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Ocular Effects of Whiplash

James Jay Brown*

COMPENSABLE SYMPTOMS following a whiplash injury (trauma) usually include¹ (1) headache, ordinarily located in the occipital region (posterior or back portion of the brain), (2) pain in the back of the neck, which may gradually radiate up into the back of the head and down into the shoulders,² (3) pain in the upper portion of the chest at the costosternal junction, (4) limited neck motion, and (5) varying degrees of paresthesia (unpleasant, morbid sensations) in the neck, shoulders, and upper back.

The absence of ocular symptoms raises a doubt whether my hypothetical client would have a cause of action. She is a secretary, aged 31, married, and mother of four. Her whiplash trauma was inflicted in the classic rear end auto collision, while she was negotiating a left turn. The force of the impact shook her up and threw her hair headband into the back seat. But she received no lacerations or contusions from striking any object within the car. Upon recovering her senses, she inspected the slight damage, procured the other driver's name, address, and license number, and then continued on to her office, where she worked a full day as though nothing had happened. That evening, prior to retiring, she complained of a stiffness in her neck and a mild headache. However, in the morning her neck was violently painful and movement was so severely restricted that she required help to get out of bed. And she had difficulty in seeing. The usual medical examinations followed, in which the objective symptom of a dilated right pupil was diagnosed as a partial internal ophthalmoplegia, which caused the patient a loss of six diopters of accommodation. Prior to the trauma, she had perfect vision, and at the time of the trial, eighteen months thereafter, her ocular condition had not improved.

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¹ Savastano & Larkin, *Whiplash Injuries*, 40 R. I. Med. J. 220 (1957).

² Billig, *Traumatic Neck, Head, and Eye Syndrome*, 20 J. Int. Coll. Surg. 558 (1953).

From the clearly objective quality of the damage, the proving of a cause and effect relation between the whiplash trauma and the dilated pupil might not be difficult. But if the symptom had been merely blurred vision, without a clearly objective damage, would she have a cause of action for eye injury?

The purpose of this article is to show how one may establish a cause of action for eye damages caused by whiplash trauma by presenting current medical literature which proves a cause and effect relation. Considering the vast complexity of the ocular mechanism, and its connection with the cervical spine region, a neuro-muscular-skeletal explanation of these areas will precede the medical documentation. Court decisions supporting petitions for this type of eye damage are unavailable. However, in order to show that whiplash trauma has, in the past, included some ocular disturbances, a few cases will follow the documentation in support of this thesis.

Medical Terminology

The whiplash classification of trauma was originally identified, and this before it appeared as a consequence of vehicle collisions, as a neck sprain resulting from a hurling back injury, observable in those performing heavy manual labor.³ Newly observed fields of neck sprain were reported⁴ before the term "whiplash" was first used by Gay and Abbott.⁵ This terminology was picked up rapidly as automobile whiplash injuries were observed to produce complex sequelae.⁶ Although the term is utilized in law, the medical profession has not widely adopted it, substitut-

³ Lonnerblad, (Upsala), Disruption Fracture of Spinal Processes as a Trade Injury, 74 Acta Chirurg Scand. 434 (1934).

⁴ McKeller-Hall, Clay Shovellers Fracture, 22 J. Bone & Joint Surg. 63 (1940) fractures in clay shovelling workmen identified as neck sprain; McMurray: Comments on Eastwood, Discussion on Fractures and Dislocations of the Cervical Vertebrae, 33 Proc. Roy. Soc. Med. 651 (1940) torticollis (a contracted state of the cervical muscles with torsion of the neck) following physical effort was held to be based entirely upon muscular conditions and not articular displacements; Jackson, R., The Cervical Syndrome, 35 Dallas Med. J. 139 (1949) describes the symptoms and signs constituting a cervical lesion.

⁵ Gay & Abbott, Common Whiplash Injuries of the Neck, 152 J. A. M. A. 1698 (1953).

⁶ Meyers, Degeneration of Cervical Intervertebral Discs Following Whiplash Injuries, 14 Bull. Hosp. Joint Dis. 74 (1953); his conclusion was that most causes of cervical syndrome were sequelae to whiplash injuries.

ing instead whip-snap, craniocervical syndrome,⁷ and cervical spine syndrome.

The mechanism of a whiplash trauma is a violent forward-backward movement or flexion-hyperextension of the victim's upper trunk, neck, and head from a previous position of rest.⁸ The trauma may center in the cervical (neck) or thoracic area of the spine.⁹ For the purposes of this study, only the first three of the total seven cervical vertebrae will be considered. The first vertebra is the atlas, the second the axis,¹⁰ and together they comprise the atlanto-axial joint. Although the first and second cervical vertebrae are structurally different from one another,¹¹ relatively uniform vertebrae begin at the level of the third cervical.¹² The connection of the cervical vertebrae is effected by means of the articular processes (the junction of two or more bones), by discs of fibrocartilage placed between the vertebral bodies, and by strong ligaments known as anterior and posterior longitudinal ligaments.¹³

The skeletal structure holds in fine balance the musculature of the neck. The atlas permits flexion and extension while the axis permits rotation of the atlanto-axial joint. The medulla and the cervical cord lie in this vertebral column of the atlas, the axis and the five adjoining cervical vertebrae. The joints of the cervical vertebrae are of the diarthrodial variety. They allow simple gliding motions. There is a joint cavity surrounded by a distinct articular capsule, lined with synovial strata.¹⁴

⁷ Seletz, Whiplash Injuries, 168 J. A. M. A. 1750 (1956); See Seletz, Cranio-cerebral Injuries and Post-Concussion Syndrome, 27 J. Int. Coll. Surg. 46-53 (1957).

