Ocular Sequelae Following Head Trauma: A Review

Nikolas Apostolou BAppSc (Orth)

Address for correspondence:
N Apostolou
The Visual Screening Centre
30 Jaserfold St, Balwyn Nth. 3104.

Submitted: March 1996.
Accepted for publication: May 1996.

Abstract

To date there have been numerous studies which have investigated the nature of ocular abnormalities as a result of head injuries. However few have presented a full spectrum of disorders that can occur. Orthoptists are skilled in the diagnosis and treatment of trauma victims who have visual disturbance as a result of orbital fractures, cranial neuropathies, brainstem damage, cerebral lesions and soft tissue injuries.

Key Words:
head trauma, ocular, injuries, orthoptics.

Introduction

Head trauma may produce a myriad of visual and visually related disturbances. Rehabilitation of the head injured patient is aimed at developing new skills that will permit the patient to optimally function as a viable and independent member of society. Many visual complaints of the head injured patient however, are sometimes ignored, and visual disturbances are misdiagnosed for long periods of time [1,2]; thus hampering long term neurological and vocational rehabilitation efforts [3].

Orthoptic evaluation of the head injured patient should therefore be carried out routinely as part of the multi-disciplinary team approach to rehabilitation. The role of the orthoptist firstly being diagnostic, and secondly, therapeutic [4].

Orthoptic assessment can also be a valuable resource for monitoring the recovery rate of head injured patients, as changes in eye movements can provide a measure of the recovery or deterioration during the acute stage [5,6].

Epidemiology

Motor vehicle accidents tend to be the most common cause of head injuries [7,8,9,10]. Other causes include direct trauma to the head, injuries sustained from a fall, or as a result of work or recreational activities. In 1991, it was estimated that there were 10,000-12,000 Victorians with head injuries; and this number was growing by 2,000 per year. Head injuries are responsible for 5,000 hospital admissions per year, two thirds of these being under 25 years of age and 71% being males. This figure is comparable throughout many industrialised nations [2]. Approximately 75% of patients who survive head trauma require rehabilitation.

Patho-physiology Of Head Injury

The term 'head injury', for the purpose of this review, will encompass open and closed head injuries, as well as open and closed facial trauma. Open head trauma is defined as direct invasion through the skull, and closed head trauma occurring when there is no direct pathway from the environment to the soft tissue and injury site. The effects of head injuries may result via several mechanisms. The major forces being 'contact' injuries and/or 'deceleration/acceleration' injuries.

With a blow to the head, contusion of the brain occurs as the bone at the site of impact indents and damages the brain surface. Contact injuries also include epidural or subdural haematomas and skull fractures including blow-out fractures of orbital bones [8].

Acceleration/deceleration injuries are related to the movement of the brain in relation to the skull. Such movements may be in anterior-posterior and superior-inferior directions. Consequences of such movements include the tearing of veins bridging the space between the skull and the brain; disruption of cranial nerves and contré coup injuries.

Evaluation of the Head Injured Patient

Examination techniques will be varied, depending on the level of consciousness and the symptoms of each individual [5]. Evaluation

of each head injured patient should be aimed at describing the ocular deficits and localising lesions attributable to the head injury. It is therefore imperative that where possible, a general and ocular history be ascertained prior to examination. This will aid in identifying the presence of disease processes which may have ocular involvement, i.e. diabetes mellitus, thyroid eye disease etc.

Clinical Categories

1. Orbital Injuries.

Motor vehicles, heavy machinery, physical confrontations and sports injuries are responsible for an ever increasing accident rate. The eye and orbit are commonly involved in middle third facial injuries. Trauma to the orbit is commonly caused by a blunt blow to the face ie. a squash ball or a fist; or through the patient making contact with a hard surface, as is the case in motor vehicle accidents, ie. the face striking the dashboard.

The effects of orbital trauma may result in soft tissue injuries which cause swelling and haemorrhage leading to limitations in eye movements. Such injuries usually resolve spontaneously. Orbital floor fractures occur when the contents of the orbit are compressed by the blow resulting in the floor, medial wall, lateral wall or roof of the orbit being fractured.

Orbital fractures may be pure, where the orbital rim remains intact, and the fracture affects the floor (antral blow-out fracture) or less commonly the medial wall (ethmoid blow-out fracture); each of which consists of bone 0.5-1.0 mm thick [19]. An impure blow-out fracture involves orbital wall fracture in association with a fracture of the body of the zygoma or the rim of the orbit.

