monocular vision loss won't be recognised until the person closes the unaffected eye. In this case, the patient may blame the loss of vision on a recent traumatic occurrence. It can be tough to tell whether your vision loss is due to a coincidence or a recent trauma. Any process that can cause vision loss, such as decompensation of a vascular aneurysm, orbital or optic nerve inflammation, anterior ischemic optic neuropathy, or acute sinus disease with orbital involvement, can be linked to a traumatic event. Atraumatic cavernous sinus fistula is usually accompanied with many orbital abnormalities, making it easier to distinguish from traumatic optic neuropathy.

PHARMACOLOGY

Corticosteroids are expected to reduce the rate of protein synthesis at the doses commonly used in clinical practise. This effect is mediated by cytosolic receptor proteins.(12) This steroid-receptor complex induces the production of

an inhibitory protein in lymphocytes.(13) Furthermore, glucocorticoids reduce the formation of prostaglandin endoperoxides and thromboxane by inhibiting the release of arachidonic acid from phospholipids.(14)

The untreated, contused portion of the spinal cord shows a rise in fluorescent lipid peroxidation products and amounts of cyclic GMP, a sensitive biomarker of lipid peroxidation, following experimental spinal cord injury(15). There is a considerable reduction in fluorescent lipid peroxidation products in similarly damaged animals treated with 30–60 mg/ kg intravenous methylprednisolone 30 minutes after injury compared to untreated injured controls. Methylprednisolone dosages of 15–30 mg/kg reduce the increase in cyclic GMP that occurs after injury without treatment.

In this dosing range, methylprednisolone reduces free radical pathology (16). Following spinal cord injuries, animals given 30 mg/kg methylprednisolone maintain normal spinal cord blood flow, preventing ischemia (17,18). Massive dosages of steroids (30 mg/kg) appear to have a different pharmacology. The antioxidant impact appears to be the most important, minimising free radical disease (159). Because one molecule of antioxidants is consumed for each free radical created, very high doses of steroids may be necessary (16). The neural tissue is protected against the secondary consequences of trauma by limiting lipid peroxidation, which is caused by free radical production (17,18).

The NASCIS II (19) study involved individuals with acute spinal cord injury in a multicenter, randomised, double-blind, placebo-controlled trial. Within 12 hours of damage, patients were randomly assigned to one of three therapy groups. Placebo, naloxone, and methylprednisolone were the therapy options. Naloxone, an opiate receptor partial agonist that has been shown to reduce neuronal damage in animals, was given as a 5.4 mg/kg bolus followed by a 4.0 mg/kg/hr continuous infusion rate. An initial dose of 30 mg/kg of methylprednisolone was given, followed by a continuous infusion of 5.4 mg/kg/hr. These treatments were given for a total of 24 hours. Pinprick, light touch, and motor function were used to assess neurological function, and neurologic scores were assigned based on these examinations.

Patients were evaluated at the beginning of the study, at 6 weeks, and at 6 months. When compared to placebo-treated individuals, this study found that therapy with methylprednisolone within 8 hours after damage at the therapeutic level suggested by animal spinal injury studies resulted in a significant improvement in motor and sensory function. When compared to placebo-treated patients, patients treated with naloxone and patients treated with either medicine more than 8 hours after injury showed no improvement in neurologic scores. According to a post-hoc review of the NASCIS II data, methylprednisolone treatment started more than 8 hours after injury was harmful . Patients who received methylprednisolone had a higher rate of infection (7.1%) than those who received naloxone (3.3%) or those who received placebo (3.6%) in this

trial, although the difference was not statistically significant. The efficacy of methylprednisolone in the treatment of spinal cord injury prompted ophthalmologists to adopt this method empirically for optic nerve trauma

MANAGEMENT

Early Japanese papers claimed that traumatic optic neuropathy was more common and more responsive to surgical treatments in Japan (20,21).

Fujitani and colleagues published a 16-year study that included 110 cases of traumatic optic neuropathy (22). In their study, 43 individuals were treated medically with corticosteroids, and 70 eyes were decompressed transethmoidally. Oral prednisone 60 mg, tapered over two weeks, was the medical treatment. After 1972, patients in the series who did not react had surgical decompression. In medically treated cases, the rate of visual improvement was 44.2 percent (19/43). The initial visual acuity of nine eyes was no light perception. Medical treatment had no effect on their eyes. Overall, eyes treated within three weeks of damage improved at a rate of 57 percent. Only 15% (2/13) of those who started treatment more than 3 weeks after their injury improved.

Seventy optic nerves were decompressed surgically, and canal fractures were discovered in 25% of patients at the time of surgery. In these situations, the total rate of improvement was 47.7% (33/70). Twenty-eight of the patients receiving surgery had no light perception at the start. In seven of the twenty-eight cases, there was a visual improvement (25 percent). In 38 patients, optic

canal decompression was performed within three weeks, with a 45 percent improvement rate (17/38). After medicinal treatment failed to improve vision in 32 eyes, surgical decompression was performed after three weeks. In these patients, 16 of 32 eyes (50%) showed improved vision following surgery. Surgery was not performed in four cases until three months following the incident. Despite the delay, three of the patients vision improved after surgery.

The widespread use of orbital imaging and neuroimaging has resulted in a more precise approach of assessing the mechanisms of vision loss. This has resulted in a far more detailed anatomical approach to surgical intervention planning(23). Imaging can reveal disease that, if treated surgically, can lead to visual improvement. Computed tomography has also revealed that bone fragments are impinging on the optic nerve. Anecdotally, surgical reduction of such bone fragments has resulted in vision recovery(24).

REPORTED SPONTANEOUS RECOVERY IN TRAUMATIC OPTIC NEUROPATHY

AUTHOUR	NO. OF PATIENTS	SPONTANEOUS IMPROVEMENT %
Tang (37)	13	38
Millesi(26)	7	57
Lessel (33)	25	20
Levin(10)	9	57
Seiff(30)	15	33

MEDICAL THERAPY FOR TRAUMATIC OPTIC NERVE INJURY

Following the spinal cord injury care protocol, mega-dose intravenous methylprednisolone is now employed. A loading dosage of 30 mg/kg of methylprednisolone is given intravenously (IV), followed by a 5.4 mg/kg/hour infusion(25). The patient's visual acuity is checked every hour, and surgical intervention is considered if any of the following conditions are met:

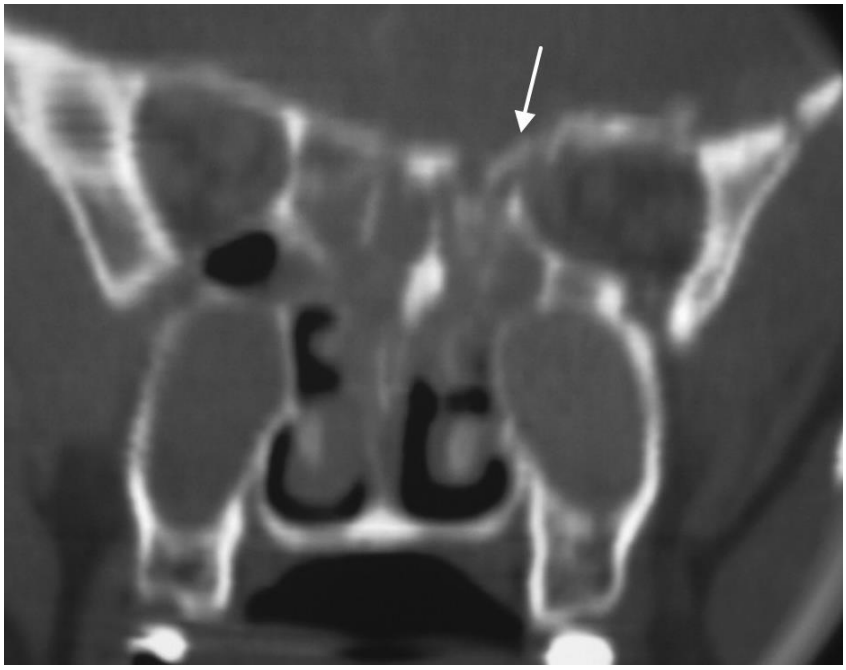
- Fracture of optic canal on CT scan with vision less than 6/60
- Fracture of the optic canal with vision . 6/60 but the patient’s vision deteriorates on steroids
- Vision is 6/60 (or there is a deterioration of vision) after 48 hours of steroid treatment with probable canal injury

REPORTED PROGNOSIS FOR CORTICOSTEROIDS

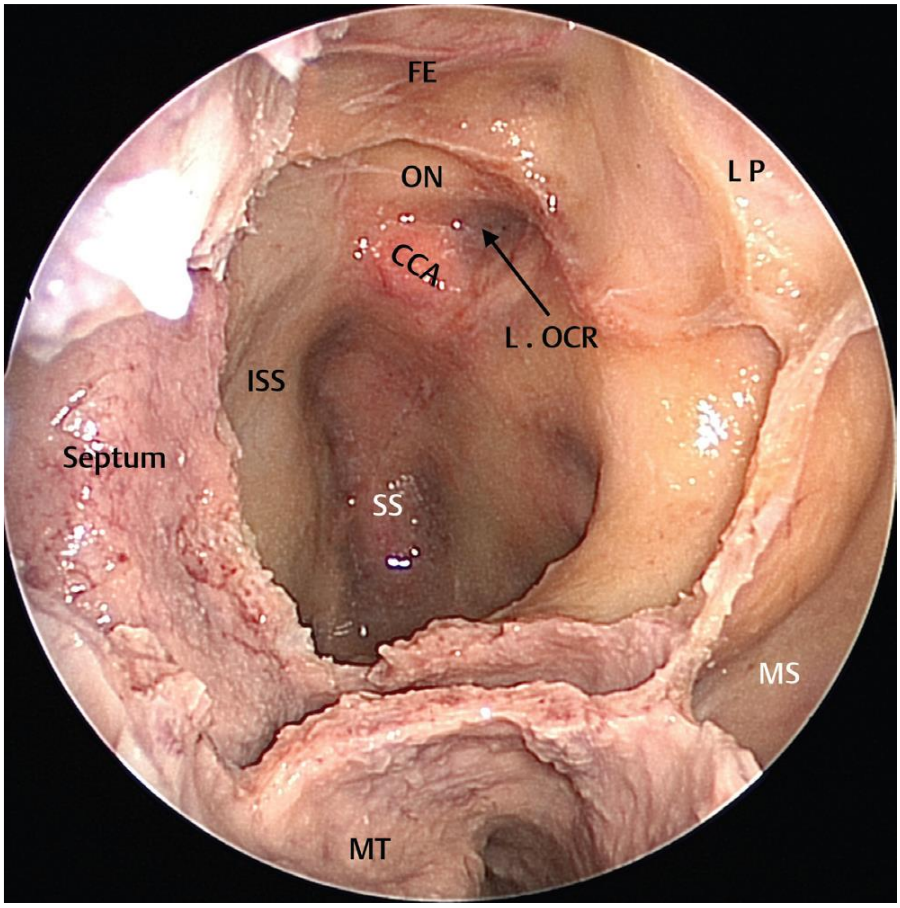
AUTHOR	NO. OF PATIENTS RECEIVED CORTICOSTEROIDS	VISUAL IMPROVEMENT %
Seiff (30)	21	62
Levin (10)	85	52
Spoor(32)	22	86
Bendel(31)	17	100

SURGICAL TECHNIQUE FOR TRAUMATIC OPTIC NEUROPATHY

- Endoscopic optic nerve decompression should be prepared in the same way as standard endoscopic sinus surgery.
- To understand the location of the optic canal, carotid artery, and accessibility of the intracanalicular section of the nerve, a thorough preoperative review of multiplanar CT imaging is required.
- Preoperative surgical planning made possible by multiplanar imaging is helpful, and intraoperative anatomy confirmation is crucial.
- The nose is decongested and infiltrated as part of the normal procedure.
- An uncinectomy is performed with the maxillary ostium exposed.
- The agger nasi cell is removed through an axillary flap.
- This makes it easier to get to the base of the skull.
- In the area above the bulla ethmoidalis, the fovea ethmoidalis is exposed.
- If the cells in the frontal recess are disrupted or there is cause to believe the frontal recess is obstructed, this will be cleared; otherwise, the cells in the frontal recess will be left alone.
- The entire skull base may be movable in some patients with extensive sinus fractures.



