Neuro-Ophthalmology in Severe Head Injury*

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If asked what the significance of neuro-ophthalmology is in the evaluation of severe head injury, many medical and surgical practitioners of neurology would promptly reply that it consists of monitoring pupillary reactivity in anticipation of the uncal herniation syndrome. A discussion of two broad premises of a factual nature, however, should easily convince these same practitioners that the neuro-ophthalmic evaluation of patients with severe head injuries offers far more than the Hutchinson pupil.

Anatomically, the substrate of the eye-brain mechanism is prodigious. It includes every cerebral lobe, the basal ganglia, all divisions of the brainstem, the cerebellum, half of the cranial nerves, and one-third of the spinal cord. The eye-brain mechanism can be involved or interrupted at virtually any level of the neuraxis. The resultant signs and symptoms provide a kaleidoscope of pupillary abnormalities, ocular motor disturbances of a peripheral and central nature, and visual disturbances ranging from impairment or loss of acuity and field to complex perceptual and associative visual disturbances.

The second factual premise is the extent of the head injury problem. Six percent of all disabling injuries involve the head. Automobile accidents, but one aspect of trauma, have a 66% incidence of head injuries. Within the American population at large, it is calculated that one person in two hundred will require medical care for head injury each year. Given the anatomic extent of the eye-brain mechanism and the magnitude of the head injury problem, the incidence and variety of the neuro-ophthalmic signs and symptoms of head injuries become mind boggling. What can the neuro-ophthalmic aspect of head injuries provide in terms of patient evaluation and care?

In the initial evaluation of any head injury victim, a thorough neuro-ophthalmic examination will provide important clues to localization of sites of trauma, whether they are primary or secondary, direct or remote effects. Analysis of this information will facilitate diagnosis and prompt, proper therapy can be instituted. Despite these efforts, however, a number of neuro-ophthalmic sequelae of head injury will remain for the patient to recognize as he recovers from more serious aspects of the head injury. Many of the neuro-ophthalmic symptoms and signs demonstrated go beyond simple signposts in directing diagnostic considerations, investigations, and therapeutic endeavor. They serve a significant role in prognostication of the patient’s course and residual neurologic and neuro-ophthalmic handicap. The neuro-ophthalmic evaluation must not be limited to the initial patient evaluation which may be not only remarkably hectic but may also be restricted by aspects of the trauma that either prevent adequate evaluation or are of such a nature that they are life-threatening and preclude neuro-ophthalmic evaluation. The neuro-ophthalmic examination should be repeated at clinically appropriate points during the hospital course and should be utilized as a monitor for following the patient’s long-term progress.

The value of the neuro-ophthalmic aspects of trauma are not restricted to the primary care of the

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patient. Suitable documentation of the neuro-ophthalmic examination can be utilized during the primary patient care and follow-up as a tool in achieving a better understanding of the basic mechanisms of brain injury. During recent years at the Medical College of Virginia, close cooperation between neurosurgery and neuro-ophthalmology has provided the opportunity to implement this concept. We are presently compiling data, helpful not only in primary patient care but also in understanding the basic mechanism of brain injury.

The broad extent of the neuro-ophthalmic aspects of brain injury has been emphasized but space prohibits comprehensive discussion of its full range. An attempt, therefore, will be made to illustrate selected aspects of the neuro-ophthalmic consequence of brain injury which may be both of interest and benefit.

An appropriate beginning to the discussion of neuro-ophthalmic consequences of trauma is the consideration of the concept of preexisting brain disease as a precursor to trauma. Eyster clearly explained that minor head trauma in the presence of basal intracranial tumors may well produce unexpected neuro-ophthalmic signs of an oculomotor nature. Other neuro-ophthalmic signs can be produced by trauma and appropriately situated tumors. It is also clear that previously undiagnosed intracranial pathology may increase a patient’s vulnerability to traumatic incidents. In a similar vein preexisting signs of a neuro-ophthalmic nature, either recognized or unknown, though unrelated and clearly independent of the traumatic incident, may cause significant diagnostic consternation.