The patient may present with a variety of signs and symptoms which include:

- Limitation of ocular movement due to oedema, haemorrhage, incarceration of tissue within the fracture; or herniation of the orbital contents into the antrum.

- Displacement of the globe backwards, commonly referred to as enophthalmos. The enophthalmos is due to the contents of the orbit prolapsing into the fracture, with the displacement progressively worsening.

- Facial asymmetry usually evident in impure blow out fractures, where there is damage to the zygoma.

- Retraction of the globe when the eyes move in the direction opposite to the entrapment site.[23]

- Infra orbital anaesthesia causing loss of sensation on the skin just below the lower eyelid; resultant from damage to the infra-orbital nerve.[23]

- Pain which is most obvious when the patient looks away from the site of the lesion, ie. on up gaze in antral blowout fractures.

- Visual symptoms which are dependant on the amount of damage to the optic nerve [21]. There may be immediate and permanent blindness, or a field deficit which usually affects the inferior half of the visual field [19].

- Diplopia, which may not be initially evident due to the considerable amount of periorbital haemotoma. The excessive oedema will not allow the eyelids to be opened, and only once this oedema subsides, does the diplopia become evident. The type of diplopia will depend on the site of the fracture. In an antral blow out fracture, where there may be incarceration of the inferior oblique and/or the inferior rectus (this may include entrapment of muscle sheaths or fat), vertical diplopia occurs . Ethmoid blowout fractures result in horizontal diplopia due to entrapment of the medial rectus; also causing limitation of abduction [19]. Displacement of the body of zygoma will also cause horizontal diplopia due to lateral rectus muscle weakness [19]. It should also be noted that horizontal diplopia may become evident in antral blowout fractures as the horizontally acting muscles have connective tissue septa which extend to the orbital floor . Finally, supra orbital fractures may cause vertical diplopia due to involvement of superior oblique or superior rectus muscles [19]. Usually this type of muscle imbalance resolves spontaneously [23].

The incidence of diplopia (in the primary position) has been reported as high as 86% , and as low as 36% in orbital floor fractures. More than one third of all orbital blow out fractures have an associated ethmoid fracture, although isolated medial wall fractures are rare.

Management of patients with orbital fractures is usually conducted by facio maxillary surgeons with ophthalmic and orthoptic intervention being crucial in assessment of the trauma and diagnosis of the injury[25]. It is the orthoptist's role to check the visual acuity; chart the size of the deviation in all positions of gaze; and measure the field of binocular single vision. Surgery should then be considered if there is persistent diplopia; or incarceration or herniation of tissue into the fracture; which shows no sign of spontaneous recovery, and in cases of cosmetically unacceptable enophthalmos [25].

2. Soft Tissue Injuries.

Avulsed extra ocular muscles are another manifestation of head injuries and are categorised as soft tissue injuries [5]. This type of injury is usually the result of a penetrating wound. The medial rectus is the most commonly avulsed muscle followed by the inferior rectus, the superior rectus, lateral rectus and the obliques. Clinical features of the lacerated muscle include limitation of movement of the affected eye, incomitant strabismus and reduced saccadic velocity in the field of action of the injured muscle [5]. Management of the injured muscle involves re-insertion to the sclera, or rejoining of the lacerated muscle ends.

3. Refractive Errors.

(a) Traumatic Cataract.

Cataracts are usually the result of blunt trauma to the eyeball or from a foreign body injury . The lens usually becomes opaque shortly after entry of the foreign body, as the aqueous and at times, the vitreous penetrate the lens structure . The patient's immediate complaints are that of blurred vision, and redness of the affected eye. There may also be intraocular haemorrhage. It is possible that the eye may become soft if the vitreous or aqueous escapes from the eyeball. Complications may include infection, uveitis, retinal detachment and glaucoma [37].

(b) Lens Dislocation.

A contusion injury, such as a blow to the eye with a fist may cause partial or complete lens dislocation. The patient may not complain of any symptoms if the dislocation is partial, however if the dislocation is complete and the lens is floating in the vitreous, the patient may experience blurring of vision. Iridodonesis, a quivering of the iris on eye movement may also become evident - this is a common sign of lens dislocation, and is due to the lack of support to the lens.

(c) Traumatic Myopia.

