

Vision and Rehabilitation after Brain Trauma

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Vision Problems after Brain Injury

Visual problems following brain trauma are frequent and often complex. It is probably easiest to define the problems based upon how they affect incoming visual information (i.e., the afferent visual pathways) or the outflow of information to the visual organs (i.e., efferent visual pathways). Afferent defects include reduction in visual acuity, visual field, color vision, contrast sensitivity, comfort (usually as it relates to glare), and higher level visual processing, including recording of visual memory and comprehension of visual stimuli. Efferent defects include reduction of the ability to visually pursue a target, focus the lens inside the eye, train the two eyes onto a single target, maintain gaze once a visual target is obtained, and open and close the eyelids. In this three part series, we will describe 1) damage to the afferent visual pathways, 2) damage to the efferent visual pathways, and 3) the role of the neuro-ophthalmologist in visual restoration and rehabilitation.

The Afferent Visual Pathway and How Brain Injury Can Affect It

Light enters the eye through the cornea, the clear window of the eye. The cornea provides the majority of the focusing power of the eye, and is an extremely poor refractive surface, but with a healthy tear film it becomes nearly perfect. Brain injury often causes dry eye thereby reducing visual acuity. Furthermore, brain injury patients often lose an adequate blink response or develop lagophthalmos, the inability to completely close the eye. Dry, unprotected corneas are subject to scarring and infection.

Trauma to the brain often entails injury that can also shake or directly damage the eye along with the rest of the body. Patients suffering a blast injury can experience rapid elevation of pressure in the chest, which is then transmitted by the blood vessels to the retina, the neural tissue lining the inner wall of the eye. This is the tissue which converts light rays into the electrical impulses that are sent to the brain. The retinal blood vessels can rupture from the sudden increase in pressure and cause bleeding within the retina, a condition called Purtscher's retinopathy. The free blood inside the eye can cause significant scarring and loss of vision.

Direct head trauma can also cause the eye to move too quickly and/or too far relative to the fixed structures in the eye socket. This can cause stretching or shearing of the optic nerve, the nerve that carries visual information to the brain. This traumatic optic neuropathy often can result in permanent visual impairment. Multiple direct head traumas also are a risk factor for problems within the eye itself,

such as detachment of the retina from the back of the eye, or formation of a cataract, a clouding of the natural lens.

Trauma to the head invariably is associated with some degree of trauma to the neck, a risk factor for damage to the blood vessels of the neck. Injury to the wall of an artery can cause it to bulge (i.e., form an aneurysm) or separate from its inner lining (i.e., arterial dissection). Either situation can lead to abnormal blood flow to the visual pathways of the brain. Furthermore, either condition can cause the vessels to physically compress portions of the visual pathways, such as the nerves that control the eye muscles or the nerves that bring visual information to the brain. This could result in double vision or in reduction of visual acuity and visual field, respectively.

It has been demonstrated that the world we see is formed into a map upon our brain, specifically onto an area called the visual cortex. This map is organized such that the image of the world is inverted and reversed; the left side of our brain sees what is to the right of where we look and the right side of the brain sees light from the left of where we gaze. Furthermore, visual information emanating from above our visual point of interest is transmitted to the lower portion of the visual cortex while the upper portion of the visual cortex maps the visual world below the object of regard. It is for this reason that damage to the visual cortex causes loss of peripheral vision rather than simply loss of visual clarity.

While we do not know how visual memories are created or stored in our brains, it is known that brain injury slows the acquisition and processing of visual information and impairs the formation of visual memory. Recent research has suggested that these impairments may result from stretching or shearing of nerve fiber bundles after head injury. Sadly, higher level visual processing failure is often particularly difficult for a patient to express. Neuropsychologists have proven to be critically important for the diagnosis of these problems and for guidance in directing rehabilitation efforts.