⁸ Medical Tribune, Auto Safety Series #8 (9/11/61); Lipow, Whiplash Injuries, 48 So. Med. J. 1304 (1955); the writer groups violent slaps on the back, clipping and body blocking in football, and falling down steps or escalators, with rear end auto collisions as sources of the whip snap.

⁹ Hamilton, et al., Textbook of Human Anatomy 63 (1956), Figure 37 shows a lateral view of the vertebral column and identifies the spinal divisions.

¹⁰ Kimber & Gray, Text Book of Anatomy and Physiology 51-53 (7th ed., 1927); Jackson, R., The Cervical Syndrome 11 (2d ed., 1958) Diagram 5 explains the atlanto-axial articulations.

¹¹ Hamilton, *op. cit. supra* n. 9 at 64-65, Figures 40 & 41 show the differences between the two.

¹² *Id.* at 64, Figure 39 shows the typical cervical vertebra.

¹³ Kimber & Gray, *op. cit. supra* n. 10 at 54; See Hamilton, *op. cit. supra* n. 9 at 69; Figure 49 shows a lateral view of the articulations between the skull and vertebrae.

¹⁴ Lipow, *op. cit. supra* n. 8.

One function of the spinal column is to channel the spinal cord, with its connective nerve fibers and vertebral arteries, to various parts of the body. The spinal nerves leave the column at different points, which forms the basis for their identification by segments. In the cervical region, there are eight nerve roots (C-1 to C-8), there being one more nerve than cervical vertebrae.¹⁵ The conduits or canals (foramina) for these fibers and arteries are contained within every vertebra except the atlas and axis.¹⁶

Before proceeding into a basic exposition of the physiology of the eye and its neurological bodies, certain terms should be understood.¹⁷

Accommodation: the changes in the ciliary muscle and the lens in bringing light rays from various distances to focus upon the retina.

Accommodation—Convergence Reaction: consists of convergence of the eyes, contraction of the ciliary muscle, and constriction of the pupils of both eyes to provide clear vision for near objects. The eye movements¹⁸ are dependent upon the synchronized action of the internal rectus muscles.

Afferent Pathway: carrying impulses toward the nervous system.

Autonomic Nervous System: the major involuntary control and regulation of automatic visceral processes, *i.e.*, heart, blood vessels, respiratory muscles, secretory glands, pupils. Comprised of:

Sympathetic and Parasympathetic Nervous Systems: Interconnected with all involuntary organs, function in opposition to one another.¹⁹

Basilar Artery: formed by the junction of the two vertebral arteries.

Cerebrospinal Nervous System (Central Nervous System): includes sensory nerves of peripheral sense organs and muscles,

¹⁵ Hamilton, *op. cit. supra* n. 9 at 711; the vertebrae and nerve roots do not correspond because of the differential growth of the spinal cord. At the level of C-1, C-2, and C-3, the nerves leave the spinal cord at right angles. Figure 584 on this page demonstrates this.

¹⁶ Jackson, *op. cit. supra* n. 10 at 16. See Figure 10 that page.

¹⁷ Definitions taken from Blakiston's New Gould Medical Dictionary (1st ed., 1951) unless otherwise noted.

¹⁸ Walsh, *Clinical Neuro-Ophthalmology* 149-150 (2d ed., 1957).

¹⁹ Krech & Crutchfield, *Elements of Psychology* 334-337 (1959); See Hamilton, *op. cit. supra* n. 9 at 906, Figure 736.

brain, motor nerves, and spinal column. Its functions to bring perception to the brain, integrate this information, and control conscious action in response.²⁰

Cranial Nerves: twelve pairs of nerves coming off the under-surface of the brain, each serving a specific function in the reception of special senses. Of particular interest are the *II, the Optic Nerve*, controlling the movement of the retinal elements in response to light; *III, the Oculomotor Nerve*, *IV, the Trochlear Nerve*, and *VI, the Abducens Nerve*, all of which concern extraocular movements.²¹

Efferent Pathway: carrying away, conveying impulses away from the nervous system.

Horner's Syndrome: an interruption of the sympathetic pathway resulting in constricted pupil, enophthalmos (deep-seating of the eyes) and ptosis (paralytic drooping of the upper eyelid).²²

Occipital Nerve: in relation with the occiput (the back part of the head). When the posterior ramus (branch of the nerve) of the second cervical nerve root reaches the scalp, it is recognized as the greater occipital nerve.²³

Oculomotor Muscles²⁴ or Ocular Muscles: pertaining to the movement of the eye. Extrinsic eye orbit muscles: superior and inferior recti (control vertical movements), Superior and Inferior Oblique (control diagonal and circular movements), External and Internal Recti (control horizontal movements).