The most common ocular complaint elicited from head injured patients was that of decreased vision[1]. This can be explained by the fact that many had either incorrect refraction or no refraction at all. Although diplopia is sometimes misinterpreted as blurred vision by the patient, Sabates et al 1991, suggests that most common complaints of blurred or decreased vision can be cured with a sometimes difficult, but accurate refraction. The occurrence of myopia after concussion injuries has been recognised since 1870 following the original observation by Kugel . The extent of the myopia varies from 1 to 6 dioptres. In most cases, the condition is transient although many cases show considerably long duration. The cause of the myopia is not clear, although two causes postulated are that of ciliary spasm, and weakening or rupture of the zonule. Spasm of accommodation initiated by the trauma is the most common etiologic factor. Characteristics of such spasm is associated with a loss of accommodative amplitude, and miosis, and disappears under the influence of atropine [41].

4. Traumatic Maculopathy.

Blunt trauma to the anterior segment of the eye may cause a contré coup injury to the macula called commotio retinae [39]. Retinal whitening occurs primarily in the outer retina, and this whitening may be confined to the macula area or conversely involve more extensive areas of the peripheral retina. The resultant impairment of central vision may be temporary as the whitening may clear completely; or it may be permanent, usually associated with a pigmented scar or macula hole.

5. Cranial Neuropathies.

(a) Neurogenic Paralytic Strabismus.

Typically, traumatic palsies of cranial nerves are caused by closed head injuries [7]. Traumatic damage of cranial nerves may be caused by tearing of the nerves, contusion, or compression. Resultant abnormalities might include varied disturbances of fusion (strabismus and diplopia), ptosis, pupil abnormalities and corneal anaesthesia; arising from damage to the ophthalmic branch of the trigeminal nerve (CNV). Ocular motility defects may occur when the cranial nerves controlling the

extra ocular muscles are affected. Except for the facial nerve (CNVII), the abducens nerve (CNVI), oculomotor nerve (CNIII) and trochlear nerve (CNIV) are the most commonly affected nerves in that order. As with any other motor nerve injury, weakness may be complete (paralysis), or partial (paresis) [5].

Deficits associated with oculomotor nerve damage, may include superior rectus and levator palperbrae superioris weakness (superior division of CNIII); medial rectus, inferior rectus and inferior oblique weakness (inferior division of CNIII), as well as damage to the pupillary fibres. As the third nerve controls so many muscles (intra ocular and extra ocular), a third nerve palsy may be complete or incomplete.

A complete third nerve palsy will result in paresis or paralysis of the medial, inferior and superior recti, the inferior oblique and sphincter pupillae; the ciliary muscle and the levator palpebrae superioris. The resultant ocular posture is that of an intorted, hypotropic and exotropic eye, with a dilated pupil and ptosis [24].

In an incomplete third nerve palsy, there may be damage to: superior or inferior divisions of the nerve, single muscle palsies, double elevator palsies, paresis of the extra ocular muscles supplied by CNIII with sparing of the intra ocular muscles [24].

As a consequence of complete third nerve palsies, a new pattern of abnormal movements may occur. This is a result of aberrant regeneration during the re-growth phase of the third nerve , where the regenerating autonomic and voluntary nerve fibres are misdirected. Clinical signs of aberrant regeneration include:
- elevation of the upper eyelid on down gaze [48,49],
- retraction of the globe on elevation and/or depression,
- constriction of the pupil on attempted adduction [24], however there is no reaction (or poor reaction) of the pupil to light stimulation [49],
- poor vertical responses to monocular vertical optokinetic testing [51].

Although the least commonly affected of the ocular nerves [48], the trochlear nerve (CNIV) may be damaged through trauma, resulting in superior oblique weakness, leading to symptoms including diplopia (torsional and horizontal) as well as V-pattern esotropias. Trauma is the most common aetiology of acquired fourth nerve paresis. The troclear nerve may be damaged anywhere along its course by direct orbital trauma, frontal trauma, or an oblique blow to the head [5]. The patient often assumes a compensatory head posture with the head tilted and face turned to the side of the unaffected eye, and the chin is depressed, so as to overcome the symptoms of diplopia and torsion.