The Efferent Visual Pathways and How Brain Injury Can Affect Them

The efferent visual pathways include both voluntary and involuntary (otherwise known as autonomic) functions affecting the muscles within and surrounding the eye. The iris, an internal muscle of the eye, is what we recognize as the colored part of the eye with a central opening called the pupil. The iris stands directly in front of the natural lens and by contracting or relaxing, it controls the size of the pupil and thereby the amount of light that enters the eye. Like the iris, the ciliary body is a ring-shaped muscle that is attached around the natural lens and controls the ability of the lens to focus. Both of these muscles are in turn controlled by nerves of the autonomic nervous system.

The autonomic nerves controlling the internal muscles of the eye are of two types, sympathetic and parasympathetic. The sympathetic nerves employ chemicals identical or similar to adrenalin, and take a circuitous path to get from the brain to the eye, going by way of the neck, down to the chest and back up the neck to the head. Along the way, the sympathetic pathway helps control breathing, heart rate,

blood pressure, sweating and even 2 millimeters of eyelid opening. Damage to this pathway from blunt trauma or shrapnel causes many problems including what is known as the Horner's syndrome. In this condition, the patient will present with a small pupil, a somewhat droopy eyelid and decreased sweating on the affected side of the face. In addition, the patient may often describe a sense of fatigue with distance vision because the affected eye's lens may not adjust focus normally.

The parasympathetic pathway starts in the brainstem, where the brain starts to narrow as it merges with the spinal cord. The parasympathetic nerves to the eye socket cause the pupil to become small and cause the lens to focus for near work. Head trauma can damage this pathway and a patient will have a large pupil and difficulty focusing for reading. This pathway also can be damaged by elevated pressure in the brain which is why patients who suffer head trauma and have large or unreactive pupils are treated as critical emergencies. Many patients suffering head injury often need reading glasses earlier than what might be expected because of the reduced ability to focus at near. These patients may also have more problems with glare. However, it has not been confirmed that these problems are a direct result of damage to the parasympathetic pathways in every patient.

Attached to the outside of each eye is a team of six muscles. These extra-ocular muscles allow the eyes to not only look in every direction but to rotate as well so that visual images remain upright-appearing even when the head tilts. The nerves serving these ocular muscles all originate in the brainstem. These nerves are quite delicate and even a mild concussion can damage them. When this occurs, the affected muscle cannot help the eye move as quickly or completely as it should and the result is double vision, or diplopia. Patients will report doubled images as being displaced horizontally, vertically or even diagonally. Although images can also be torsionally displaced, patients have a much harder time expressing this complaint in specific terms. For this reason, neuro-ophthalmologists, orthoptists and Behavioral Optometrists, all of whom are particularly trained to evaluate eye teaming disorders, play a key role in diagnosing and treating diplopia.

The extra-ocular muscles normally work in unison so that the two eyes fixate on the same target and smoothly pursue that target when necessary. They permit the eyes to move in the same direction such as when a patient tracks an object that is moving up, down, right or left. They also allow the eyes to move in opposite directions (i.e., to cross or uncross) during times when a patient is tracking objects that are approaching or departing. Head trauma routinely causes a reduction in the ability of the eyes to work as a team. Convergence insufficiency is a reduction in the ability of the brain to make the eyes cross for close vision and is one of the most common deficits following concussion. Deficiency of smooth pursuits is also common. In this case, the eyes have difficulty following a moving object such as a swinging pendulum; the patient reports dizziness or even nausea trying to complete this simple visual task.

Nystagmus is a repetitive movement of the eyeball during which the eye seems to drift off target and then quickly corrects to re-gaze at the target. This condition can occur after brain injury and causes

considerable reduction of visual clarity as well as a sense of dizziness. Although all the centers of the brain controlling the maintenance of fixation are not well understood, nystagmus often suggests damage to the centers of the brain associated with balance and equilibrium, specifically the cerebellum located at the back of the brain, and the vestibular system inside the ears. Even relatively mild trauma to the skull can cause damage to the delicate bony walls of the vestibular system. Patients might not have any hearing loss but still have balance problems or nystagmus. Interestingly, nystagmus from damage to the wall of the vestibular system may not be constant but rather might be initiated by a repetitive sound such as a ringing telephone or by increased air pressure in the chest such as during inflation of a balloon; this is called Tullio's phenomenon.