Ophthalmic: pertaining to the eye.

Pupil: the aperture in the iris of the eye for the passage of light.

Sequelae: an abnormal condition following a disease upon which it is directly or indirectly dependent.

Sympathetic Pathway: Postganglionic sympathetic fibers, associated with the long ciliary nerves and branches of the naso-

²⁰ Krech & Crutchfield, *id.* at 326.

²¹ Shannon, *Physiology of the Brain and Related Trauma*, 11 Cleve-Mar. L. R. 521 (1962); Ranson & Clark, *Anatomy of the Nervous System* 248-249 (9th ed., 1953); See Hamilton, *op. cit. supra* n. 9 at 836-839; the Trochlear nerve (IV) supplies the superior oblique muscle, the Abducens nerve (VI) supplies the lateral rectus muscle, and the Oculomotor nerve (III) supplies the superior, inferior, and medial recti, inferior oblique, and the levator palpebrae superioris. Figures 688 and 689 help to explain these functions.

²² Hamilton, *id.* at 930.

²³ McNeal, *Defending the Plaintiff's Headaches*, 10 *Defense L. J.* 69 (1961).

²⁴ Cross, *Neuromuscular Aspects*, 65 *Tr. Ophth. Soc. U. K.* 21 (1945).

ciliary nerves, connect with the dilator pupillae muscles of the iris.²⁵

Syndrome: a group of symptoms and signs, which, when considered together, characterize a disease or lesion.

Vergences: term applied to the associated disjunctive movements of the eyes: as convergence, divergence.

The pupil and iris of the eye function as a unit to produce a sharp focus by eliminating light stimuli. Active in this task are the iris muscles, consisting of the sphincter pupillae, which closes the iris, and dilatator pupillae, which opens it. The constrictor muscle receives its impulses from the parasympathetic fibers of the third nerve; whereas the dilatator pupillae is stimulated by sympathetic fibers. These pupillae counteract one another under bright or dark stimulus.

Light impulses are received by the rods and cones in the retina and carried toward the central nervous system by the afferent pathway through pupillomotor fibers which pass backward through the optic nerve. Impulses coming back to the constrictor muscle originate in the Edinger-Westphal center and are carried on preganglionic fibers passing through a branch of the third nerve, which, also, goes to the inferior oblique muscle. A center for pupillary contraction is believed to exist in areas of the occipital lobes (the most posterior of the brain lobes responsible for the interpretation of visual impulses). Contraction may be induced by stimulating the occipital cortex.²⁶

The reactive mechanism controlling dilatation is not clearly understood.²⁷ However, it is believed that after the fibers leave the retina, they travel to the midbrain, and thence to the cilio-spinal center of Budge, which has been postulated to exist between the fourth and sixth thoracic vertebrae.²⁸ Middleton's writings on this area are very illuminating.

. . . In these primary ocular reflexes, the afferent path begins in the retina and ends in the Edinger-Westphal nucleus situated in the midbrain . . . The dilator afferent

²⁵ Hamilton, *op. cit. supra* n. 9 at 930.

²⁶ Walsh, *op. cit. supra* n. 18 at 148. See also Nathan & Turner, *Article, 65 Brain (Eng.)* 343 (1942); the authors suggest a second route the efferent pathway of pupillary contraction might follow. Under this theory, if the primary route were severed, the pupil, although failing to contract to light, would contract to accommodation.

²⁷ Walsh, *op. cit. supra* n. 18.

²⁸ *Id.*, Figure 18 diagrams this connection.

pathway begins in the retina and travels through the optic nerve with a partial decussation (the point of crossing, or where one part is athwart another and similar part) in the chiasm, through some extra pyramidal tract to the midbrain, pons, medulla, and spinal cord, to the lateral horns from vertebrae, C-8 to T-4, the so-called ciliospinal center of Budge . . .

The efferent path of the dilator fibers consists of pre-ganglionic fibers, anterior roots, white rami, and cervical sympathetic chain of inferior, middle, and superior cervical ganglia. The final postganglionic fibers are transmitted to the eyes via the carotid plexus, and from this plexus, three divisions reach the palpebral muscle of the upper lid, the orbital muscle of the lower lid, and the dilators of the pupil.²⁹ (Parentheses added.)

It is clear, then, that the autonomic system's pathways do become extra-cranial or leave the brain region to connect with nerve areas outside the brain and many paths will pass to or through the cervical region. If the nerve fibers were primarily intracranial, like the pupillary constrictors, they would be relatively protected. But where they traverse the cervical area, like the dilator pathway traveling to the ciliospinal center, they become directly susceptible to a whip-snap trauma.³⁰

This relative exposure of the extra-cranial nerve areas has been recognized as a contributing factor in traumatically caused ocular palsies. Holmes³¹ recorded three ocular palsies arising from trauma to those ocular nerves exposed at the base of the brain.³² He found nuclear palsies involving the nerve nucleus, stem palsies from an interruption of the motor fibers, and root palsies where the extra-medullary portions of the nerves were affected. Horwich and Kasner wrote that because the brain, medulla, and cord were "floating suspended in the bony compartment of the skull and spine" the whip-snap motion "causes a forward and/or backward thrust" of these elements "against the bony walls," causing an impairment of the fine blood supply in these vascular structures, resulting in multiple symptoms.³³

²⁹ Middleton, *Ophthalmic Aspects of Whiplash Injuries*, 169 *Int. Rec. Med.* 19 (1956).