Abducens nerve (CNVI) paresis, like trochlear nerve paresis, occurs frequently in head trauma [5], with the deviation usually being larger. Damage of the sixth nerve nucleus results in paresis or paralysis of the lateral rectus muscle, and therefore paralysis of ipsilateral horizontal gaze . Lesions to the sixth nerve are usually found where the nerve leaves the pons and passes through the subarachnoid space. Ocular posture of sixth nerve lesions is that of an esotropic eye (the deviation being more marked for distance fixation); and a compensatory head posture where the face is turned to the affected side [24].

(b) Visual Field Deficits.

Traumatic optic neuropathy has been described by many authors. The optic nerve is commonly injured following closed head trauma. Jeanet et al 1981 described an incidence of up to 13% of survivors of severe head injury. The most common area of injury sustained by the nerve is the intra nuclear portion, where the nerve is tethered to the dura and is relatively immobile [1]. Should a basal skull fracture occur and extend through the optic canal, it could readily damage the optic nerve, as the nerve occupies the entire canal space. Chiasmal damage is also possible when there is a blow to the frontal or parietal region. Lesions of the chiasm occur as a result of jarring to the head and sudden displacement of moveable parts of the brain, with the consequent ruptures of small blood vessels which enter the chiasm [60].

Optic nerve damage may result in transient visual loss, and visual field loss. This loss may be unilateral, bilateral and/or altitudinal. Such field losses may be mirrored by damage to the occipital lobe region. As in circulatory disturbances, trauma destroys masses of brain tissue at once. Portions of the visual pathway included in the lesions are likely to be totally interrupted.

Visual deficits following closed head injuries are not uncommon. Sabates et al 1991 described an incidence of 35% in their series of patients, with functional (tunnel) fields being the most common. The most severe extent of damage was that of cortical blindness. Other types of field deficits included:
- arcuate field anomalies (optic nerve damage),
- quadrantinopia
- bitemporal hemianopia
- homonymous hemianopia
- infero temporal island.

6. Cerebral Lesions.

The occipital cortex is comprised primarily of the parieto occipital visual association areas, which play a role in the control of smooth pursuit movements, and the primary visual cortex (area 17), which is the sensory cortex for sight. A lesion in the occipital lobe may result in visual field deficits, as described in section 5(b), but it may in turn, also cause disruptions to eye movements. Such disruptions are apparent in patients with hemianopic field deficits, which result in cogwheeling eye movements instead of smooth eye movements, as well as saccadic dysfunction. However this is an adaptation and not an ocular motor defect in the true sense.

The purpose of smooth pursuit movements is to maintain a clear image of a moving target (ie. maintaining foveal stimulation [24]). The areas of the cerebral cortex which initiate and co-ordinate smooth pursuit movements are many and varied. Generally, control over smooth pursuit movements is primarily in the parieto occipital visual association areas; with control being ipsilateral ie. left occipital cortex controlling movements to the left. Hence any damage to the parieto occipital area may lead to anomalous pursuit movements. Damage to the cerebellum may also impair smooth pursuit function as the cerebellum plays a role in the co-ordination of eye movement and fine motor control.

Saccadic movements are described as voluntary rapid eye movements between two points. The speed of this movement is usually between 200 and 400 degrees per second, having a peak velocity of 700 degrees per second [67]. Control of horizontal saccades is via the frontal eye fields (Brodman's area 8) for voluntarily directed saccades; and the superior colliculi for re-orienting gaze to novel stimuli. The cerebellum is also responsible for controlling saccades, by monitoring pulse size and aiding accurate and co-ordinated eye movement.

Damage to one frontal lobe produces a conjugate deviation of the eyes to the side of the injury and an inability to look towards the side opposite the lesion [5]. This sort of disturbance is usually transient. Bilateral lesions of the frontal lobes can produce ocular motor apraxia, a quite marked eye movement disorder, where there is an inability to voluntarily direct horizontal saccades to both sides.

7. Intra Axial Brainstem Damage.

Mid brain lesions, and brainstem damage can lead to the disruption of conjugate gaze movements, accommodation convergence relationships, nystagmus and a variety of disturbances of resting fixation, ie. skew deviation or Parinaud's Syndrome. A horizontal gaze palsy may result from an ipsilateral pontine lesion or a contra lateral frontal lobe lesion. Unilateral lesions produce ipsilateral conjugate gaze paresis, and bilateral lesions (at the level of the abducens nucleus) may cause paralysis of all horizontal eye movements [5]. Vertical gaze palsies are indicative of pretectal damage. As vertical eye movements are under bilateral hemispheric and mid brain control, vertical gaze palsies due to isolated cerebral lesions do not occur [24]. In trauma, it is likely that up gaze paresis may be associated with other signs of dorsal mid brain damage. These include light near dissociation of the pupils, limited convergence, eyelid retraction and skew deviation.