- The posterior ethmoid cells in most patients will be full of blood, and when this is combined with the lamina papyracea and skull base mobility, the surgeon may become disoriented.
- As a result, only highly skilled endoscopic sinus surgeons should perform this procedure. A sphenoidotomy and posterior ethmoidectomy should be performed.
- The posterior lamina papyracea and fovea ethmoidalis should be identified in the posterior ethmoids.

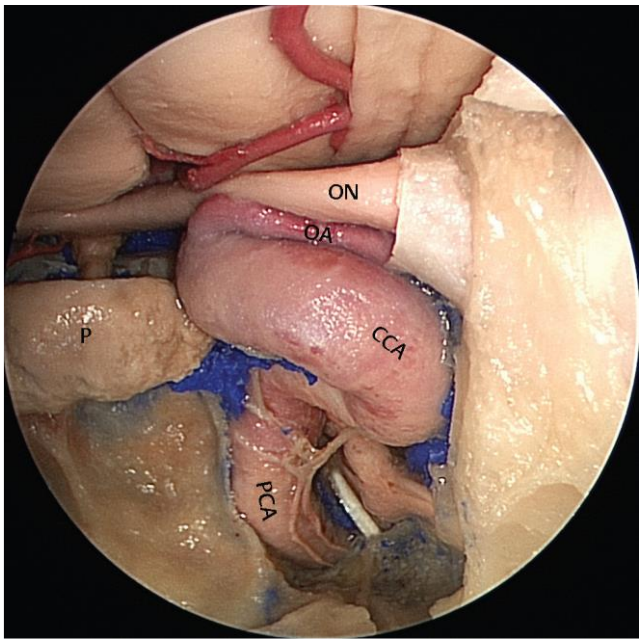


- If the posterior ethmoids and lamina papyracea have been significantly disrupted, a large middle meatal antrostomy gives an additional reference point and reduces the risk of surgeon disorientation.
- The sphenoid sinus natural ostium should be located, and the sphenoid anterior face should be widely opened.
- The roof of the sphenoid and the posterior ethmoids must be continuous, thus the anterior aspect of the sphenoid must be taken as high as possible.
- The optic nerve, carotid artery, and pituitary fossa should all be inspected in the sphenoid.

- Identification of these basic structures can be difficult if the orbital apex or lateral wall of the sphenoid have been significantly disrupted. Image guidance may be useful in these situations.
- The optic tubercle is a thick bone that covers the intersection of the orbital apex and the sphenoid sinus. This bone is generally too thick to flake off, so it is thinned with an irrigated diamond burr until it is practically transparent.
- A blunt Freer elevator is placed 1.5 cm anterior to the junction of the posterior ethmoids air cell(s) and the sphenoid through the lamina papyracea. It is important to preserve the orbital periosteum intact during this procedure, as protrusion of orbital fat might severely block the optic nerve dissection. The underlying orbital periosteum is flaked away from the bone at the posterior orbital apex.
- The bone of the optic canal is reached after the bone over the orbital apex has been removed. This bone is usually fairly thin, and in many cases, it can simply flake away from the underlying nerve. However, the bone over the nerve may be too thick in some situations and will need to be thinned with a diamond burr before being removed.
- Only specially specialised devices should be used once the bone is thin enough to flake away from the underlying nerve. Any instrument with a thick operating end should be avoided. It should not be used if the rear of

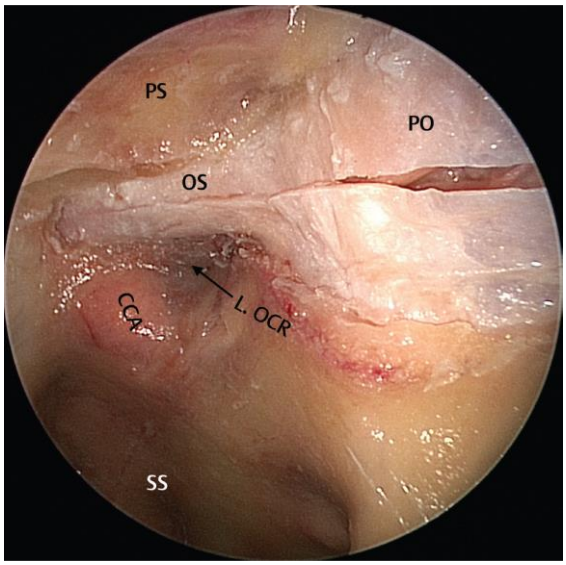
the instrument indents the nerve as the edge of the instrument engages the edge of the optic canal bone.

- The sheath should be incised once all of the bone has been removed from the optic canal and the underlying optic nerve sheath is plainly visible.
- Its important to remember the ophthalmic artery location. The ophthalmic artery is frequently found in the postero inferior region of the nerve. This artery, however, can migrate along the lower margin of the nerve and potentially into the surgical field in a tiny percentage of patients. The risk to this artery is minor if the nerve is incised in the upper medial quadrant.

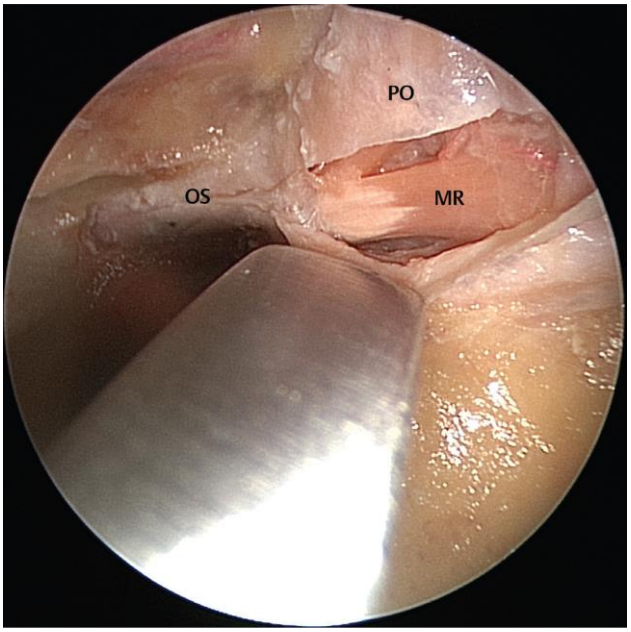


- The optic nerve sheath is incised with a sharp sickle knife.
- As the sheath is incised, the pressure from the enlarged optic nerve usually causes it to split.

- This incision is carried on to the orbital periosteum of the posterior orbital apex, causing orbital fat to protrude.



- Because the orbital fat covering this part of the medial rectus muscle is thin, it's important to avoid damaging it.
- Although such surgical incision has the potential to cause a CSF leak, none has been reported thus far.
- This could be because the nerve has swollen to the point where any potential CSF space has been obliterated.
- There are no packs applied on the nerve or in the sinuses.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SURGICAL TREATMENT- REPORTED PROGNOSIS

REFERENCE	TOTAL NO. OF PATIENTS IN STUDY	NO OF PATIENTS SURGICALLY TREATED	IMPROVED %
Fujitani et al (22)	113	70	47.7%
Wi yan (39)	72	30	Within 7 days 61.5% Later than 7 days 35%
Gu et al (35)	41	35	37.1%
Li et al (6)	45	45	71%
Chou et al (34)	58	25	60%

MATERIALS AND METHODS

A prospective study on the outcome of endoscopic optic nerve decompression in traumatic optic neuropathy patients admitted in Tirunelveli Medical College Hospital was conducted in department of ENT, Tirunelveli Medical College Hospital.

This study was conducted from period of January 2020 to December 2021.All eligible traumatic optic neuropathy neuropathy patients as per inclusion criteria were included in the study.

INCLUSION CRITERIA

Both male and female patients of all age group, diagnosed as traumatic optic neuropathy, who have given consent for endoscopic optic nerve decompression were included in the study

EXCLUSION CRITERIA

Unconscious patients, patients who haven't given consent for the surgery were excluded from the study

METHODOLOGY

After getting approval from institutional ethical committee, patients satisfying the above inclusion criteria were included in the study after getting informed consent

Preoperatively, all basic investigations including CBC, RBS,RFT, serum electrolytes, chest x ray, ECG were done. Detailed ocular examinations including visual acuity, colour vision, pupillary reaction, visual field examinations, fundus examinations were done.

All patients were started on Inj. Methyl prednisolone IV at dose of 1g/day for 3 days followed by T. Prednisolone 1 mg/kg for a period of 11 days and tapered there after .

After getting informed written consent from patients, and after stabilizing patients general condition, the patients were taken for endoscopic traumatic optic nerve decompression under general anaesthesia

SURGICAL PROCEDURE

1. Under general anesthesia patient in supine position, using 0^0 endoscope, uncinectomy and middle meatal antrostomy
2. Removal of Anterior & Posterior ethmoidal air cells. Removal of Lamina papyracea and prolapsed orbital fat. opening of Sphenoid sinus and Aspiration of blood clots.Exploration of optic canal & removal of fractured bony segments
3. Exposure of Optic nerve and Incision of nerve sheath to decompress the nerve.

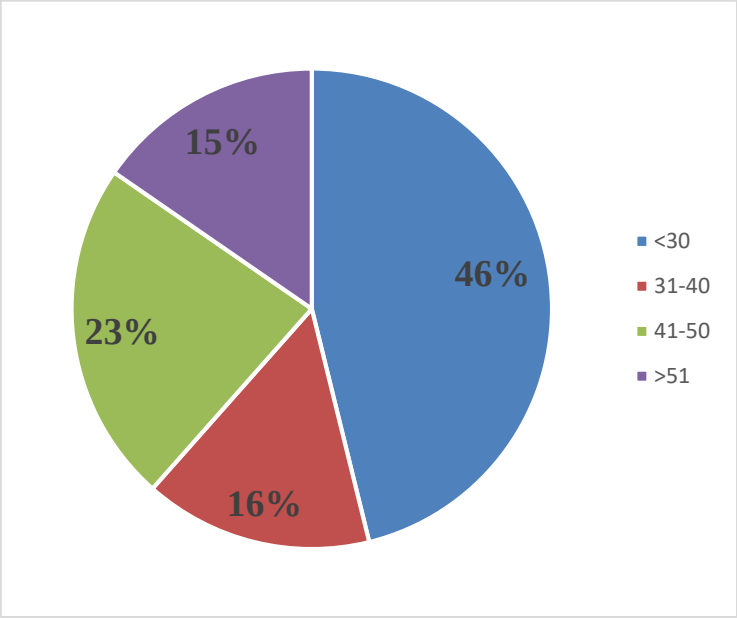
Postoperatively ocular examinations including visual acuity, colour vision, field of vision, papillary reactions were done to analyse the prognosis and patients were followed up monthly for three months.

RESULTS

AGE DISTRIBUTION

The most common age group presented with traumatic optic neuropathy is less than 30 years.