In introducing the topic, neuro-ophthalmology of severe head injury, it was noted that perhaps the most commonly sought neuro-ophthalmic sign of trauma was a pupillary abnormality. The abnormality most feared is the Hutchinson pupil—the dilation of the pupil ipsilateral to a mass lesion, producing transtentorial uncal herniation with third nerve impingement. It is generally an evolving sign, although it may be present by the time of the patient’s initial medical attention. Because of its sinister import, it must be differentiated from a similar pupillary abnormality—traumatic mydriasis.

Patients with severe head injuries also frequently receive direct blows to the eye. With direct blows to the globe, contusion of the iris may occur which results in a dilated and fixed pupil, usually unilaterally. If seen early, the initial response to the contusion will be miosis; dilation and iridoplegia follow within minutes, however, and may last minutes, hours, days, or may be permanent. Often other signs of periorbital trauma will point to the correct diagnosis of traumatic mydriasis. In selected cases, however, it may be impossible to differentiate a Hutchinson pupil from traumatic mydriasis. In these situations investigations to rule out the transtentorial herniation syndrome are imperative.

Oculomotor nerve palsies in head trauma are common; they are also significant parameters for localization and valuable monitors of the evolution of the trauma patient’s course. Their diagnosis is most difficult in the comatose patient. In this situation, the third nerve paralysis is most easily recognizable, basically, because of the prominent features of ptosis, mydriasis, and generally prominent deviation of the eye downward and outward. Sixth nerve palsies are less readily documented and fourth nerve palsies are almost never recognizable in the presence of coma. The oculomotor evaluation in comatose patients, however, is facilitated with the use of doll’s head phenomenon and caloric responses. Differentiation of primary and secondary palsies is of great practical significance to the physician responsible for the care of the trauma patient. Oculomotor palsies, as primary traumatic injuries, are almost always present immediately. Secondary palsies, reflecting an evolving intracranial mass, generally occur many minutes to hours or days later. If the trauma patient is delayed in receiving medical attention, or if the initial examination is incomplete, this differentiation may not be possible.

Clearly, early and accurate evaluation and documentation of ocular motility, as well as its continued monitoring during the patient’s recovery, is important. The ophthalmologist, who must commonly deal with the motility disturbance at a time distant from the injury, may be extremely dependent upon this documentation for his analysis and decision regarding surgery of the paretic muscle. It is currently recommended that surgery on paretic muscles be performed at any point after the fourth month, if maximal improvement has occurred. Statistics on the incidence of oculomotor involvement in trauma are less reliable than is desirable, due largely to misdiagnosis. Involvement of the third and sixth cranial nerves is probably nearly equally common. Fourth nerve injury is distinctly less common.

Third cranial nerve palsies are the result of significant frontal injuries generally associated with
concussion and skull fracture; they may be isolated sequelae of trauma. The site of third nerve injury is probably at its point of dural penetration at the posterior aspect of the cavernous sinus. The mechanism of injury is probably by virtue of shearing and stretching forces exerted upon the nerve with movement of the brainstem and skull base. Traumatic third nerve palsy is almost always unilateral, bilateral involvement being so rare as to suggest an additional pathologic process. Aberrant reinnervation is common in traumatic third nerve palsy and generally becomes evident at two-to-three months after injury. Lid and pupil signs are easily recognized.

Although fourth nerve palsy is less common than third and sixth nerve injury, it is by no means rare. It is easily missed. Fourth nerve involvement is often bilateral. Usually, it is the result of a vertex or frontal blow in which the dorsal midbrain and anterior medullary velum are contused on the tentorium. Motorcycle accidents are the common inciting event and permanent dysfunction is not uncommon.

Sixth nerve palsies commonly occur with frontal blows; they may be bilateral and, if the mode of onset is unknown or not documented, it may be impossible to differentiate direct injury of the sixth nerve from the secondary effects of increased intracranial pressure. Injuries producing sixth nerve paralysis may be based upon the presence of petrous ridge fractures. In this circumstance, associated paralysis of the seventh and eighth cranial nerve is common.