Lesions in the pretectal mid brain, rostral to the third nerve nucleus may cause paralysis of convergence. Divergence insufficiency has also been reported after trauma, as a lesion in the area of the sixth nerve nucleus can produce paralysis of divergence. Paralysis of accommodation following head trauma has also been described [77,78], with the deficiency usually being temporary, although recovery may be incomplete. Baker and Epstein 1991, suggest that it is not uncommon for the condition to be mistaken for inattention, lack of effort or alexia [5]. The simple prescription of convex lenses may compensate for the paralysis.

Vestibular dysfunction is also possible in the head injured patient. Symptoms include vertigo and oscillopsia; although due to diffuse representation of the vestibular system in the brainstem, makes isolated lesions unlikely [5].

Central lesions are usually accompanied by other signs of brainstem dysfunction.

8. Glaucoma Secondary to Trauma.

Head trauma and specifically contusion injuries of the globe may be associated with an early rise in intra-ocular pressure. This rise in ocular pressure is due to hyphema. The presence of free blood in the anterior chamber blocks the trabecular meshwork which is rendered oedematous by the injury. Management of the condition is initially medical, and surgery may be necessary if the pressure remains elevated [39].

Visual Deficits and their Implications.

The orthoptist is capable of making a significant contribution to evaluation of the visual status and level of function of the head injured patient, thereby aiding the rehabilitation process. It is important that all therapists are made aware of any visual deficiencies the head injured patient may possess, as many ocular anomalies may interfere with treatment regimes being initiated by any member of the rehabilitation team. Such anomalies may range from the easily correctable blurring of vision to the complex gaze anomalies and the chronic head postures adopted by patients to overcome symptoms of diplopia and pain of ocular movements. Orthoptic findings can aid effective therapy.

References

1. Sabates N R, Gonce M A, Farris B K. Neuro ophthalmological findings in closed head trauma. J Neuro-Ophthal. 1991; 11(4): 237–277.

2. Cohen Rein L D. The effects of head trauma on the visual system: The doctor of optometry as a member of the rehabilitation team. 1992; 68(8): 530–536.

3. Rodriguez Barros R, Bottinelli M D. The study of ocular motility in the comatose patient. J Neurol Sci. 1966; 3: 183–06.

4. Mein J. The orthoptic role in head injuries. Aust Orth Jnl 1980–81; 18: 10–14.

5. Baker R S, Epstein A D. Ocular abnormalities from head trauma. Surv Ophthalmol. 1991; 35(4): 245–267.

6. Ron S, Najeson T, Pryworkin W. Eye movements in brain damaged patients. Scand J Rehab Med. 1978; 10:39–44.

7. Lee J P. Ocular motility consequences of trauma and their management. Brit Orth J. 1983; 40: 26–33.

8. Fitzsimmons R, Fells P. Ocular motility problems following road accidents. Brit Orth J. 1989; 46: 40–48.

9. Heinze J. Cranial nerve avulsions and other neural injuries in road traffic accidents. Medical J Aust. 1969; 1246–1249.

10. Keane J R. Neurologic eye signs following motorcycle accidents. Arch Neurol. 1989; 46: 761–762.

11. Headway Victoria Head injury impact impact report. April 1991.

12. Porter M F. Head injuries following road traffic accidents. Brit Orth J. 1983; 34: 83–88.

13. Raju N S. Ocular manifestations in head injuries Indian J Ophthal. 1983; 31(6): 789–792.

14. Sieve E, Freishart B. Perceptual and cognitive dysfunctions in the adult stroke patient. Slack Inc. 1986.

15. Davidoff A D, Kessler H R, Laibstain D F et al. Neural behavioural sequelae of minor head injury: A consideration of post–concussive syndrome versus post–traumatic stress disorder. Cognitive Rehabilitation. 1988; 8.

16. Gurdjian E S, Re–evaluation of the bio mechanics of blunt injury of the head. Surg Gynecol Obstet. 1975; 140: 845–850.