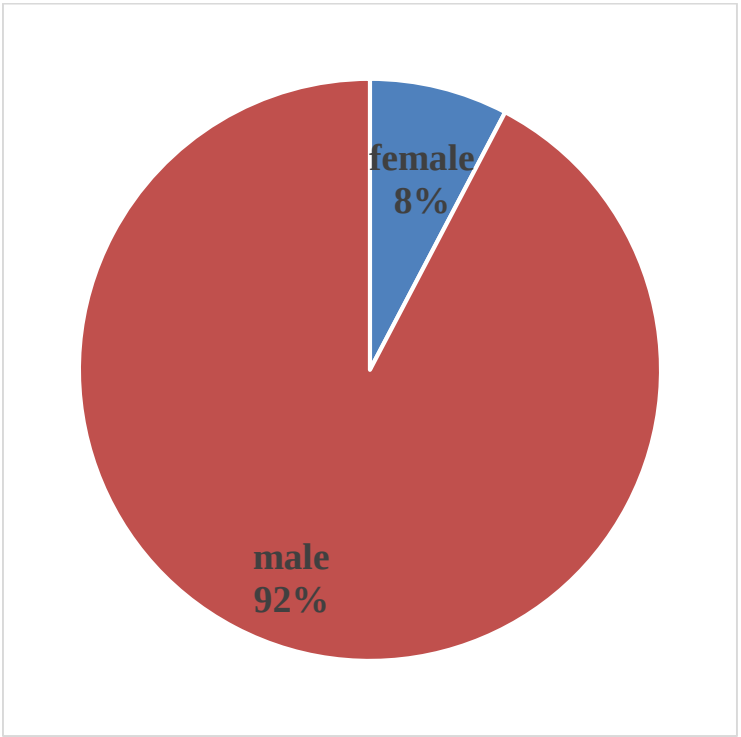
AGE	NO OF PATIENTS	PERCENT
<30	12	46.2
31-40	4	15.4
41-50	6	23.1
>51	4	15.4
Total	26	100.0



SEX DISTRIBUTION

Majority of TON patients in this study are males (92%)

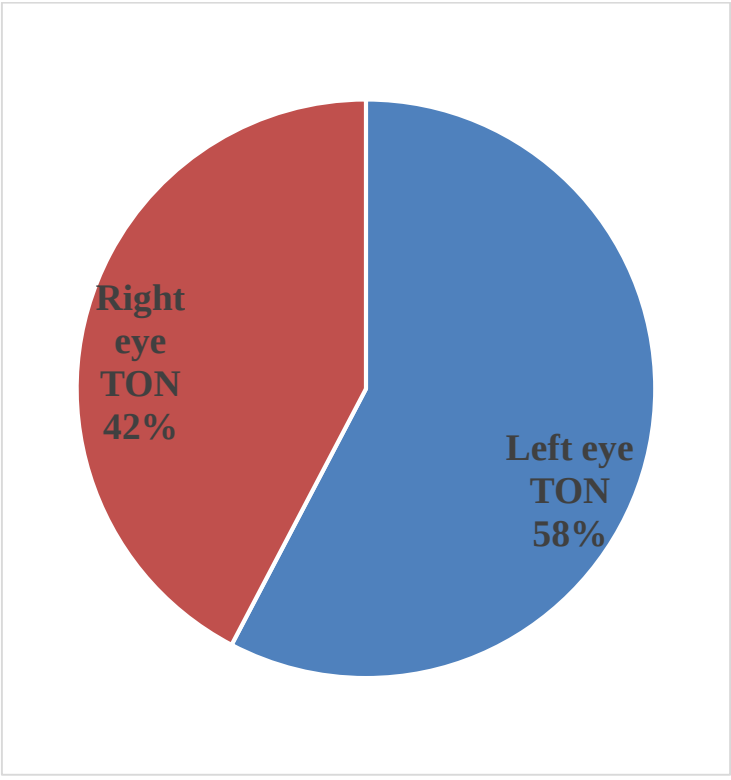
SEX	NO OF PATIENTS	PERCENT
female	2	7.7
male	24	92.3
Total	26	100.0



LATERALITY

In this study, incidence of TON is more in left eye (57%) than the right eye (42%)

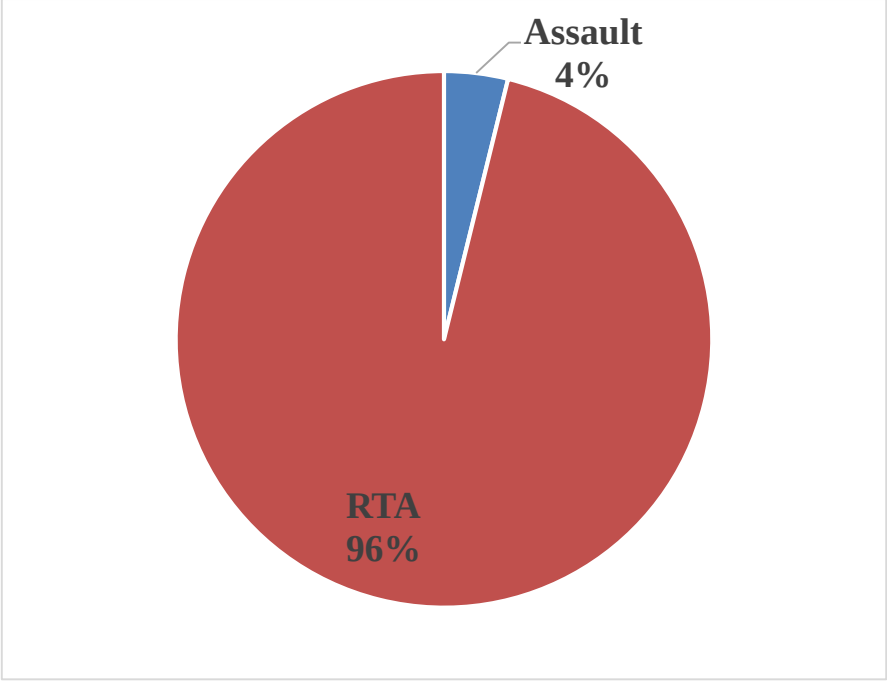
LATERALITY	NO OF PATIENTS	Percent
Left eye TON	15	**57.7**
Right eye TON	11	**42.3**
Total	26	100.0



MODE OF INJURY

RTA (96.2%) comprises the major cause of injury in this study

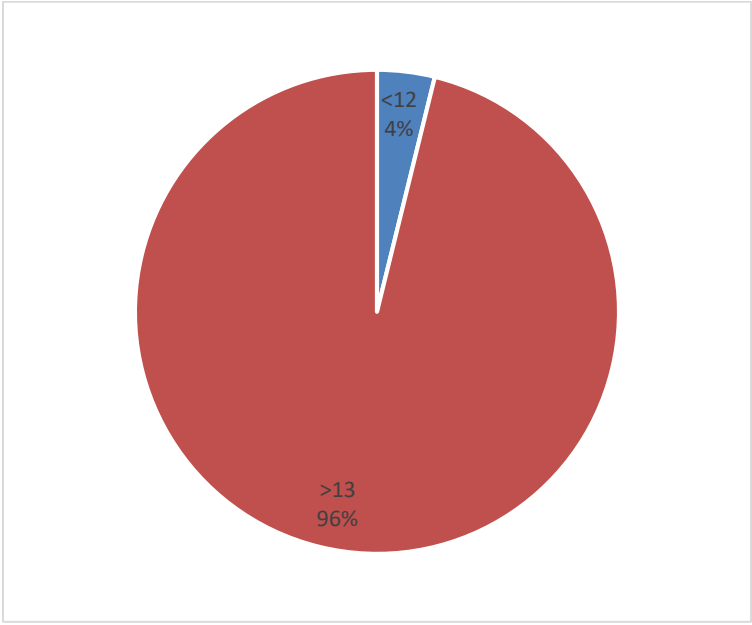
MODE OF INJURY	NO OF PATIENTS	Percent
Assault	1	3.8
RTA	25	96.2
Total	26	100.0



GCS AT THE TIME OF PRESENTATION

In this study, 96% of TON patients had >13 GCS at the time of presentation

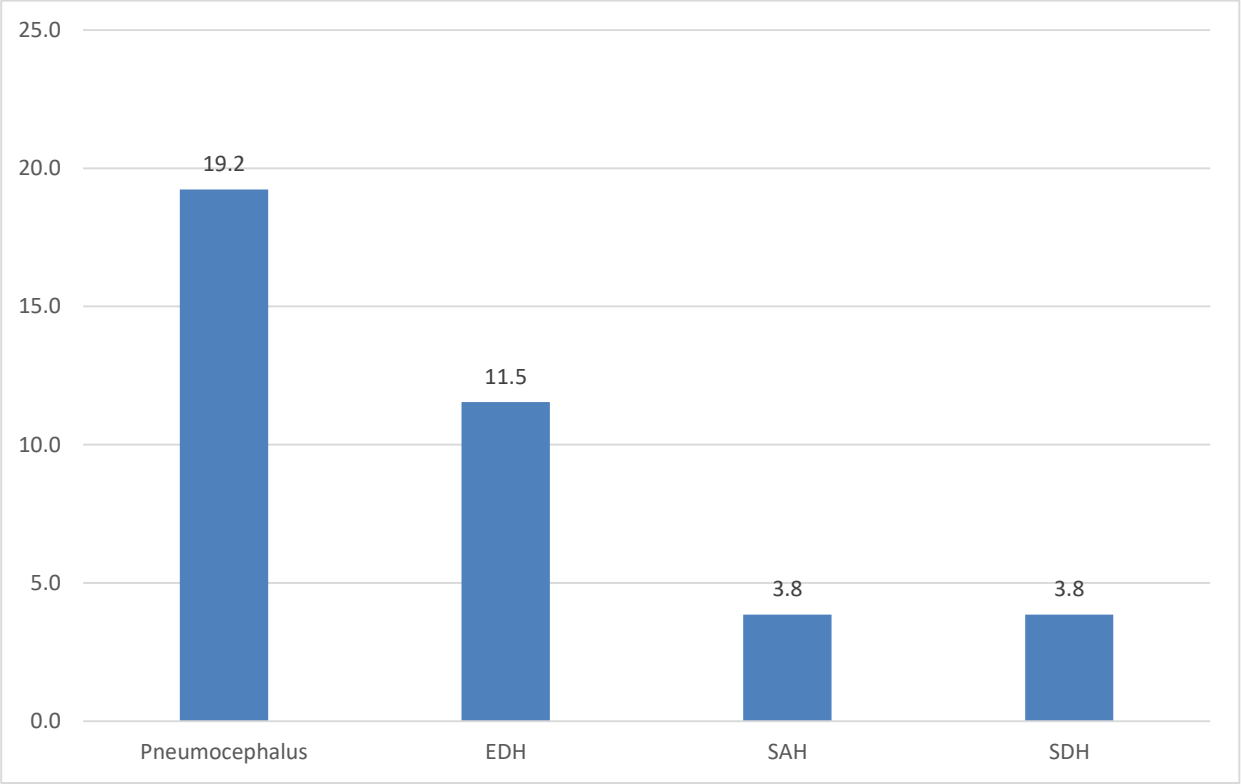
GCS AT THE TIME OF PRESENTATION	NO OF PATIENTS	PERCENT
<12	1	3.8
>13	25	96.2
Total	26	100.0



OTHER INTRACRANIAL RADIOLOGIAL FINDINGS

38% of TON patients had various co existing intra -cranial abnormal findings. Of these, pneumocephalus is the commonest (19%)

