ORIGINAL CONTRIBUTION

Disorders of Ocular Motility Following Head Trauma

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Objective: To determine the types and frequency of symptomatic ocular motility disturbances following head trauma and their association with severity of trauma.

Design: Retrospective study of patients with (1) diplopia unless visual loss is present, (2) heterotropia for far or near targets, and (3) prior head injury.

Setting: Office and in-hospital consulting practice of a university neuro-ophthalmologist.

Subjects: Sixty patients with posttraumatic ophthalmoplegia.

Main Outcome Measures: Paralytic and nonparalytic heterotropias were quantitated in prism diopters or percentage limitation of ductions. Convergence insufficiency was assessed by determining the near point of convergence.

Results: Fifty-one patients had nuclear or infranuclear findings, ie, trochlear palsies (n=20), oculomotor palsies (n=17), abducens palsies (n=7), combined palsies (n=5), and restrictive ophthalmopathy (n=2). Nine patients had supranuclear dysfunction, including seven patients with convergence insufficiency. Bilateral ocular motor palsies and combined palsies were significantly (by means of χ^2 test) associated with head trauma of severity sufficient to cause corticospinal tract dysfunction. Individual or combined ocular motor palsies were not significantly (by means of χ^2 test) associated with intracranial hemorrhage and/or skull fracture or loss of consciousness.

Conclusions: Trochlear palsy was the most common nuclear or infranuclear basis for traumatic diplopia, and convergence insufficiency was the most common supranuclear cause of double vision. Head trauma distinguished by upper motor–neuron signs was correlated with specific subsets of disordered ocular motility.

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ALTHOUGH Hutchinson's studies[1] of laborers' head injuries provided a clinicopathologic basis for the nascent understanding of the relationship of head trauma and disordered ocular motility over 125 years ago and the subsequent ascendancy of motorized transportation (and its attendant mishaps) has increased the modern clinician's exposure to traumatic ophthalmoplegia, no consensus regarding the relative frequency of specific kinds of ocular motor dysfunction following head injury has been forthcoming. The most commonly injured ocular motor nerve was variously reported as the oculomotor nerve,[2,3] the trochlear nerve,[4] and the abducens nerve.[5-7] With this background of conflicting observations, I studied patients with abnormal ocular motility following head trauma for the purpose of determining the types of ocular motor dysfunction, their relative frequency, and their association with severity of trauma.

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RESULTS

Head trauma was attributed to motor vehicle accidents in 53 patients, sports in four patients, falls in three patients, assault in one patient, and an industrial accident in one patient (two patients had multiple head injuries). Trauma was sufficiently severe to cause loss of consciousness in 48 patients, skull fracture in 25 patients, and intracranial hemorrhage in 28 patients (with intracerebral hemorrhage [n=21], with subdural hematoma [n=4], with subarachnoid hemorrhage [n=2], and with

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PATIENTS AND METHODS

In an 8-year retrospective study, 60 patients (28 men, average age, 33.9 years, with an age range of 17 to 66 years; 32 women, average age, 30.7 years, with an age range of 8 to 67 years) fulfilled the criteria of (1) diplopia unless visual loss was present, (2) heterotropia for far or near targets, and (3) prior head injury. Fifty-eight patients underwent outpatient evaluations, and two patients were seen while hospitalized. An average of 25.1 months elapsed between head injury and neuro-ophthalmologic evaluation. I performed the neurologic examination and measured the ocular motility of all patients. Paralytic and nonparalytic heterotropias were quantitated in prism diopters or percentage of limitation of ductions. The near point of convergence was measured by moving a fixation target along the sagittal plane toward the patient's nose until the patient reported diplopia or one eye turned out. Convergence insufficiency was characterized by a near point of convergence farther than 10 cm from the lateral bony margin of the orbit.[8]

epidural hematoma [n=1]). Twenty-eight patients had upper motor–neuron signs.

Fifty-one patients had nuclear or infranuclear ocular motor abnormalities, ie, trochlear palsies (n=20); oculomotor palsies (n=17); abducens palsies (n=7); combinations of oculomotor, trochlear, and abducens palsies (n=5); and restrictive ophthalmopathy due to orbital fracture (n=2) (**Table**). Bilateral ocular motor palsies (χ^2, $P=.001$) and combined palsies of at least two different ocular motor nerves (χ^2, $P=.043$) were significantly associated with head trauma of severity that was sufficient to cause corticospinal tract dysfunction. Bilaterality of traumatic ocular paresis was not significantly (by means of a χ^2 test) linked to a particular ocular motor nerve. Individual, bilateral, or multiple ocular motor nerve palsies were not significantly (by means of a χ^2 test) associated with intracranial hemorrhage and/or skull fracture or loss of consciousness.

Nine patients had supranuclear disturbances of ocular motility, including seven patients with convergence insufficiency (Table). Convergence insufficiency was not significantly (by means of a χ^2 test) associated with severity of head trauma as characterized by intracranial hemorrhage and/or skull fracture, corticospinal tract deficits, or loss of consciousness.