17. Roy W, Beck M D. Ocular deviations after head injury. Am Orth J. 1985; 35: 103–107.

18. Gennarelli T A, Head injury in man and experimental animals: Clinical aspects.

Acta neurahir (Wein) (Suppl). 1970; 32:1–13.

19. Garston J B, Kirkpatrick J. Injuries around the orbit. Brit Orth J. 1970; 27:37–50.

20. Orthoptic Department. Hallamshire Hosp, Sheffield. Orthoptics in orbital injuries. Brit Orth J. 1970; 27:58–69.

21. Fells P. Orbital wall fractures. Brit Orth J. 1970; 27.

22. Converse J M, Smith B, Brit J Plast Surg. 1957; 9:265.

23. Bleeker G M, Gillisen J P A, Oei T H et al, Orbital fractures. Brit Orth J. 1972; 29:25–45.

24. Mein J, Harcourt B, Diagnosis and Management of Ocular Motility Disorders. Blackwell London. 1986.

25. Anderson R L, Panje W R, Gross C E, Optic nerve blindness following blunt head trauma. Ophthalmol. 1982; 89: 445–455.

26. Merchant, N.E. Fractures of the middle third of the face. Brit. Orth. J. 1983; 40: 34–39.

27. Emery J M, von Noorden G K, Schlrnitzauer D A, Orbital floor fractures: a long term follow up of cases without surgical repair. Trans Ophthalmol Otolaryngol. 1971; 802–812.

28. Korneef L, Sectional Anatomy of the Orbit. Escalapius, Birmingham. 1979.

29. Daroff R B, Hoyt W F, The control of eye movements. Academic Press, New York. 1971.

30. Liebsohn J, Burto T C, Scott W E, Orbital floor fractures: a retrospective study. Ann Ophthalmol. 1976; 8:1057–1062.

31. Converse J M, Smith B, Obear M F et al. Naso orbital blowout fractures: A ten year survey. Plast Reconst Surg. 1967; 39: 20–36.

32. Cullen J F. Orbital fractures and other ocular complications of head injuries. Head injuries: Proceedings of an international symposium. Churchill & Livingstone, Edinburgh. 1971.

33. Smith B, Regan J F Jr. Blowout fracture of the orbit: Mechanism of correction and internal orbit fracture. Am J Ophthalmol. 1957; 733–739.

34. Mailer C M, Avulsion of the inferior rectus. Can J Ophthalmol. 1974; 9:262–266.

35. Helveston E M, Grossman R D, Extraocular muscle lacerations. Am J Ophthalmol. 1974; 81:754–760.

36. A M A, Cataract in the otherwise healthy adult eye. Am Academy Ophthalmol. 1989.

37. Rasooly R, BenEzra D. Congenital and traumatic cataract: The effect on axial length. Arch Ophthalmol. 1988; 106:1066–1071.

38. Wilson J. Clearing the cataract backlog. Brit J Ophthalmol. 1987; 71:158–161.

39. Vaughn D G, Asbury T, Riordan–Eva P. General Ophthalmology. Prentice Hall London. 1992.

40. Duke–Elder S, Abrams D. System of Ophthalmology Vol V. Henry Kimpton, London. 1970.

41. Duke–Elder S, Abrams D. System of Ophthalmology Vol XII. Henry Kimpton, London. 1970.

42. Gass J D M. Stereoscopic atlas of macula diseases. Mosby, London 1987.

43. Akiba J. Risk of developing a macula hole. Arch Ophthalmol. 1990; 108:1088–1092.

44. Zagora E. Eye injuries. Charles C Thomas, Springfield II. 1970.

45. Stevens N M. Post concussion syndrome. J Neurosurg Nurs. 1982; 5:239–244.

46. Vogel M S. An overview of head trauma for the primary care practitioner; Part I. Etiology, diagnosis, and consequences of head trauma. J Am Optom. 1992; 63: 537–541.

47. Rucker C W. Paralysis of the third, fourth, and sixth cranial nerves. Am J Ophthalmol. 1972; 73:451–453.

48. Rush J A, Young B R. Paralysis of cranial nerves,third, fouth, and sixth: cause and prognosis in 1000 cases. Arch Ophthalmol. 1981; 99:76–79.

49. Forster R K, Shatz N J, Smith J L. A subtle eyelid sign in abberant regeneration of the third nerve. Am J Ophthalmol. 1969; 67:696–698.