³⁰ *Id.*

³¹ Holmes, *Observations on Ocular Palsies*, 2 *Brit. Med. J.* 1165 (1931).

³² Hamilton, *op. cit. supra* n. 9 at 729; Figure 604 is a basal view of the brain showing the first cervical and oculomotor nerves and the optic tract.

³³ Horwich & Kasner, *The Effect of Whiplash Injuries on Ocular Functions*, 55 *So. Med. J.* 70 (1962).

Cause of Action

Primary Medical Support

In the past, any symptom for which no organic cause could be found was labeled psychoneurotic.³⁴ This included such subjective complaints as vertigo, ataxia (failure of muscular coordination), diplopia (seeing single objects as double with either one or both eyes), hemicrania with nausea and vomiting (having a headache on only one side of the head), or disturbances of speech and swallowing. Many whiplash victims exhibit similar subjective symptoms, *i.e.*, blurred vision, or inability to hold objects in focus.³⁵ Until recently no objective causes could be medically established to compensate for these injuries. Yet, injuries they are. Major medical proof identifying a cause for them has now been published. They spell out ocular disturbances as sequelae of the whiplash trauma.

Horwich's³⁶ findings are that damage from the whiplash trauma caused a shearing effect on the branches of the basilar artery supplying the oculomotor nucleus.³⁷ When such a shearing occurs, the tiny vessels supplying the area of the third nerve nucleus are interfered with, causing disorders in accommodation, convergence, and the pupillary mechanism. He found, in patient interviews, few, if any, objective abnormalities, although he did note pupillary disturbances and intraocular hemorrhages.

A visualization of the organic involvement of the whiplash injury with the neck in hyperextension

. . . is a momentary posterior subluxation (incomplete or partial dislocation) of the various joints with fleeting narrowing of the foramina, so that the nerve root is caught in a pincers between the superior and inferior facets.³⁸ (Parentheses added.)

³⁴ 6 *Current Med. for Attorneys* 37 (5/59); Gay & Abbott, *op. cit. supra* n. 5 at 1699; the authors attempt to separate the symptoms of a concussion syndrome occurring in a sudden flexion and extension of the head and neck from psychoneurotic symptoms; Gipner, *Ocular Changes in Head Injuries*, 53 *N. Y. St. J. Med.* 2481 (1953); in a study of head traumas, loss of convergence and esophoria (inward tendency of the visual lines) were concluded to be mild minor injuries attributed to excess nervousness.

³⁵ Billig, *op. cit. supra* n. 2.

³⁶ 2 *Am. Jur., Proof of Facts, Back Injuries* 27 (1962).

³⁷ Walsh, *op. cit. supra* n. 18 at 85; Figure 55 diagrams the path of the IIIrd nerve from the mid-brain to the orbit showing the basilar artery and oculomotor nerve; *Id.* at 79; Figure 49 is a diagram of the oculomotor nucleus.

³⁸ Seletz, *op. cit. supra* n. 7 at 1752.

The trauma adversely affects the neuro-skeletal cervical region.

Other nerves involved are the spinal accessory, the sympathetic trunk, and the vertebral. The vertebral nerve lies within the transverse foramina and travels alongside the vertebral artery. The vertebral nerve originates from the stellate ganglion and supplies the vertebral and basilar vessels. Injury to this nerve produces spasms of the vertebral arteries and gives rise to disturbed circulation to the pons and portions of the medulla containing the nuclei of origin of the lower seven cranial nerves, with resulting far-flung symptoms.³⁹

So little slack exists in the vertebral artery during severe hyperextension and hyperflexion, and especially during extreme lateral rotation, that it may become partially to completely obstructed. Such an obstruction will impair the flow of blood and oxygen to the brain stem.⁴⁰

When thirty patients, a majority having suffered whiplash, were observed, ocular disturbances with a loss of convergence and some accommodation were found in all of them. The researcher concluded the cause to be a sprain of the ligaments and capsular structures of the cervical spine, especially the sympathetic nervous system because of its vulnerable cervical location.⁴¹

In patients complaining of pain over the eyes, having radiated to that area from the occipital region, vertigo, and double vision, a study pinpointed the organic cause as a lesion (fracture) of the first cervical vertebra and/or its partial rotation off of the occipital condyles (the rounded eminence of the articular end of a bone.)⁴² It appears from previously cited material that the sympathetic pathway would be seriously affected in this case.

Another study of dilated and fixed pupils identified probable cause as pressure on the third nerve center in the midbrain.⁴³

In the strongest article on this subject, Horwich and Kasner⁴⁴ maintain that the manifestation of the whiplash trauma is an impairment of convergence and accommodation. Symptoms of this will be inability to focus at near and/or far distant objects,

³⁹ *Ibid.*

⁴⁰ *Id.*, at 1753; see a diagrammatical explanation on the same page.

⁴¹ Knight, *Orthoptic Variations*, 9 *Amer. Orthoptic J.* 62 (1959).