Injuries producing fractures of the superior orbital fissure frequently result in combined involvements of the third, fourth, and sixth cranial nerve. Such injuries have associated optic nerve injury and visual loss 50% of the time. Trigeminal and facial nerve injury occur an additional 25% of the time.

Frontal and superior orbital fractures may be associated with ptosis and elevator palsy due to paresis of the superior rectus. Although this is the superior division of the third nerve, it indicates involvement at a different site—within the orbit. Associated involvement of the superior oblique as well as the optic nerve may occur.

Also to be distinguished from true third nerve injury is the elevator palsy caused by the incarceration of the inferior rectus in "trapdoor" fractures of the orbital floor. This, as part of the orbital blow-out fracture syndrome, is usually associated with entophthalmos and sensory loss in the distribution of the inferior orbital nerve.

Supra- and internuclear ophthalmoplegia, from brainstem contusion on the free edge of the tentorium or from other less clear mechanisms, may be confused with partial third nerve injury. Their characteristic appearance and a knowledge that they may occur will usually avoid confusion.

The presence of a congenital Duane's retraction syndrome may cause confusion by its resemblance to a traumatic sixth nerve injury. Past history and the characteristic refraction of the globe with associated narrowing of the palpebral fissure on adduction will usually assist in the distinction. In a comatose patient, differentiation may not be possible.

The presence of obvious evidence of local orbital injury will generally suffice to distinguish oculomotor palsies of myogenic origin based upon local hemorrhage, contusion, and laceration of muscle. Obviously they may coexist with nerve injuries and their separation may be impossible.

Finally, one must recognize that skew deviation and wandering, dysconjugate eye movements are common in the coma of concussion. They are felt to represent disruption of oculomotor integration by the same mechanism responsible for the alteration of consciousness—physiologic alteration of the reticular formation. With return of consciousness, oculomotor function also returns to normal.

That blindness—visual loss—is a common result of head injury, is reflected in a quotation attributed to Hippocrates: "Dimness of vision occurs in injuries to the brow and in those placed slightly above." Visual loss can occur at any point in the visual pathways. It is most commonly encountered, if we exclude the eye itself, at the beginning and end of the intracranial pathways—the optic nerve and the occipital cortex. The differentiation of optic nerve and cortical blindness is seldom a problem. The exact definition of the mechanism of visual loss in both may be difficult yet extremely important.

The mechanism of optic nerve injury is varied. Generally, the blow is severe but may be extremely mild. The site of injury is most frequently the optic foramen, with involvement of contiguous orbital and intracranial optic nerve. Despite this localization, x-rays are seldom helpful. If visual loss is immediate and severe, the mechanism is likely to have been tearing, nerve hemorrhage, or contusion necrosis. If the onset has been delayed minutes-to-hours, hemorrhage into the vaginal sheaths, secondary...
edema or vascular compromise may be responsible. It is important to separate these two presentations since delayed visual loss may indicate a situation reversible by decompressive surgery. Immediate visual loss is virtually never remediable by surgical decompression. Unfortunately, the time of onset of visual impairment is frequently clouded by unconsciousness. Spontaneous recovery, if it is to occur, usually will do so in a few days. Significant residual is the rule.

Another mechanism of optic nerve and chiasm involvement in trauma is the compression of the nerve and chiasm against the sphenoid bone by the posterior orbital convolutions. The gyri recti may actually shift into the anterior chiasmal angle. Resulting visual loss with central scotomas or bitemporal field defects may not be recognized by virtue of altered consciousness. This syndrome is frequently associated with diabetes insipidus.

Cortical blindness results basically from two mechanisms—direct injury to the occipital cortex and infarction secondary to vascular compromise by the transtentorial herniation syndrome. Cortical blindness is a frequent result of a direct blow to the occiput. The resulting blindness is seldom permanent and usually resolves in minutes, hours, or days. Although recovery is often complete, residual visual field defects may occur. When they do, they are homonymous and frequently consist of congruous paracentral scotomas.