Ocular Motor Findings in Diplopia of Traumatic Origin

Abnormalities	No. of Patients
Nuclear/infranuclear	
Unilateral third-nerve palsy	15
Bilateral third-nerve palsies	2
Unilateral fourth-nerve palsy	14
Bilateral fourth-nerve palsies	6
Unilateral sixth-nerve palsy	4
Bilateral sixth-nerve palsies	3
Unilateral third- and fourth-nerve palsies	2
Unilateral third-, fourth-, and sixth-nerve palsies	1
Unilateral third- and fourth- and bilateral sixth-nerve palsies	1
Unilateral fourth- and sixth- and bilateral third-nerve palsies	1
Hypertropia secondary to restrictive ophthalmopathy	2
Supranuclear	
Convergence insufficiency	7
Decompensation of intermittent esotropia	1
Convergence spasm	1
Total	60

COMMENT

Trochlear nerve palsy was the most common nuclear or infranuclear cause of double vision in this series of 60 patients with traumatic diplopia. A similar predominance of trochlear palsies was found in 181 patients with closed head injury studied by Sabates et al.[4] Although other series have reported oculomotor nerve[2,3] or abducens nerve[3,6,7] palsies as the most frequent ocular motor palsies in head trauma, this may reflect specialty bias since "fourth nerve pareses tend to be underdiagnosed on neurologic and neurosurgery services."[9] In the acute setting of head trauma, the patient may not be sufficiently alert to assist in the diagnosis of trochlear palsy. Consequently, the sensitivity of detection of trochlear palsy may be greater after the patient has had time to recover from the head injury, and it is notable that, in series in which trochlear palsies predominate, the average time from injury to time of examination was 8.5 months[4] and 25.1 months (this series).

The clinical aphorism that "all traumatic fourth nerve pareses should be assumed to be bilateral until examination proves otherwise"[10] may enhance the clinician's sensitivity to a subtle trochlear paresis accompanying a clearly evident trochlear paresis. The present series did not find that traumatic bilateral fourth-nerve palsies occurred significantly more frequently than bilateral third- or sixth-nerve palsies.

Can traumatic ocular motor palsy serve as a marker for severity of head trauma? The answer is affirmative in the special circumstances of bilateral ocular motor palsies or combinations of different ocular motor palsies that are correlated with head injury severe enough to cause pyramidal tract deficits. Other characteristics of head injury, including intracranial hemorrhage, skull fracture, and loss of consciousness, were not linked with specific subsets of disordered ocular motility. These gross cranial insults are not reliable predictors of ophthalmoplegia, and this underscores the complexity of the relationship of head trauma to neural injury. A similar relationship may exist in diffuse axonal injury that occurs more frequently in fatally head-injured patients without skull fracture than in those with skull fracture.[11] In addition to diffuse axonal injury of the brain stem,[10] there are other central and peripheral mechanisms, eg, brain-stem infarction and cranial nerve root stretching or avulsion, postulated to underlie traumatic ophthalmoplegia[7,10] and some of these can become operative in cases where no skull fracture, intracranial hemorrhage, or loss of consciousness occurred. The present study reaffirms that it is not

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usually the skull injury per se but the damage sustained by the brain that ultimately determines the clinical outcome.[1]

Convergence insufficiency was the most common supranuclear cause of double vision, and it has been frequently documented in series of patients with head trauma.[4,10] The anatomic substrate of convergence remains elusive, and no discrete nucleus controlling vergence has been found in humans. Some neurons involved in the control of vergence lie 1 to 2 mm dorsal and dorsolateral to the oculomotor nucleus in the mesencephalic reticular formation.[12] It has not been resolved whether medial rectus motoneurons or oculomotor interneurons are the individual neurons discharging exclusively for vergence within the oculomotor nucleus. Even less is known about higher-level vergence inputs.[13] In the absence of known peripheral mechanisms for convergence insufficiency, injury to the brain stem or more rostral structures governing vergence accounts for convergence insufficiency in more than 10% of the patients in this series. The occurrence of convergence insufficiency may be underestimated in this series since determination of a "remote" near point of convergence[8] may be a less sensitive test of convergence insufficiency than measuring decreased fusional amplitudes and defective vergence might be "concealed" by an ocular motor palsy, eg, bilateral abduction palsies might mask diminished convergence in a patient with traumatic injury to the sixth nerves and the midbrain vergence "centers."

As might be extrapolated from the well-known predominance of motor vehicle accidents as a cause of head injury,[14] vehicular injury accounted for diplopia in over 88% of the patients in this series. However, the great majority of head-injured patients do not develop diplopia.[3,10] Although this study suggests that the clinician should look for (in descending order of frequency) trochlear, oculomotor, and abducens nerve palsies and convergence insufficiency in patients complaining of diplopia following head injury, further research is required to elucidate the critical factors that determine why some head injuries cause double vision and others do not.

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