⁴² Abel, *Occult Fractures of the Cervical Spine*, 8 *Clinical Med.* #4 (1962).

⁴³ Cross, *op. cit. supra* n. 24 at 20.

⁴⁴ Horwich & Kasner, *op. cit. supra* n. 33 at 69.

diplopia, spots before the eyes, aching in the eyeballs, inequality of the pupils, and asthenopia (weakness and speedy tiring of the visual organs). Pain behind the eyes, noted frequently in other studies reported herein, was another symptom. The doctors, measuring the accommodation of their whiplash patients, found a frequent depression of the power below the mean and often below the minimum.⁴⁵ They believe, as McIntire did, that this pain emanated from the second cervical nerve group via the great occipital nerve.⁴⁶

The above studies establish a direct cause and effect relation between whiplash and internal eye damage. In support of this thesis, secondary material, which provides the medical pieces for the previous conclusions, illuminates the intricacies of this medical problem. The material consists of independent studies conducted on the eye, internal brain injury, or cervical spine, which do not propose the major thesis, but are included as significant contributions to it.

Secondary Medical Support

The organic structure of the nerves within the cervical spine has been recognized by many as being susceptible to great stresses and strains. Of all the nerves in this region, the second cervical, the anterior and posterior primary ramus (branch) of C-2, is the most susceptible.⁴⁷ This is true because it lacks the relative bony canal (foramina) protection found elsewhere, *i.e.*, at the C-1 and C-3 levels.⁴⁸

The second cervical nerve, as it leaves the dura (or dura mater: the outermost membrane of the brain and spinal cord) passes laterally for a short distance of approximately $\frac{1}{4}$ of an inch. Here it rests on the midportion of the medial margin of the atlanto-axial articulation. It then follows the margins of this joint laterally and slightly downward. It lies beneath the posterior arch of the atlas until it turns posteriorly within the upper neck muscles. Its close proximity to the lateral joint and the posterior arch make it potentially

⁴⁵ *Ibid.* See Duane, Subnormal Accommodation, 54 Arch. Opth. 568 (1925).

⁴⁶ *Id.* at 70; McIntire, Whiplash Injuries, A Panel Discussion, 28 J. Int. Coll. Surg. 54 (1957).

⁴⁷ Hunter & Mayfield, Role of Upper Cervical Roots in Pain of the Head, 78 Am. J. Surg. 743 (1949).

⁴⁸ *Id.* at 743-744; which presents a diagram of the lack of foraminal protection for C-2.

vulnerable to irritation or compression.⁴⁹ (Parentheses added.)

The cervical nerves would be greatly compressed when the neck is in hyperextension because the nerves would "be drawn backward toward the superior margin of the articular process of the vertebra below."⁵⁰ Such compression or irritation may cause pain and sensory motor disturbances anywhere along the nerve pathway.⁵¹ Dr. Jackson believes that this compression occurs most frequently at the third cervical vertebra, at which point the arteries are most vulnerable. Whereas, Lewis and Coburn identified the second cervical level as the situs of irritation because it is the point of greatest rotation of the head on the neck.⁵² They attributed the symptoms of dizziness, nausea, transient diplopia, vertigo, ataxia (failure of muscular coordination), and headache to constriction or occlusion at this level.

On the subject of sympathetic pathways, Middleton found that an interruption or dysfunction in it, following a whiplash trauma, produced Horner's Syndrome or sympathetic ophthalmoplegia.⁵³ If the sympathetic paralysis occurred, a loss of accommodation was the consequence.

. . . sympathetic stimulation flattens the lens of the eye, anteroposteriorly, and accommodates the eye for distant objects. This is associated with dilatation of the pupils. Dilated pupils and a flattened lens are part and parcel of the same stimulation. In contrast, the parasympathetic system helps to focus closer objects. The effect of sympathetic paralysis, while usually not sufficient to make the patient obviously myopic, is nonetheless apparent with refraction. Changes in accommodation that are not completely subjective can thus be explained on the basis of sympathetic irritation or interruption.⁵⁴

A related ocular complication, observed by Jackson in 65% of those patients suffering cervical nerve root injuries,⁵⁵ was the feeling that the eyeball was being pulled into the skull. She

⁴⁹ Jackson, *op. cit. supra* n. 10 at 27, including accompanying Fig. 16.

⁵⁰ *Id.* at 51.

⁵¹ *Id.* at 30.

⁵² Lewis & Coburn, *The Vertebral Artery: Its Role in Upper Cervical and Head Pain*, 53 *Missouri Med.* 1063 (1956); this article demonstrates, through x-rays, the impairment of the vertebral artery.

⁵³ Middleton, *op. cit. supra* n. 29.

⁵⁴ *Id.* at 20.

⁵⁵ Jackson, *op. cit. supra* n. 10 at 82.

attributed this to reflex stimulation of the sympathetic nerve pathway leading to the orbital muscle.

Ocular motor nerves are subject to possible pressure interference because of their close contact with other internal arteries.