While compression of the third nerve is well recognized in transtentorial herniation of the medial temporal lobe with asymmetric supratentorial traumatic mass effects, compression or “pinching” of the posterior cerebral arteries with infarction of calcarine cortex is not. The resultant homonymous hemianopsia or cortical blindness is infrequently recognized because, again, there is usually coma. If the homonymous defect is recognized, it is of lateralizing value as it almost always occurs on the side of the mass effect. Most commonly, they are first recognized if and when the patient recovers.

The funduscopic examination in patients with severe head injuries may reveal pathology of diagnostic value. Several factors enhance the examiner’s opportunity to obtain a satisfactory and relatively complete evaluation of the patient’s fundus. Pupillary dilation may make the difference between an adequate exam and complete failure to visualize the fundus. Obviously, the decision to dilate the pupil of a head-injured patient must not be taken lightly and will depend upon a variety of factors. With the utilization of modern methods of evaluation and intracranial monitoring, an appropriate dilated examination is more feasible. It remains imperative to note on the patient’s bed and chart what has been done and when. Another invaluable factor is adequate illumination—a bright light.

Papilledema, previously felt to be relatively uncommon in closed head injury, may occur far more frequently than suspected in a mild form lasting several days. Occasionally, hidden globe perforation may occur, resulting in hypotony—low intraocular tension. This may produce disc edema. The syndrome will be unilateral.

The appearance of preretinal hemorrhages in the fundus of a head-injured adult usually reflects a severe head injury with significant subarachnoid blood. The presence of a hemorrhagic retinopathy with prominent preretinal hemorrhages in an infant suggests several possible causes, the most prominent of which is the presence of subdural hematomas. It has been estimated that one-half of infants with subdural hematomas have intraocular hemorrhages. Their significance is that they are relatively unrelated to the severity of the infant’s presenting symptoms. The child may appear only mildly ill yet harbor subdurs. The association of subdurs with the battered baby syndrome gives the fundus picture of preretinal hemorrhages added significance. The severity of the preretinal hemorrhages is highly variable; severe hemorrhage is common, however, and on occasion may fill the vitreous. Their presence is generally bilateral. The prognosis is also variable. In extreme cases, fibrosis may occur with traction and retinal detachment. They most frequently clear without significant residual.

Commotio retinae or retinal contusion results from severe direct blows to the globe itself. Abrupt rises in intraocular pressure occur, with distortion and shearing forces affecting intraocular structures. The resulting contusion necrosis with cloudy gray swelling, usually affecting the maculae and posterior pole, generally culminates in severe permanent visual impairment.

Many patients with severe head injuries suffer additional trauma to other parts of the body. Specific types of injuries result in characteristic retinal pictures.

In compression or crushing chest injuries, the resulting increased intravascular pressure is transmitted to the eye producing a specific fundus picture—Purtscher’s retinopathy or traumatic retinal
angiopathy. This consists of multiple superficial cotton-wool spots, of one disc diameter or less in size, generally located in the posterior pole between arterioles and veins. Associated retinal and preretinal hemorrhages are the rule. Initial visual impairment is variable but usually significant. The fundus picture may be delayed two-to-three days. The course is usually one of progressive return to normal over weeks to several months.

The occurrence of retinal fat emboli in fractures of long bones is frequently overlooked since the patients are either comatose or have no visual symptoms. The retinal picture consists of several small cotton-wool exudates, usually but not invariably, fringed by hemorrhage as well as additional retinal hemorrhages which usually occur in the posterior pole but seldom affect vision. The presence of the better known aspects of fat embolization should facilitate their recognition.

In closing, a single classic and nearly diagnostic external neuro-ophthalmic sign in head injury might be mentioned. This is the so-called "panda sign" of bilateral orbital ecchymosis restricted by the palpebral fascia to the orbital margin resulting in its nearly circular configuration. Its characteristic purplish color and commonly delayed appearance of two-to-three days are strong evidence of an anterior cranial fossa fracture.