50. Lepore F E, Glaser J S. Misdirection revisited: a critical appraisal of the acquired oculomotor sykinesis. Arch Ophthalmol. 1980; 98:2206–2209.

51. Bender M B. The nerve supply to the orbicularis muscle and the physiology of movements with the upper lid: with particular reference to the pseudo Graef phenomenon. Arch Ophthalmol. 1936; 15:21–30.

52. Harley R D. Paralytic strabismus in children. Etiologic incidence and management of third, fourth and sixth nerve palsies. Ophthalmol. 1980; 86:24–43.

53. Burger L J, Kalvin N A, Smith J L. Acquired lesions of the fourth cranial nerve. Brain. 1970; 92:567–574.

54. Coppeto J R, Lessell S.

Cryptogenicunilateral paralysis of the superior oblique muscle. Arch Ophthalmol. 1978; 96:275–277.

55. Syndor C F, Seaber J H, Buckley E G. Traumatic superior oblique palsies. Ophthalmol. 1982; 89:134–138.

56. Mazow M L, Tang R. Strabismus associated with head and facial trauma. Am Orth J. 1982; 32:31–35.

57. Crouch E R Jr, Urist M L. Lateral rectus muscle paralysis associated with closed head trauma. Am J Ophthalmol. 1975; 79: 990–996.

58. Schneider R C, Johnson F D. Bilateral traumatic abducens palsy: a mechanism of injury suggested by the study of associated cervical spine fractures. J Neurosurg. 1971; 34:33–37.

59. Anderson R L, Nevins R C Jr. Optic atrophy after ocular contusion. Am J Ophthalmol. 1972; 73:278–280.

60. Ferris F. Traumatic lesions of the visual pathways. Handbook of Clinical Neurology. 1974; Vol. 24.

61. Hillman J S, Myska V. Complete avulsion of the optic nerve. Brit J Ophthalmol. 1975; 59:503–509.

62. Kline L B, Morawetz R B, Swaid R N. Indirect injury of the optic nerve. Neurosurg. 1984; 14:756–764.

63. Jennet B, Snoeck J, Bond M R et al. Disability after severe head injury: observations on the use of the Glasgow outcome scale. J Neurosurg Psychiat. 1981; 44: 285–293.

64. Rucker C W. The interpretation of visual fields. Am Acad Ophthalmol. 1957.

65. Tierhey D W. Visual dysfunction in closed head injury. J Am Optom Assoc. 1988; 59:614–666.

66. Schikawa T I, Jorzumi J, Sakuma K et al. Long term cortical blindness after head trauma. Jpn J Psych Neurol. 1987; 41:19–23.

67. Leigh R J, Zee D S. The Neurology of Eye Movement. Davis, Philadelphia. 1983.

68. O'Day J. Horizontal movement disorders of the eye. Brit Orth J. 1982; 39:12–16.

69. Hughes J L, O'Connor P S, Larsen P D et al. Congenital vertical motor apraxia. J Neuro–Ophthalmol. 1985; 5:153–157.

70. Carroll R P, Seaber J H. Acute loss of fusional convergence following head trauma. Am Orth J. 1980; 24:57–59.

71. Kingele T G, Schultz R, Murphy M G. Pontine gaze paresis due to traumatic craniocervical hypertension. J Neurosurg. 1980; 53:249–251.

72. Beck R W. Ocular deviations after head injury. Am Orth J. 1985; 23:103–107.

73. Cogan D C. Paralysis of down gaze. Arch Ophthalmol. 1974; 91:192–199.

74. Buttner–Ennever J A, Akert K. Medial rectus subgroups of the oculomotor nucleus and their abducens internuclear input in the monkey. J Comp Neurol. 1981; 197:17–27.

75. Jampel R S. Convergence, divergence, pupillary reactions and accommodation of the eyes from Faradic stimulation of the Marque brain. J Comp Neurol. 115.

76. Rutlowski P C, Burian H M. Divergence paralysis following head trauma. Am J Ophthalmol. 1972; 73:660–662.

77. Duane A. Subnormal accommodation. Trans Am Ophthalmol Soc. 1925; 23: 159–183.

78. Holmes G M. Disturbance of vision by cerebral lesions. Brit J Ophthalmol. 1918; 2:353–383.

79. van Bursick E M. Clinical Atlas of Glaucoma. Saunders. 1986.