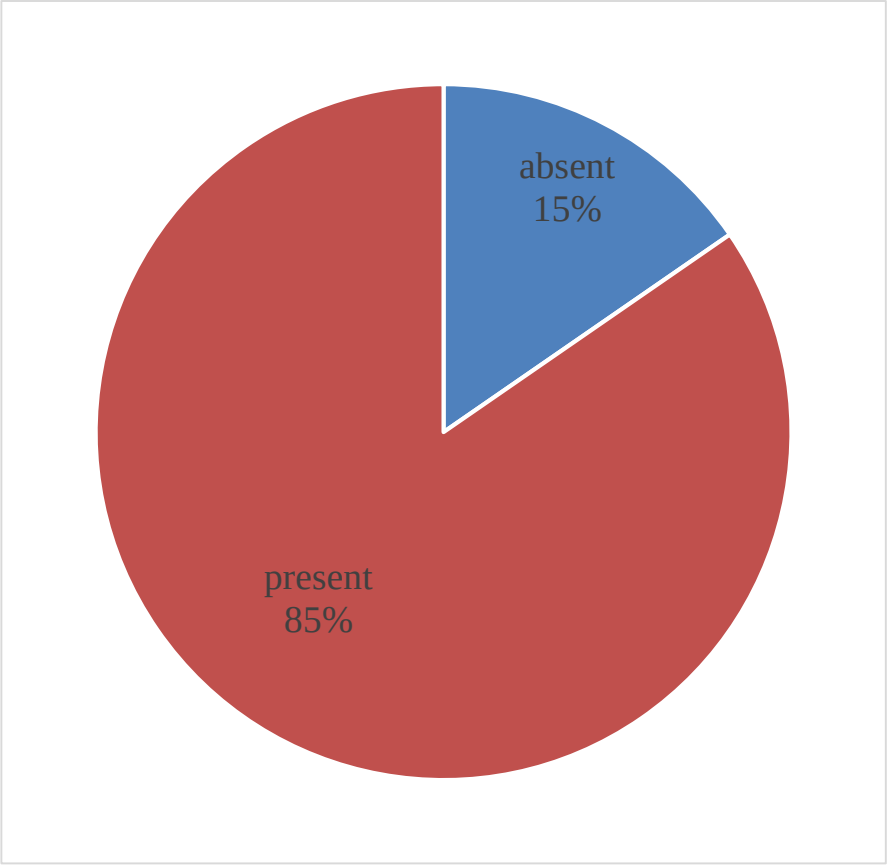
OTHER INTRACRANIAL FINDINGS	NO OF PATIENTS	PERCENT
Pneumocephalus	5	19.2
EDH	3	11.5
SAH	1	3.8
SDH	1	3.8
Total	10	38.5



FRACTURE SITES (RADIOLOGICAL)

Medial orbital wall fracture occurred in 85% of patients in this study

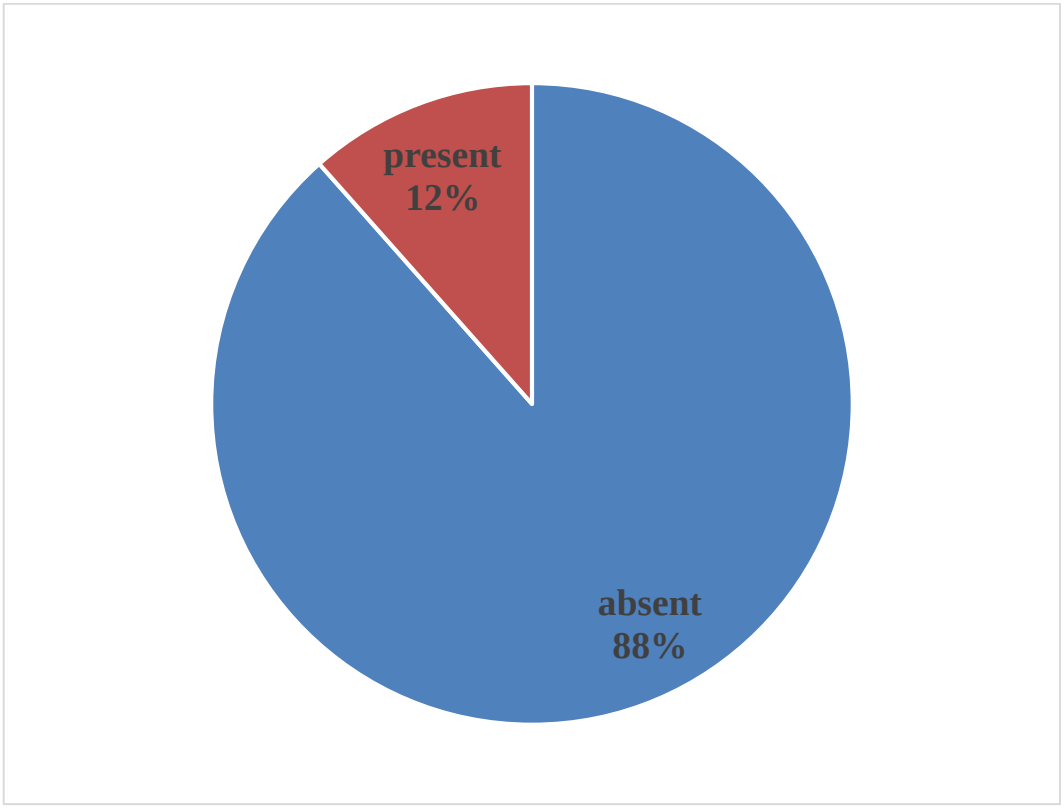
FRACTURE MEDIAL WALL OF ORBIT	NO OF PATIENTS	PERCENT
present	22	**84.6**
absent	4	15.4
Total	26	100.0



FRACTURE SITES (RADIOLOGICAL)

11.5% of patients had fracture of roof of orbit

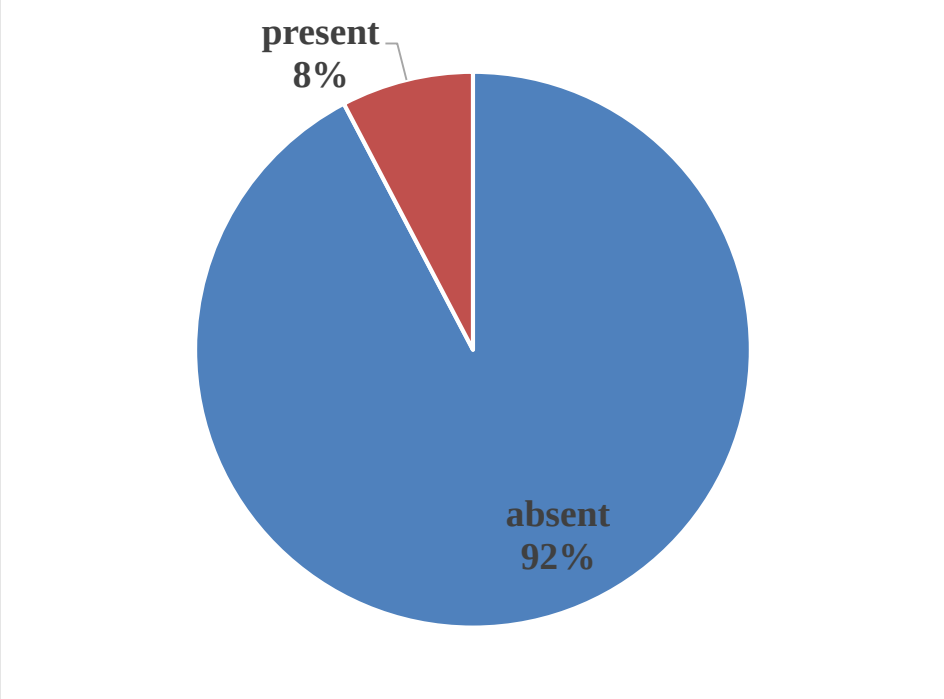
FRACTURE ROOF OF ORBIT	NO OF PATIENTS	Percent
present	3	**11.5**
absent	23	88.5
Total	26	100.0



FRACTURE SITES (RADIOLOGICAL)

7.7% of patients had fracture in floor of orbit

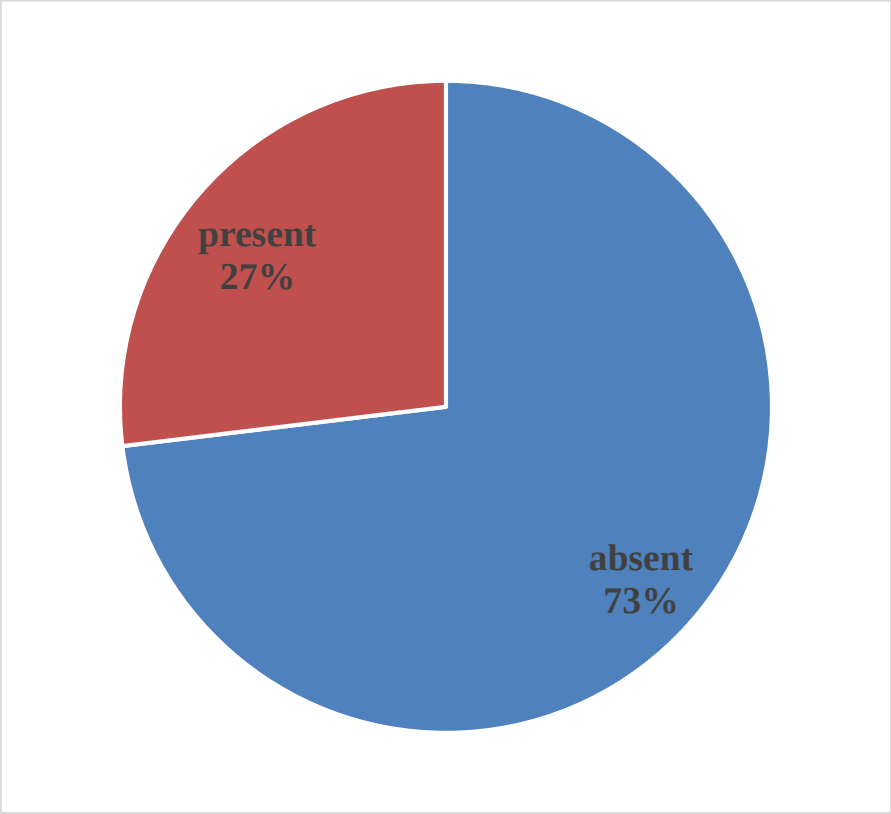
FRACTURE FLOOR OF ORBIT	NO OF PATIENTS	Percent
present	2	**7.7**
absent	24	92.3
Total	26	100.0



FRACTURE SITES (RADIOLOGICAL)

27% of patients had fracture lateral wall of orbit

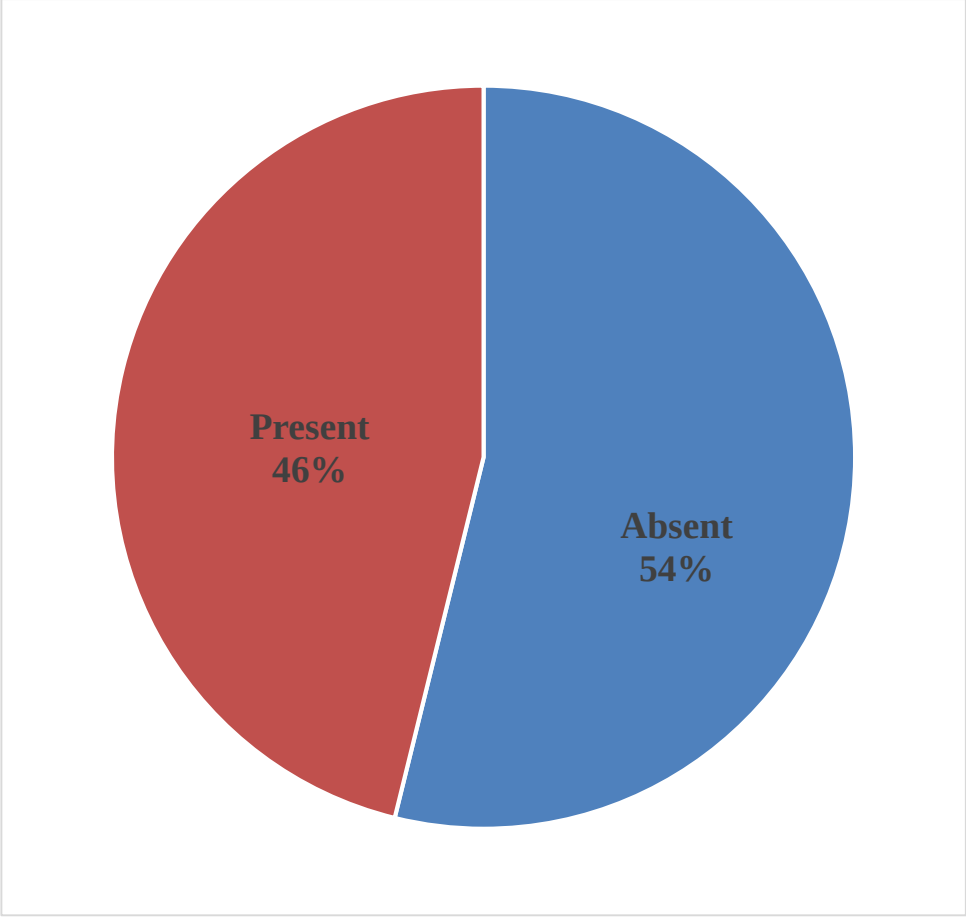
FRACTURE LATERAL WALL OF ORBIT	NO OF PATIENTS	Percent
present	7	**26.9**
absent	19	73.1
Total	26	100.0