As mentioned previously, glare frequently occurs after traumatic brain injury, and it is unclear why this occurs. In some patients, it may be secondary to elevations in intracranial pressure. In others, it seems to accompany pain to the eyes and forehead from injury to a nerve at the back of the skull, just above the neck, called the Greater Occipital Nerve. This nerve exits from the base of the skull and runs forward to the eye socket. The pain from this nerve can be felt along its entire course and even gentle pressure on the nerve root can bring exquisite pain. Patients with this type of headache are sometimes misdiagnosed as having migraine headaches or even as being insincere because clinicians focus their attention on the forehead and eye rather than considering the Greater Occipital Nerve as the cause of this referred pain. In addition, pain from irritation of this nerve can appear months or years after the original injury and clinicians might not correlate the patient's complaint of pain with a head trauma they suffered years before.

The Role of the Neuro-Ophthalmologist in Vision Rehabilitation after Brain Injury

Vision rehabilitation after brain injury is usually initiated by the neuro-ophthalmologist. Efforts during the acute period usually involve simple but important measures such as replacing a patient's damaged spectacles, offering the patient an eye patch to alleviate diplopia, or protecting a lagophthalmic eye from exposure and drying out.

Long-term visual rehabilitation evaluations take into consideration the wealth of information often accompanying patients, such as the assessments from other ophthalmologists, physiatrists, neuropsychologists, neurologists, and neurosurgeons. After offering the patient a full eye examination and vision evaluation, the neuro-ophthalmologist can then best decide what ancillary testing and consultations might be needed. Some commonly employed testing devices include:

Automated Perimeter: This device helps to map a patient's peripheral vision and evaluate for possible damage to the visual cortex and other parts of the visual pathways.

Visual Evoked Potential Analyzer: This device measures the speed and strength of the neuro-electrical signals passing along the optic nerve to determine whether the nerve was damaged in a way often too subtle to detect via other means.

Electroretinogram: This device measures the speed and power of neuro-electric signals created by the retina during the conversion of light energy in order to determine whether there was a loss of function that might be too subtle to detect otherwise.

Synoptophore: This device helps determine whether a patient can still use the two eyes as a team to form a single 3-dimensional image.

Notably there are many variations of these devices and each can provide helpful information to pinpoint and sometimes even confirm the presence of damage.

The Neuro-ophthalmologist will always evaluate a patient for abnormal eye movements and often refer the patient to orthoptists or Behavioral optometrists, colleagues with particular training in eye-teaming disorders. He will also investigate whether a patient can be helped with low vision assistance devices such as magnifiers and glare-control measures. At times he will be required to discuss medical and surgical options for patients with uncontrollable diplopia or nystagmus. A referral to a Blind Rehabilitation Specialist will be required for those patients whose vision loss is too severe to rehabilitate or not amenable to the novel restorative surgeries currently available. . The neuro-ophthalmologist must also ensure that he coordinates his rehabilitative plans with those of occupational-, physical- and vestibular-therapists.

For many patients, the neuro-ophthalmologist represents the last step in the rehabilitation plan and the first step in the care patients will need for the remainder of their lives. This includes maintaining vigilance for the appearance of systemic disorders that often result after brain injury and which can directly or indirectly threaten vision, such as depression, weight gain, sleep apnea, migraine, diabetes, hypertension, stroke, and heart attack. Some of the eye conditions that can stem from these medical problems include stroke in the blood vessels of the eye and optic nerve, glaucoma, bleeding in the eye, swelling of the optic nerve, loss of peripheral vision, diplopia, difficulty reading, and loss of visual clarity.

Patients suffering traumatic brain injury might unfortunately experience depression, which can lead to neglect of medical problems and delay in diagnosis of associated medical conditions, including visual problems. Brain trauma can also exacerbate or accelerate underlying cranial disease. It cannot be stressed enough that the team who helped the patient return to maximal medical improvement must also ensure that continuity of care for the patient is available. The patients and their families will rely on that continuity to help them adapt to and accept their new, post-brain injury life in which there are both unfamiliar challenges and new opportunities.