Another cause of ocular palsies which is usually overlooked, though in my opinion it is relatively common, is compression of one of the nerves by an arterio-sclerotic vessel (an artery which has hardened). All the three ocular motor nerves lie in close contact with large arteries in the base of the brain; the sixth bends sharply round the middle cerebellar, and it lies so close to the basilar artery in its forward course that it may be compressed when this vessel is thick and tortuous; the fourth nerve crosses the posterior cerebral, and the third lies in the narrow space between the superior cerebellar and the posterior cerebral arteries as the latter leave the basilar . . . It might be therefore expected that when any of these arteries are dilated or tortuous, they might press on one of the nerves sufficiently to interfere with the conduction of impulses through it.⁵⁶

This study, implying the existence of the patient's ocular symptoms, develops an organic causation involving the important III, IV, and VI cranial nerves.

As noted in the Primary Medical subsection, the tiny vessels supplying the area of the third nerve nucleus were directly involved in disorders of accommodation, convergence, etc. In 1945, ruptures of the small nutrient vessels in the optic nerve were identified as a cause of damage to the optic nerve, where the direct head trauma did not produce a tearing of the nerve fibers.⁵⁷ Obviously, the importance of the small vessels, both nutrient and nerve, cannot be disregarded.

On the subject of the IIIrd cranial nerve, previous writing has established a connection between it, the iris muscles and the cervical spine. Palsy to this nerve causes internal ophthalmoplegia (paralysis of the iris and ciliary apparatus).⁵⁸ The trauma could be from a nuclear lesion resulting from damage to the brainstem, a tearing or bruising of the nerve trunk, a compression at the base of the skull or near the sphenoid fissure or within

⁵⁶ Holmes, *op. cit. supra* n. 31 at 1166; See Walsh, *op. cit. supra* n. 18 at 85; Fig. 55B demonstrates internal pressure on the IIIrd nerve.

⁵⁷ Symonds, Discussion on the Ocular Sequelae of Head Injuries; Visuo-Sensory Aspects, 65 *Trans. Ophth. Soc. U. K.* 3 (1945); See Turner, Article, 66 *Brain (Eng.)* 140 (1943).

⁵⁸ W. Stewart Duke-Elder, 4 *Textbook of Ophthalmology* 3749 (1949).

the orbit by hemorrhage, or a lesion of the ciliary ganglion, any of which is sufficient to cause palsy. Gipner also connected head trauma and ocular injury with the IIIrd nerve.⁵⁹ Walsh attributed the condition of a dilated fixed pupil which fails to react to light to interruption of the afferent pupillometer pathways which results in paralysis of the third nerve in the midbrain.⁶⁰ The trauma, which occurs internally to the third nerve when the head is subject to violent and abrupt movements, was suggested in 1957.⁶¹ In a whiplash-type motion, where the forward thrust of the head is abruptly stopped, the ventral surface of the mid-brain will experience trauma in that the lower portions of the nuclei of the IIIrd and IVth nerves are injured along with the IIIrd nerve fibers, which exit from the pons.

Although a major portion of this subsection has dealt with the organic basis for the internal eye damage, symptoms exhibited in whiplash victims form an important part of the entire thesis. One very common and purely subjective symptom is the headache, which, beginning in the suboccipital area (the continuation of the second cervical nerve group), radiates to the center of the skull and settles behind the eyes. This pain has been connected with damage to the second and third cervical nerves.⁶² Linked with the headache producing whiplash trauma are ocular difficulties.

. . . The most common ocular symptoms are loss of accommodation and very weak convergence . . . This is understandable, since injury to the second and third cervical nerves involves the cervical plexus (opposite the four upper vertebrae), which in turn affects, most initially, the trigeminal branches (these branches inter-connect with ophthalmic functions) . . .

. . . Loss of accommodation is one of the most difficult to correct. In some cases the intervertebral spaces are so narrow that there is constant impingement upon the cervical nerves.⁶³ (Parentheses added.)

Wiesinger and Guerry studied ten whiplash cases involving ocular sequelae. They found seven patients with partial or complete

⁵⁹ Gipner, *op. cit. supra* n. 34 at 2482; See Holman & Scott, 84 J. A. M. A. 1329 (1925).

⁶⁰ Walsh, *op. cit. supra* n. 18 at 165.

⁶¹ Peterson, C., in Brown, H. W., Isolated Inferior Oblique Paralyses, 55 Tr. Am. Ophth. Soc. 415 (1957).

⁶² McIntire, *op. cit. supra* n. 46; these headaches have been found to last a year or more.

⁶³ *Id.* at 55.

palsy of both accommodation and convergence, and two exhibiting slight extra-ocular muscle palsies with diplopia.⁶⁴ Unilateral mydriasis (great dilatation of the pupil) as a result of sympathetic irritation or Horner's Syndrome from sympathetic palsy are recognized as other whiplash symptoms,⁶⁵ which, like other sympathetic irritations, may be diagnosed by pupillography.⁶⁶ Although many symptoms have been observed, there is a dearth of published material on how long the symptoms may exist. Horwich and Kasner's list of symptoms, which include diplopia, unequal pupils, and asthenopia, noted in the Primary Medical subsection, were observed to last from two to six weeks; rarely over two years.⁶⁷

An extraordinary case history was reported in 1952, which inadvertently brings together the pieces of this medical jigsaw puzzle.⁶⁸ It involved a 17 year old boy who developed attacks of syncope (fainting) followed by vertigo, unsteadiness, nystagmus (continuous rolling of the eyes), and diplopia, which were found to be inducible by forcing the head backward. The organic cause was identified as a defect in his odontoid process (a bony projection arising from the axis [second cervical vertebrae] which forms a pivot around which the atlas [first cervical] rotates as the head is turned) causing an excessive mobility at the second cervical level. This mobility resulted in an intermittent obstruction of the vertebral arteries. The researcher observed a definite anatomical relation between the vertebrae and arteries which could produce obstruction.