FRACTURE SITES (RADIOLOGICAL)

46% of patients had fracture sphenoid bone

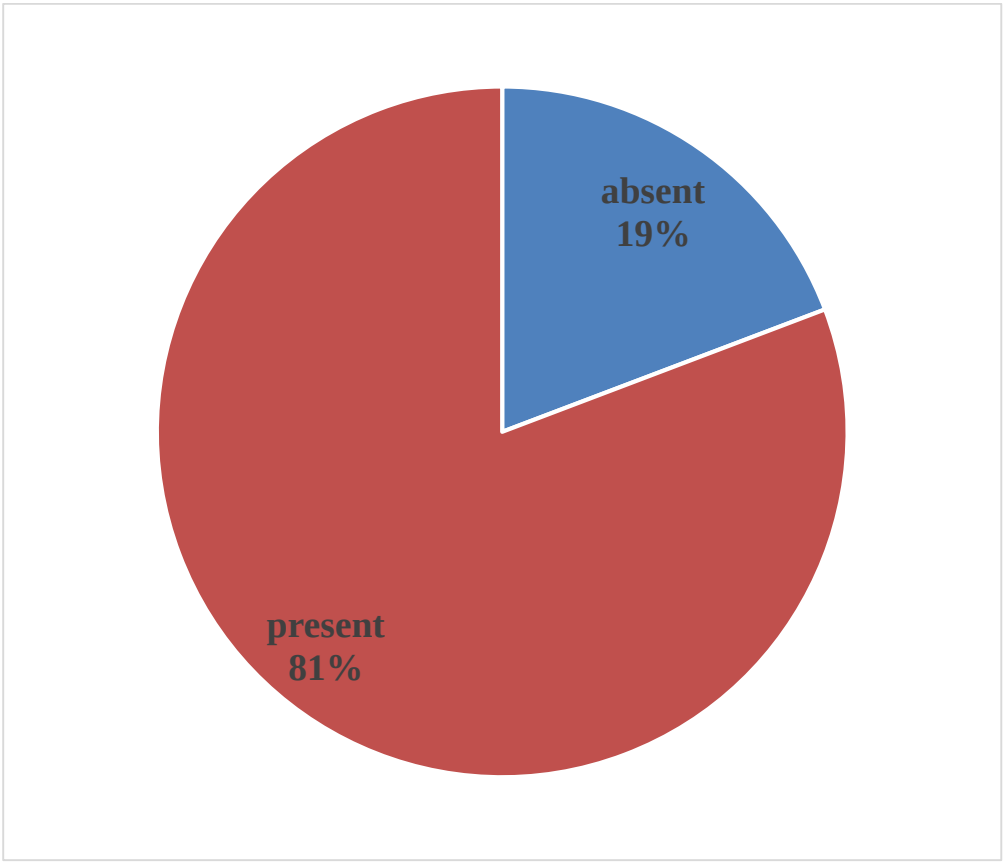
FRACTURE SPHENOID	NO OF PATIENTS	Percent
present	12	**46.2**
absent	14	53.8
Total	26	100.0



INTRAOPERATIVE FINDINGS

81% of patients had fracture lamina papyracea intraoperatively

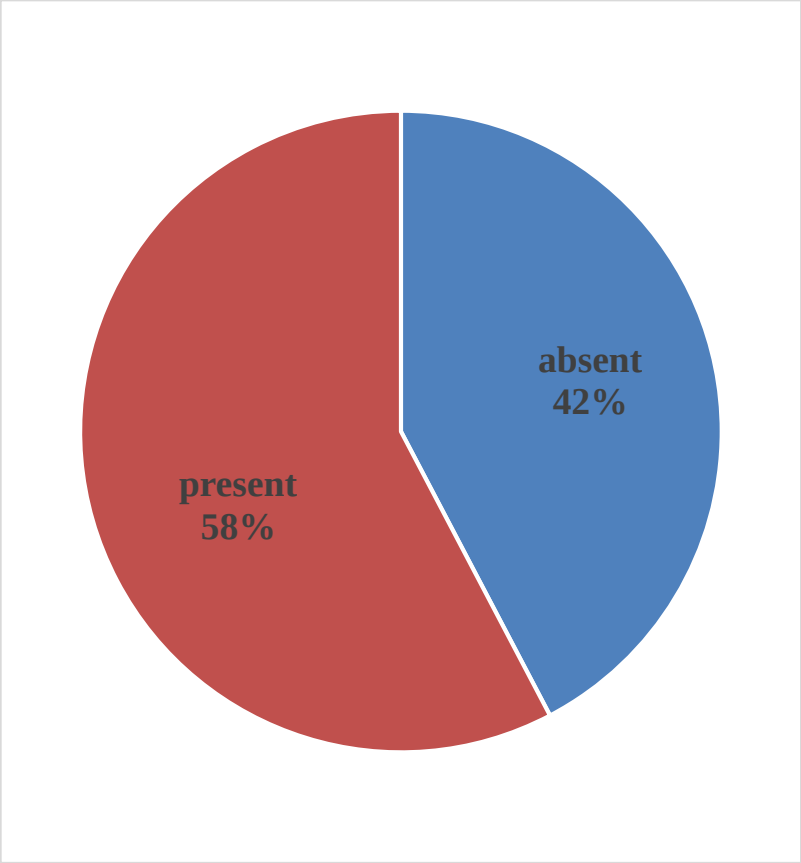
FRACTURE LAMINA PAPYRACEA	NO OF PATIENTS	Percent
present	21	**80.8**
absent	5	19.2
Total	26	100.0



INTRAOPERATIVE FINDINGS

58% of patients had fracture segments over optic canal intraoperatively

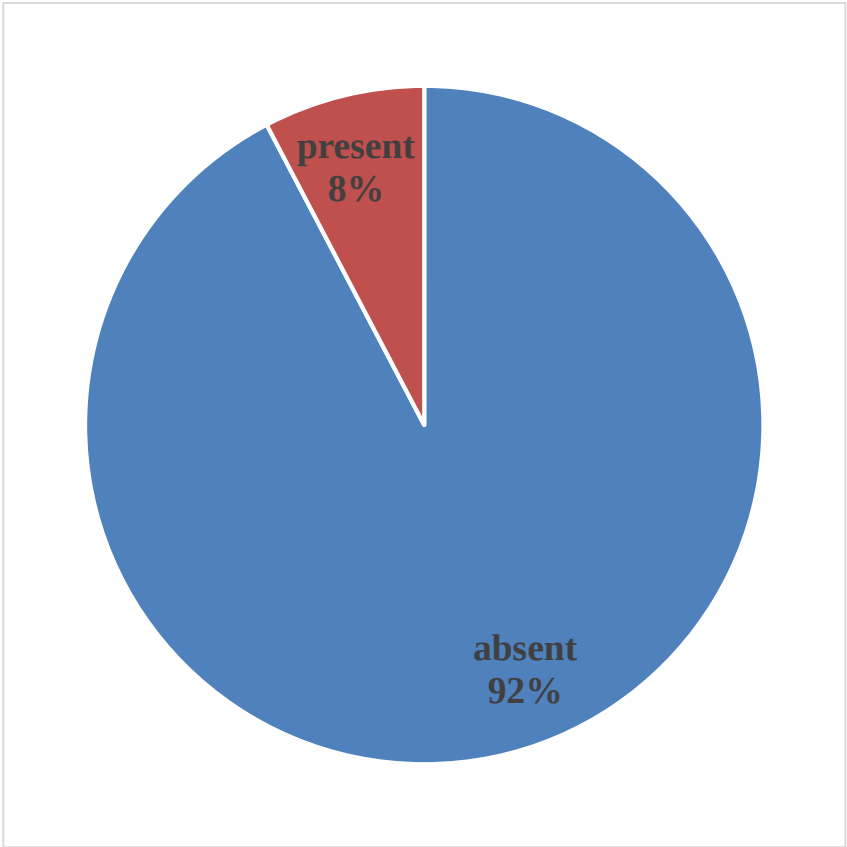
FRACTURE SEGMENTS OVER OPTIC CANAL	NO OF PATIENTS	Percent
present	15	**57.7**
absent	11	42.3
Total	26	100.0



INTRAOPERATIVE FINDINGS

7.7% of patients had cribriform plate fracture intraoperatively

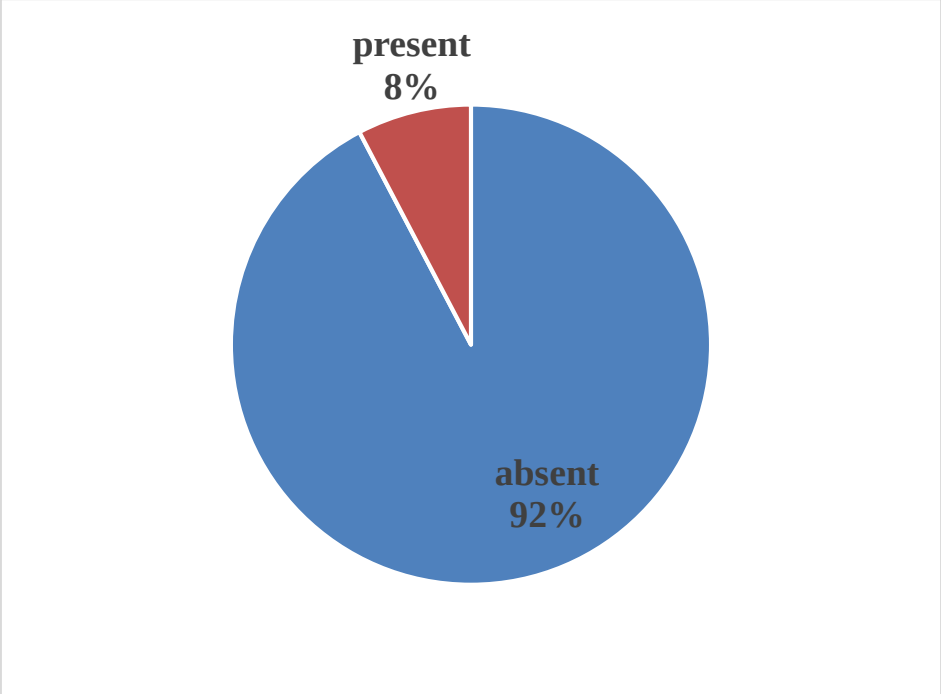
CRIBRIFORM PLATE FRACTURE	NO OF PATIENTS	Percent
present	2	**7.7**
absent	24	92.3
Total	26	100.0



INTRAOPERATIVE FINDINGS

7.7% of patients had cribriform plate fracture with CSF leak intraoperatively

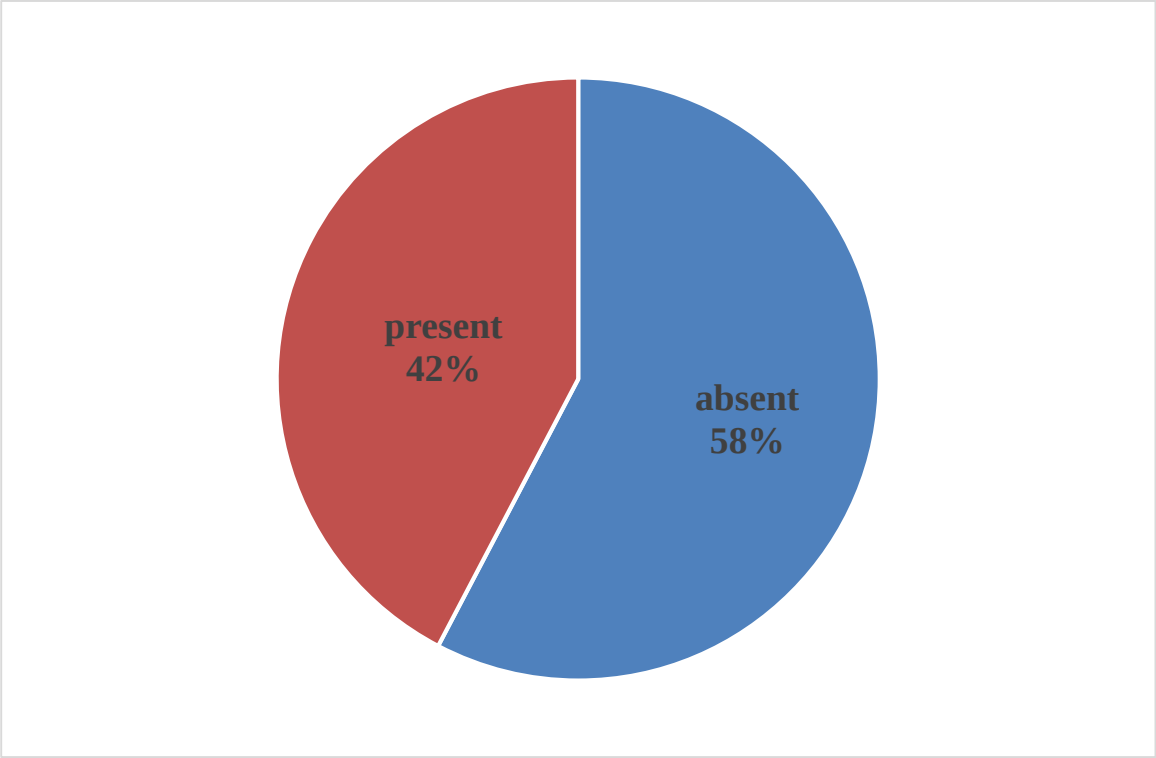
CSF LEAK	NO OF PATIENTS	Percent
present	2	**7.7**
absent	24	92.3
Total	26	100.0



INTRAOPERATIVE FINDINGS

42.3% of patients had ethmoid hemosinus

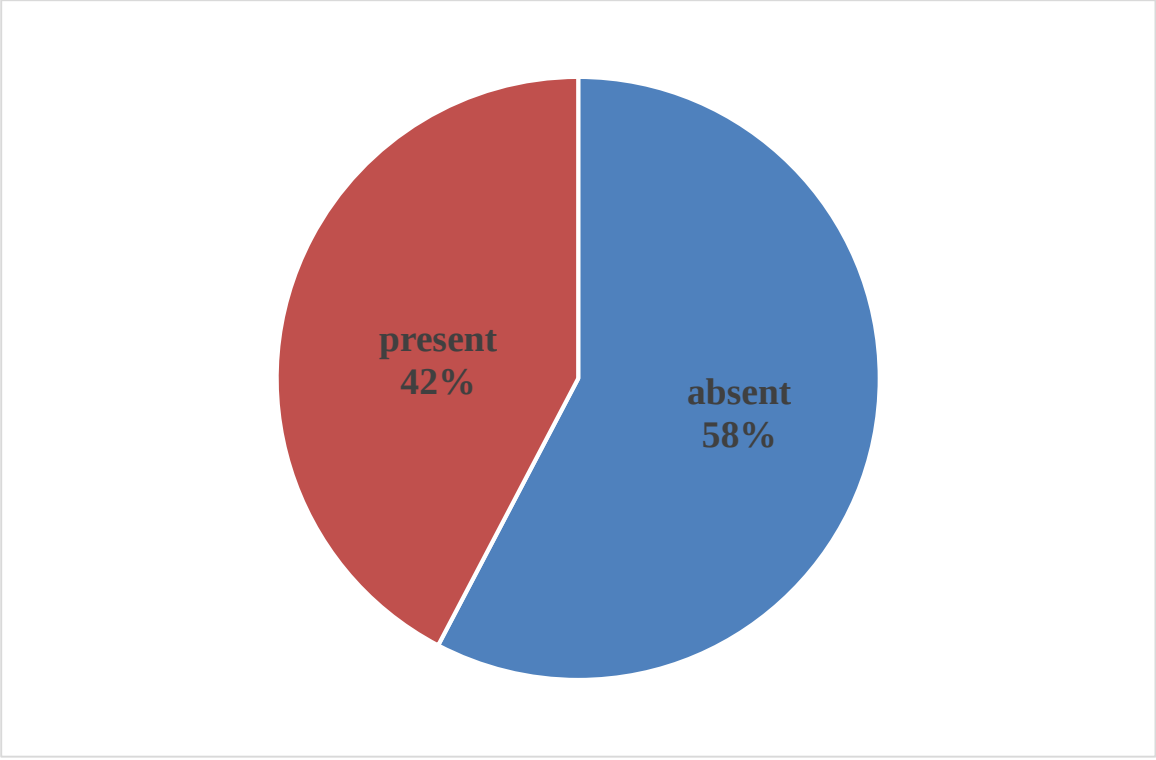
ETHMOID HEMOSINUS	Frequency	Percent
present	11	**42.3**
absent	15	57.7
Total	26	100.0



INTRAOPERATIVE FINDINGS

42.3 % of patients had sphenoid hemosinus

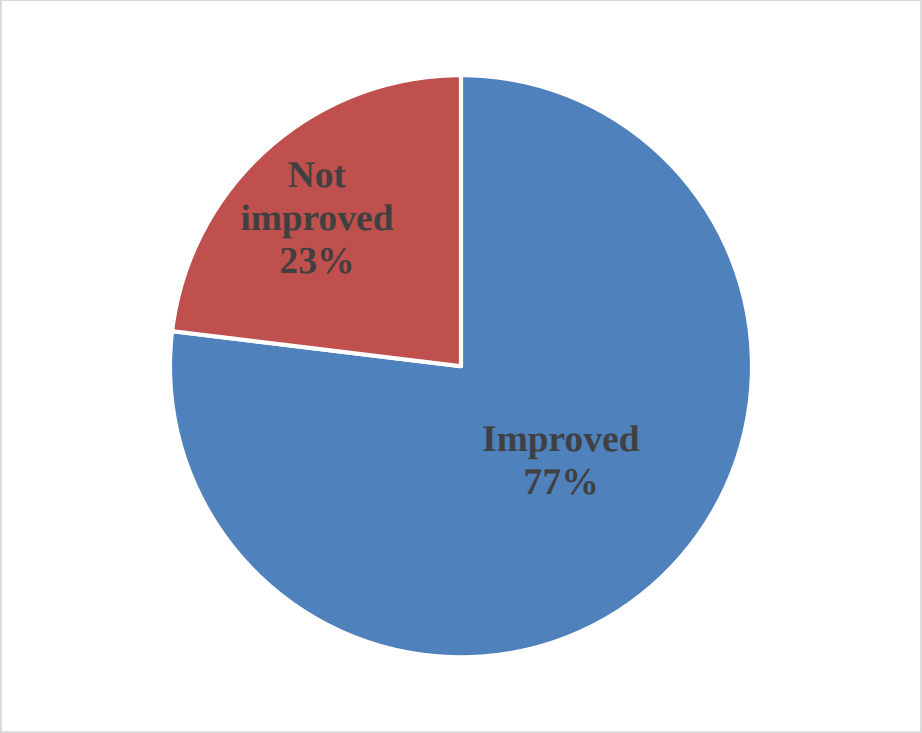
SPHENOID HEMOSINUS	Frequency	Percent
Yes	11	**42.3**
No	15	57.7
Total	26	100.0



POSTOPERATIVE VISUAL ACUITY IMPROVEMENT

In this study, postoperatively 77% of patients had improvement in visual acuity

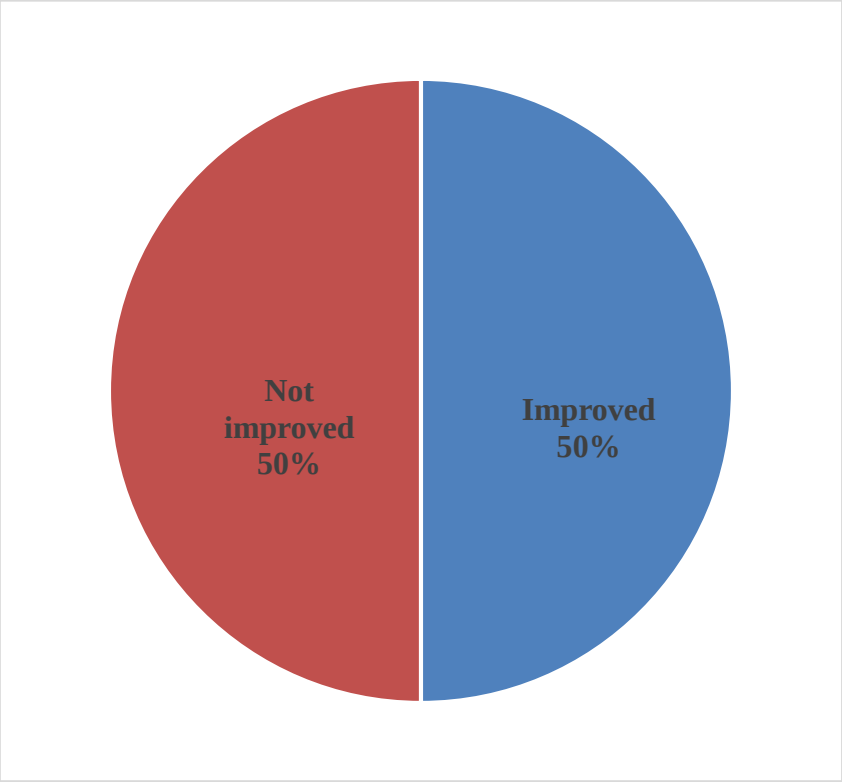
Postoperative Visual acuity	No.of patients	Percent
Improved	**20**	**76.9**
Not improved	6	23.1
Total	26	100.0



POST OPERATIVE COLOUR VISION IMPROVEMENT

In this study, 50% patients had colour vision improvement post operatively

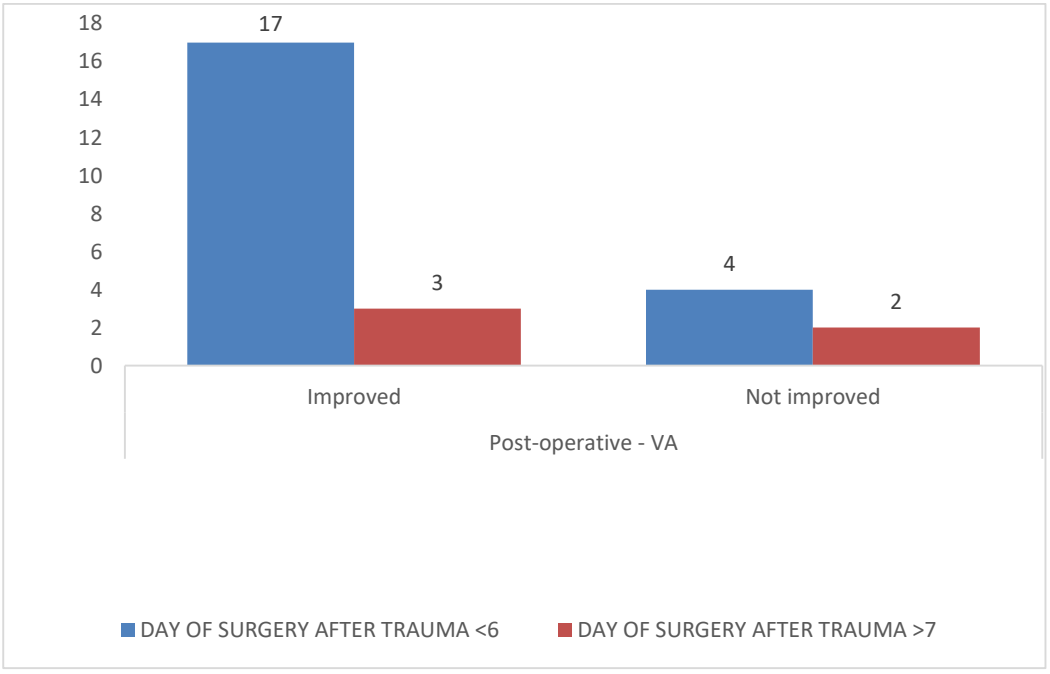
Postoperative CV	No of patients	Percent
Improved	**13**	**50.0**
Not improved	13	50.0
Total	26	100.0