. . . In their second portion, the vertebral arteries ascend through the transverse foramina in the transverse processes of the first six cervical vertebra. As these arteries traverse the foramina in the axis they abandon their previous course which was almost vertical and pass upwards and outwards to reach the foramina in the atlas. As they emerge from these foramina they pass horizontally to the outer side and back of the atlas and enter the suboccipital triangle. They then penetrate the posterior occipito-atlantoid ligament and enter the spinal canal. Thus, it is evident that the vertebral arteries pass through a series of superimposed

⁶⁴ Wiesinger & Guerry, *The Ocular Aspects of Whiplash Injury*, 89 *Va. Med. Monthly* #3, 167 (1962).

⁶⁵ *Id.* at 165.

⁶⁶ *Id.*; Middleton, *op. cit. supra* n. 29.

⁶⁷ Horwich & Kasner, *op. cit. supra* n. 33.

⁶⁸ Ford, *Syncope, Vertigo, and Disturbances of Vision . . .*, 91 *Bull. Johns Hopkins Hosp.* 168 (1952).

bony rings which normally have a limited range of movement in relation to each other. It is obvious that excessive displacement of any of these vertebrae may cause stretching, angulation, or compression of the arteries.⁶⁹

If the obstruction occurred, he believed the patient's symptoms would appear.

. . . the basilar artery is formed by the junction of the two vertebral arteries and that the posterior cerebral arteries arise from the basilar, it is evident that the medulla, pons, cerebellum, and midbrain all derive their nutrition from blood which flows through the vertebral arteries. It seems quite plausible therefore to conclude that syncope, vertigo, nystagmus, unsteadiness, blurring of vision, and diplopia may result from transient reduction of blood flow in one or both vertebral arteries.⁷⁰

This boy underwent a fusion operation of his upper cervical spine. His symptoms disappeared in toto!

Exceptions to Thesis

In researching material for this article, medical evidence in contradiction of the major thesis was discovered. It is included in order to shed light on the relative merits and weaknesses of this thesis and for those attorneys pleading the defense.

Heavy emphasis was placed on the sympathetic nervous pathways and its vulnerability in the cervical region. However, this nervous system has its inherent weakness.

The exact anatomy and physiology of the sympathetic nervous system is not known and its pattern is, moreover, not consistent in all species. There is not even consistent anatomy in the same species, nor is there necessarily a symmetry of the sympathetic nervous chain on each side of the neck in a given individual.⁷¹

Such inconsistency renders medical rules applicable only to a particular case history. This must be applied to statements made about pupillary movements. Little is known about the precise effect of dilatating stimuli upon the sympathetic or parasympathetic nerves and final pupillary reaction.⁷²

The symptoms constituting the internal eye damage may be similar to those of other traumatic occurrences. Therefore,

⁶⁹ *Id.* at 172.

⁷⁰ *Ibid.*

⁷¹ Middleton, *op. cit. supra* n. 29 at 20.

⁷² Walsh, *op. cit. supra* n. 18 at 145-146.

identifying the organic cause becomes a problem in differential diagnosis. Basilar impression should not be mistaken for cervical nerve root irritation. Basilar impression is a congenital malformation of the craniovertebral boundary which encroaches on the bone of the adjacent neural bodies.⁷³

Another symptomatic condition which should not be confused is congenital mydriasis. This is a condition in which the pupil is large and inactive to light stimulation.⁷⁴ Direct head trauma, including laceration or contusion of the neck and head muscles or hemorrhage, often appears as blurred vision, double vision, vertigo, nervousness, and/or headache.⁷⁵ A bump on the head from an overhanging door could develop into a chronic subdural hematoma (a tumor containing effused blood located below the dura). The symptoms from such a trauma are progressive headaches, drowsiness, convulsive seizures, motor weakness, and failing or double vision.⁷⁶ Jackson suggested that reflex sympathetic dystrophy⁷⁷ could produce similar symptoms. This condition occurs where the pain of sympathetic nerve root irritation, left unrelieved, becomes a self-perpetuating pain stimulus.

Final mention must be made of that group of patients, often termed malingerers, who, while involved in litigation, have post-traumatic complaints. Although apparently in great misery, no structural changes in the brain can be found to substantiate their pains. These symptoms often disappear following the termination of the litigation.⁷⁸

Cases

The reported instances of eye injury in a whiplash case are few indeed. Whatever the reason, whether settlement or the psychoneurotic classification of ocular symptoms, only a very small number include ocular complaints. The legal reports below are included to demonstrate that prior whiplash litigation did have the necessary elements, *i.e.*, upper cervical spine involvement, subjective symptoms like headache, and internal eye dis-

⁷³ Jackson, *op. cit. supra* n. 10 at 125.