VISUAL ACUITY IMPROVEMENT IN RELATION TO DAY OF SURGERY AFTER TRAUMA

In patients treated within 6 days, 81% of patients showed improved. In patients treated on or later 7 days, 60% of patients showed improvement

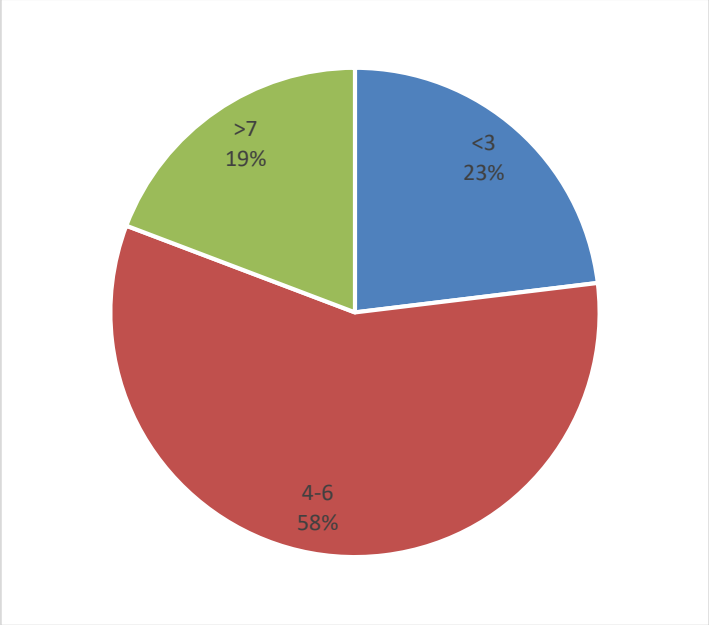
			Post-operative - VA		Total
			improved	not improved	
DAY OF SURGERY AFTER TRAUMA	≤6	NO OF PATIENTS	17	4	21
		PERCENTAGE	**81.0%**	19.0%	100.0%
	≥7	NO OF PATIENTS	3	2	5
		PERCENTAGE	**60.0%**	40.0%	100.0%
Total		NO OF PATIENTS	20	6	26
		PERCENTAGE	76.9%	23.1%	100.0%



DAY OF SURGERY AFTER TRAUMA

In our study, only 23% of patients underwent surgery within 3 days. 58% of patients underwent surgery in 4-6 days. 19% of patients underwent surgery on or after 7 days.

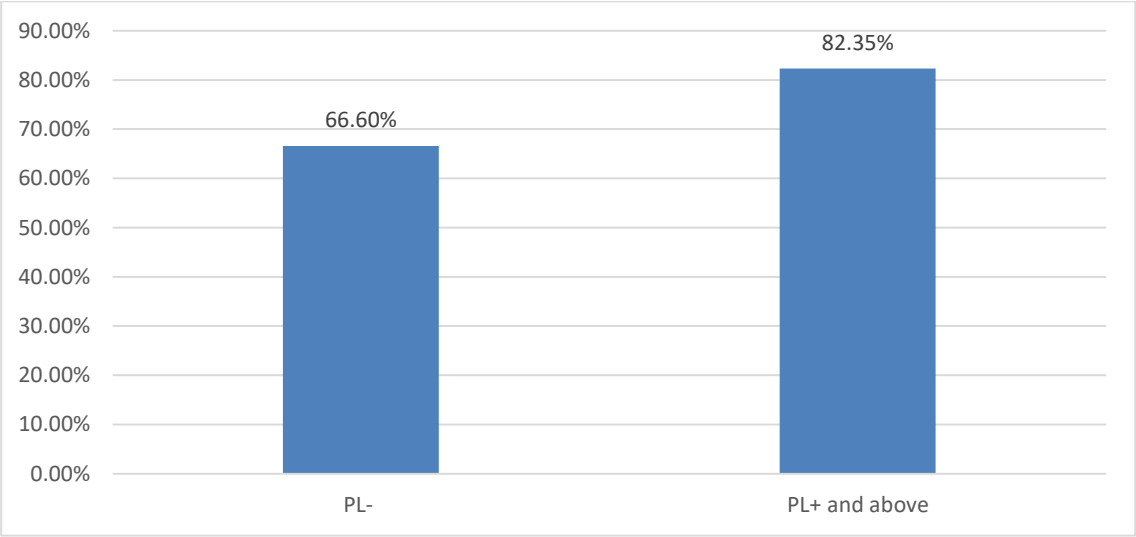
Day Of Surgery After Trauma	No. Of Patients	Percentage
<3	6	23.1%
4-6	15	57.7%
>7	5	19.2%
Total	26	100.0%



PROGNOSIS WITH RESPECT TO SEVERITY OF VISUAL IMPAIRMENT AT PRESENTATION

In patients with initial VA of no PL, 67% of patients improved. In patients with initial VA of PL+ and above, 82% of patients showed improvement

VA At Presentation	Total No Of Patients	No Of Patients Improved	Percentage Of Improved Patients
PL-	9	6	**66.60%**
PL+ and above	17	14	**82.35%**



DISCUSSION

In this study, there were totally 26 TON patients, of these, most common age group was below 30 years of age (46%). Majority of patients in this study were males (92%). Majority of patients presented with left eye TON (58%). RTA (96%) was the most common mode of injury.

At the time of presentation, 96% of patients were having GCS above 13.

38.5% patients had various co- existing intracranial abnormalities, of which pneumocepalus was the commonest (19%), followed by EDH(11%)

In this study, radiologically, fracture medial wall of orbit was seen in 85% of cases, , fracture sphenoid bone was seen in 46% of cases, fracture lateral wall of orbit was seen in 27% of cases, fracture roof of orbit was seen in 11.5% of cases, fracture floor of orbit was seen in 7.7% of cases.

Intraoperatively, fracture lamina papyracea was seen in 81% of cases, fracture segments over optic canal was seen in 58% of cases, ethmoid and sphenoid hemosinus were seen in 42% of cases

Cribriform plate fracture with CSF leak was found in 2 patients (7.7%) which was corresponds with rajinikanth et al (7%) (36)

In this study, of the total 26 patients, 20 patients (77%) had improvement in visual acuity post-operatively. Yan et al (2017) (38) reported improvement in

81.2% of patients who have underwent endonasal optic nerve decompression after steroid therapy .

In our study, 81% of patients who have underwent endonasal decompression within 6 days showed improvement in visual acuity whereas 60% of those operated on or after 7 days showed improvement in visual acuity. Wi-yan et al reported visual acuity improvement in 61.5% of TON patients treated with endonasal optic nerve decompression within 7 days and in 35% of cases later than 7 days.

In our study, only 23% of patients underwent surgery within 3 days. 58% of patients underwent surgery in 4-6 days. 19% of patients underwent surgery on or after 7 days. There could have been better improvement if these patients were taken for surgery earlier.

In our study, 82% of patients with initial vision of PL+ and above showed improvement whereas 67% of those with no PL also showed improvement in vision. Wi-yan et al reported visual acuity improvement in 83% of patients with initial VA of PL+ and better, and 33% of patients with initially no light perception.

CONCLUSION

- Endoscopic endonasal optic nerve decompression have a significant effect in visual acuity improvement in traumatic optic neuropathy
- Results are better if endonasal optic nerve decompression is carried out as early as possible.
- Minimal Improvement is reported in patients even if the surgery is carried out after 7 days. Hence no patients should be denied of this surgery even in such delayed circumstances
- Results are better in patients having some residual vision pre-operatively than in totally blind patients

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PROFORMA

Name:

Ip No:

Age:

Gender: 1. Male 2. Female

Laterality: 1. RE 2. LE

Mode of injury:1. RTA 2.ASSAULT 3.OTHERS

Time of presentation:

Presenting VA:

Pupillary reaction:

Fundus status:

Colour Vision

Field defects

CT facial bone findings

Other associated intracranial abnormal findings if any

Day of surgery after trauma

Intraoperative findings

Post op day 1

- visual acuity
- colour vision
- pupillary reaction

- fundus
- field defects

Post op day 8

- visual acuity
- colour vision
- pupillary reaction
- field defects
- fundus

Follow up after 3 months

- Visual acuity
- colour vision
- pupillary reaction
- field defects
- fundus

ஒப்புதல் படிவம்

எனது முகத்தில் ஏற்பட்டுள்ள எலும்புமுறிவுகளால், கண் நரம்பில் அழுத்தம் ஏற்பட்டு கண் பார்வை பாதிப்பு ஏற்பட்டுள்ளதால், மூக்கின் வழியாக எண்டோஸ்கோப்பி மூலம் கண் நரம்பின் அழுத்தத்தை சீர் செய்வதன் மூலம் கண் பார்வையில் முன்னேற்றம் ஏற்படலாம் என மருத்துவர் மூலம் அறிந்து கொண்டேன். மேலும் இதன் முலம் கண் பார்வையில் முன்னேற்றம் ஏற்படாமல் இருக்கவும் வாய்ப்புண்டு என்பதையும் அறிந்து கொண்டு அறுவை சிகிச்சை செய்துகொள்ள சம்மதிக்கிறேன். மேலும் இந்த அறுவை சிகிச்சையின் பலன்கள் தொடர்பாக திருநெல்வேலி மருத்துவக்கல்லூரி காது, மூக்கு, தொண்டை பிரிவில் நடத்தப்படும் ஆராய்ச்சியில் பங்குகொள்ள சம்மதிக்கிறேன்.

														PRE-OPERATIVE								POST-OPERATIVE									FOLLOW UP AFTER 3 MONTHS			
														visual acuity		colour vision		direct reflex		indirect reflex		visual acuity			colour vision			direct reflex		indirect reflex		visual acuity		colour vision	
s.no	name	age	age	sex	IP.no	diagnosis	mode of injury	day of presentation at our institution after injury after injury	GCS at the time of presentation	fracture site (RADIOLOGICAL)	other Intracranial findings (RADIOLOGICAL)	INTRAOP FINDINGS	day of surgery after trauma	right eye	left eye	right eye	left eye	right eye	left eye	right eye	left eye	right eye	left eye	Improvement	right eye	left eye	improvement	right eye	left eye	right eye	left eye	right eye	left eye	right eye	left eye
1	belson	23	1	male	66253	Left eye TON	RTA	0	14	fracture medial wall, lateral wall, roof and floor of left orbit	-	fracture lamina papyracea, fracture segments over optic canal	7	6 / 6	3 / 60	Normal	Defective	RTL	RAPD	NRTL	RTL	6 / 6	6 / 9	improved	Normal	Normal	improved	RTL	RAPD	NRTL	RTL	6 / 6	6 / 9	Normal	Normal
2	senthil kumar	45	3	male	19393	Left eye TON	RTA	0	13	fracture medial wall, lateral wall,left orbit. Fracture body of sphenoid involving roof and extending to left lateral wall of sphenoid	EDH/Pneumocephalus	prolapsed orbital pad of fat, sphenoid hemosinus, fracture lateral wall of sphenoid	4	6 / 6	2 / 60	normal	defective	RTL	RAPD	NRTL	RTL	6 / 6	6 / 18	improved	Normal	Normal	improved	RTL	RAPD	NRTL	RTL	6 / 6	6 / 18	Normal	Normal
3	saravanan	38	2	male	31115	Right eye TON	RTA	0	13	fracture medial and lateral wall of right orbit,fracture right frontal bone involving both tables	pneumocephalus	fracture lamina papyracea, fracture segments over optic canal	4	1/60	6 / 6	Defective	Normal	RAPD	RTL	RTL	NRTL	6 / 36	6 / 6	improved	Normal	Normal	improved	RAPD	RTL	RTL	NRTL	6 / 24	6 / 6	Normal	Normal
4	ramesh	23	1	male	31153	Left eye TON	RTA	0	15	fracture lateral wall of left orbit, sphenoid and ethmoid hemosinus	-	b/l sphenoid hemosinus, left ethmoid hemosinus	1	6 / 18	5 / 60	Normal	Defective	RTL	RAPD	NRTL	RTL	6 / 18	6 / 24	improved	Normal	Normal	improved	RTL	RAPD	NRTL	RTL	6 / 18	6 / 24	Normal	Normal
5	sankara narayanan	44	3	male	72150	Right eye TON	RTA	0	15	fracture right lamina papyracea, fracture body of sphenoid		fracture lamina papyracea, fracture segments over optic canal, sphenoid hemosinus	5	HM+	6 / 9	Defective	Normal	RAPD	RTL	RTL	NRTL	6 / 12	6 / 9	improved	Normal	Normal	improved	RAPD	RTL	RTL	NRTL	6 / 12	6 / 9	Normal	Normal
6	arichandran	47	3	male	62700	Left eye TON	RTA	0	15	fracture left lamina papyracea, fracture body of sphenoid		fracture lamina papyracea, fracture segments over optic canal, sphenoid hemosinus	4	6 / 9	HM+	Normal	Defective	RTL	RAPD	NRTL	RTL	6 / 9	6/ 12	improved	Normal	Normal	improved	RTL	RAPD	NRTL	RTL	6 / 9	6/ 12	Normal	Normal
7	kaliraj	41	3	male	15258	Right eye TON	RTA	0	15	fracture right lamina papyracea		right ethmoid hemosinus, fracture right lamina papyracea	4	HM+	6 / 9	Defective	Normal	RAPD	RTL	RTL	NRTL	6 / 18	6/ 9	improved	Normal	Normal	improved	RAPD	RTL	RTL	NRTL	6 / 18	6/ 9	Normal	Normal
8	venkatesh	20	1	male	8392	Left eye TON	RTA	0	13	fracture body of sphenoid, fracture lamina papyracea, fracture cribriform plate	pneumocephalus	left ethmoid hemosinus, fracture roof of ethmoid and CSF leak present, fracture segment over left optic canal	4	6 / 6	PL-	Normal	Defective	RTL	RAPD	NRTL	RTL	6 / 6	6 / 18	improved	Normal	Normal	improved	RTL	RAPD	NRTL	RTL	6 / 6	6 / 18	Normal	Normal
9	paulraj	51	4	male	47821	Left eye TON	RTA	0	15	left lamina payracea fracture		optic canal dehiscent, lamina papyracea fracture	4	6 / 9	1 / 60	Normal	Defective	RTL	RAPD	NRTL	RTL	6 / 9	6 / 24	improved	Normal	Normal	improved	RTL	RAPD	NRTL	RTL	6 / 9	6 / 24	Normal	Normal
10	sankar	27	1	male	76220	Right eye TON	RTA	0	15	fracture right lamina papyracea, fracture body of sphenoid, fracture cribriform plate		right ethmoid hemosinus, right sphenoid hemosinus, fracture lamina papyracea, fracture segment in cribriform plate and CSF leak noted	7	1 / 60	6 / 6	Defective	Normal	RAPD	RTL	RTL	NRTL	6 / 6	6 / 6	improved	Normal	Normal	improved	RAPD	RTL	RTL	NRTL	6 / 6	6 / 6	Normal	Normal
11	azhagar	55	4	male	48187	Right eye TON	RTA	0	13	fracture right lamina papyracea, fracture body of sphenoid	EDH	right ethmoid hemosinus, right sphenoid hemosinus, fracture lamina papyracea	6	HM+	6 / 9	Defective	Normal	RAPD	RTL	RTL	NRTL	6 / 36	6 / 9	improved	Normal	Normal	improved	RAPD	RTL	RTL	NRTL	6 / 36	6 / 9	Normal	Normal
12	suresh	23	1	male	42863	Left eye TON	RTA	0	15	fracture left lamina papyracea, fracture body of sphenoid		left ethmoid hemosinus, left sphenoid hemosinus, fracture lamina papyracea	4	6 / 6	PL-	Normal	Defective	RTL	RAPD	NRTL	RTL	6 / 6	6 / 18	improved	Normal	Normal	improved	RTL	RAPD	NRTL	RTL	6 / 6	6 / 18	Normal	Normal
13	vinoth kumar	32	2	male	74295	Right eye TON	RTA	0	15	fracture right lamina papyracea, fracture body of sphenoid		right ethmoid hemosinus, right sphenoid hemosinus, fracture lamina papyracea	5	0.5/60	6 / 6	Defective	Normal	RAPD	RTL	RTL	NRTL	6 / 12	6 / 6	improved	Normal	Normal	improved	RAPD	RTL	RTL	NRTL	6 / 12	6 / 6	Normal	Normal
14	selvakumar	29	1	male	14156	Left eye TON	RTA	0	15	fracture medial wall and floor of left orbit	-	fracture lamina papyracea, fracture segments over optic canal	1	6/9	PL-	Normal	Defective	RTL	RAPD	NRTL	RTL	6 / 9	PL+	improved	Normal	Defective	not improved	RTL	RAPD	NRTL	RTL	6 / 9	HM+	Normal	Defective
15	sankarapandi	38	2	male	33792	Left eye TON	RTA	0	15	fracture medial wall of left orbit	-	fracture lamina papyracea, fracture segments over optic canal	5	6/6	PL+	Normal	Defective	RTL	RAPD	NRTL	RTL	6 / 6	HM+	improved	Normal	Defective	not improved	RTL	RAPD	NRTL	RTL	6 / 6	HM+	Normal	Defective
16	velmurugan	22	1	male	14154	Right eye TON	RTA	0	13	fracture B/L lamina papyracea and B/L roof of orbit, Right frontal bone communited fracture	Pneumocephalus	fracture lamina papyracea, fracture segments around optic tubercle	9	HM+	6/6	Defective	Normal	RAPD	RTL	RTL	NRTL	HM+	6/6	not improved	Defective	Normal	not improved	RAPD	RTL	RTL	NRTL	HM+	6/6	Defective	Normal
17	sivanammal	50	3	female	22315	Left eye TON	RTA	0	15	fracture medial wall of left orbit, Left optic nerve thickened and irregular	-	fracture lamina papyracea, fracture segments over optic canal	4	6/ 9	PL-	Normal	Defective	RTL	RAPD	NRTL	RTL	6 / 9	HM+	improved	Normal	Defective	not improved	RTL	RAPD	NRTL	RTL	6 / 9	HM+	Normal	Defective
18	starvin	22	1	male	78485	Right eye TON	RTA	0	15	fracture medial wall of right orbit	-	fracture lamina papyracea, fracture segments over optic canal	3	PL-	6 / 6	Defective	Normal	RAPD	RTL	RTL	NRTL	PL+	6 / 6	improved	Defective	Normal	not improved	RAPD	RTL	RTL	NRTL	PL+	6 / 6	Defective	Normal

19	pandi	56	4	male	80162	Left eye TON	RTA	0	12	fracture B/L lamina papyracea, fracture squamous part of left temporal bone	SAH/ SDH	fracture lamina papyracea, fracture segments over optic canal	7	6 / 9	PL+	Normal	Defective	RTL	RAPD	NRTL	RTL	6 / 9	PL+	not improved	Normal	Defective	not improved	RTL	RAPD	NRTL	RTL	6 / 9	PL+	Normal	Defective
20	anjali devi	26	1	female	23979	Right eye TON	RTA	0	15	fracture greater wing of R sphenoid with fracture extending into body of sphenoid with sphenoid hemosinus, right ethmoid hemosinus, b/l preseptal emphysema with b/l pneumoorbit,left frontal bone communited fracture	EDH/pneumocephalus	right ethmoid and sphenoid hemosinus, fracture lamina papyracea	1	2 / 60	6 / 9	Defective	Normal	RAPD	RTL	RTL	NRTL	2 / 60	6 / 9	not improved	Defective	Normal	not improved	RAPD	RTL	RTL	NRTL	2 / 60	6 / 9	Defective	Normal
21	jeri durai	20	1	male	8720	Left eye TON	Assault	0	15	fracture left lamina papyracea, left ethmoid hemosinus with posterior fracture fragment extending to intraconal space, left frontal hemosinus with left frontal sinus anterior wall fracture extending to left superior orbital plate		left lamina papyracea fracture,left ethmoid and frontal hemosinus	3	6 / 6	0.5 / 60	Normal	Defective	RTL	RAPD	NRTL	RTL	6 / 6	0.5 / 60	not improved	Normal	Defective	not improved	RTL	RAPD	NRTL	RTL	6 / 6	0.5 / 60	Normal	Defective
22	mahendran	44	3	male	89542	Right eye TON	RTA	0	15	fracture medial, posterolateral wall of right orbit, b/l preseptal emphysema, fracture right ethmoid with ethmoid hemosinus	-	right posterior ethmoid hemosinus, oblique fracture line over optic canal, granulation tissue impinging on optic nerve	5	6 / 9	PL+	Defective	Normal	RAPD	RTL	RTL	NRTL	6 / 9	HM+	improved	Normal	Defective	not improved	RAPD	RTL	RTL	NRTL	6 / 9	HM+	Normal	Defective
23	jeyachandran	40	2	male	80210	Left eye TON	RTA	0	15	fracture left ethmoid sinus extending to ethmoid bone and lateral wall of left orbit with ethmoid and sphenoid hemosinus	-	fracture segment over anterior wall of sphenoid sinus	1	6 / 9	PL-	Normal	Defective	RTL	RAPD	NRTL	RTL	6 / 9	PL-	not improved	Normal	Defective	not improved	RTL	RAPD	NRTL	RTL	6 / 9	PL-	Normal	Defective
24	muppidathi	25	1	male	60606	Left eye TON	RTA	0	15	fracture left lamina papyracea, fracture body of sphenoid		fracture lamina papyracea, fracture segments over optic canal, sphenoid hemosinus	8	6 / 6	PL-	Normal	Defective	RTL	RAPD	NRTL	RTL	6 / 6	HM+	improved	Normal	Defective	not improved	RTL	RAPD	NRTL	RTL	6 / 6	HM+	Normal	Defective
25	samikannu	72	5	male	72150	Right eye TON	RTA	0	15	fracture right lamina papyracea, lateral wall of orbit, fracture body of sphenoid		fracture lamina papyracea, fracture segments over optic canal, sphenoid hemosinus	4	PL-	6 / 18	Defective	Normal	RAPD	RTL	RTL	NRTL	PL-	6 / 18	not improved	Defective	Normal	not improved	RAPD	RTL	RTL	NRTL	PL-	6 / 18	Defective	Normal
26	muthusamy	28	1	male	89312	Left eye TON	RTA	0	14	fracture left lamina papyracea, fracture body of sphenoid		left ethmoid hemosinus, left sphenoid hemosinus, fracture lamina papyracea	4	6 / 6	PL-	Normal	Defective	RTL	RAPD	NRTL	RTL	6 / 6	6 / 60	improved	Normal	Defective	not improved	RTL	RAPD	NRTL	RTL	6 / 6	6 / 60	Normal	Defective