⁷⁴ Walsh, *op. cit. supra* n. 18 at 146.

⁷⁵ McNeal, *op. cit. supra* n. 23 at 74; the author suggests glaucoma as another possible cause of the same ocular disturbances.

⁷⁶ Shannon, *op. cit. supra* n. 21 at 528-529.

⁷⁷ Jackson, *op. cit. supra* n. 10 at 52; See Evans, Reflex Sympathetic Dystrophy, 26 *Ann. Int. Med.* 417-426 (1947).

⁷⁸ Shannon, *op. cit. supra* n. 21 at 531.

turbance (inability to focus, dilated pupil) for making out a valid claim for internal eye damage.

*Lucas v. State Farm Auto Insurance Co.*⁷⁹ involved a terrific rear end collision in which the 18 year old female plaintiff was thrown forward in a whiplash fashion, causing her to feel a neck snap and to strike her head against the rear-view mirror. This direct contact inflicted two scalp cuts above her hair line, which had to be closed by sutures. Two days later, the plaintiff, complaining of headaches, dizziness, and pain in the lower back and neck, consulted a physician. He found a flattening of her cervical spine (loss of the normal curve), muscle spasm, and double vision. At the trial, conflicting medical testimony was presented on the objective cause of her nervousness and anxiety. On appeal, the previous award of \$8,000 was reduced to \$4,300.

The woman plaintiff in *Apling v. Cascade Oil Co.*⁸⁰ was involved in a rear end collision in which she sustained a sprain of the cervical and lumbar spine. The injuries to the muscles and ligaments of her upper and lower back caused her to suffer headaches, pain in her back and breast, visual disturbances, dizziness, leg twitching, and intermittent vomiting. She was hospitalized four times within 90 days in order to alleviate these miseries. Prior to the trial, a psychiatrist found her suffering from a traumatic neurosis attributed to the accident. One year after her accident, the trial was held and her objective symptoms had been reduced to a minimum. On remittitur the Court awarded her \$10,000 general damages and \$2,600 special damages because the discomforts were real to her and serious during that year.

Field Packing Co. v. Denham:⁸¹ an auto-truck rear-end collision; whiplash injury to a 45-year old mother who was employed at a rural feed mill. Her subjective symptoms included neck pain, intermittent blurring of vision when in a bent over position, and near blackout upon straightening up. Under examination by a neurologist, no objective signs of neurological involvement or nervous disturbance from fracture or dislocation were found. The only objective symptoms exhibited were a 1/3 impairment in her hearing, and an internal hemorrhage. The \$50,000 award was reversed because of a failure to establish the permanency of the injuries, and jury prejudice.

⁷⁹ 17 Wis. 2d 568, 117 N. W. 2d 660 (1962).

⁸⁰ 374 P. 2d 575 (Wash. Sup. Ct. 1962).

⁸¹ 342 S. W. 2d 524 (Ky. App. 1961).

A cervical sprain superimposed upon an arthritic neck involving the second, third, fifth, sixth and seventh cervical vertebrae was reported in *Faulkner v. Ryder Tank Lines, Inc.*⁸² The arthritic process caused an encroachment on the neural canal and intervertebral notches. The victim complained of occipital neuralgia, stiffness and soreness in his neck, and shoulders, including his shoulder blades, and headaches in the back of his neck. After 30 months the plaintiff was not fully recovered, and received an award of \$5,000.

A typewriter serviceman, who received a whiplash trauma, complained of a heavy feeling in his eyes, impaired neck movement, headaches, deafness, and pains in his ears, head, spine, back, shoulders and neck. In light of the doubts raised about these symptoms, because the lower court did not have a stenographic record made of the trial, and for other reasons, the \$15,000 award was reduced to \$6,000.⁸³

*Agnew v. Cox*⁸⁴ involved whiplash trauma to a 38 year old housewife and mother of four, who was a passenger at the time of the accident. The court affirmed the \$20,000 award to the plaintiff for pains through her abdomen, lower back, female organs, and, also, her restricted neck movement, headaches, nervousness, and disturbed eyesight. Medical history showed that she had a pre-accident upper back deformity and low-back arthritis. Her pains were intensified by the trauma when some of the osteo-arthritic hiping on the vertebral bodies was broken off. Testimony established, also, that traumatic neurosis was, in part, responsible for the upper head stiffness, pain, and headaches. The court report included a prophetic statement (*i.e.*, 1958) on the probable cause of her disturbed eyesight.

. . . change of vision quite often is a complaint following whiplash injury to the neck; that any blow may affect the sympathetic nervous system and send the impulses into the brain and manifest evidence of pathology by some disturbance in vision, or even in hearing, and that he (the medical expert) is of the opinion that that is the part involved which has had something to do with her decrease in vision since the accident . . .⁸⁵

⁸² 135 So. 2d 494 (La. App. 1961).

⁸³ *Cermak v. Hertz Corp.*, 53 N. J. S. 455, 147 A. 2d 800 (1959).

⁸⁴ 254 F. 2d 263 (8th Cir. 1958).

⁸⁵ *Id.* at 267.