CLINICAL CARE CE

The Pupillary Response in Traumatic Brain Injury: A Guide for Trauma Nurses

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ABSTRACT

Traumatic brain injuries (TBIs) affect more than 1.4 million Americans annually. Trauma nurses caring for these patients routinely perform serial neurologic assessments, including pupillary examinations. While trauma nurses are likely familiar with basic components of the pupillary examination, some confusion about more specific aspects of the examination and the physiologic basis of the pupillary response may still remain, particularly as it pertains to patients with TBI. Therefore, the purpose of this article is to identify the key components of a pupillary examination and its associated physiologic response. A case study is provided to illustrate the application of this information among patients with TBI.

KEY WORDS

Brain injury, Pupil, Trauma

Traumatic brain injury (TBI) affects more than 1.4 million Americans annually.[1] These injuries, defined as a blow or penetrating injury to the head that disrupts normal brain function,[2] occur as a result of falls (28%), motor vehicle crashes (20%), being struck by or against a moving or stationary object (19%), and assaults (11%).[3] Injuries to the brain are classified as focal, such as cerebral contusions, lacerations, and hematomas/hemorrhages, or diffuse, which include concussions and diffuse axonal injuries.[4] Because the signs and symptoms of patients who have sustained TBIs may change quickly and evidence of deterioration may be subtle, trauma nurses must be adept at performing astute neurologic assessments.

A key component of any neurologic assessment is the pupillary examination.[4] Trauma nurses at all levels of care routinely perform pupillary assessments on patients and are likely to be familiar with the basic components of the pupillary examination. However, some confusion may still remain about more specific aspects of the examination and the physiologic basis of the pupillary response, particularly as it pertains to patients who have sustained TBIs. Therefore, the purpose of this article is to identify the key components of the pupillary examination, explain the physiologic basis of the pupillary response, and illustrate the application of this information in patients with TBI through a case study.

CASE STUDY

An 18-year-old woman was a restrained driver of a vehicle that was T-boned (lateral) at a high speed on the driver's side of the car. The car subsequently rolled over onto its roof. Emergency medical services arrived to find the victim conscious but with a Glasgow Coma Scale (GCS) Score of 13 (Table 1). The victim's pupils were noted to be equal and reactive. The victim's level of consciousness continued to deteriorate while the emergency medical services worked to free her from the wreckage. She arrived at the regional emergency department after 40-minute extrication with a GCS Score of 8: she was given a 2 for opens eyes to painful stimuli, a 2 for unintelligible verbal response, and a 4 for withdraws to pain. Her pupils were now unequal. The right pupil measured 5 mm and was nonreactive to a direct light, whereas her left pupil measured 2 mm and displayed a sluggish reaction

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TABLE 1

The Glasgow Coma Scale

Eye opening	
Spontaneous	4
Response to verbal command	3
Response to pain	2
No response	1
Best verbal response	
Oriented	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
No verbal response	1
Best motor response	
Obeys commands	6
Locales to pain	5
Withdraws to pain	4
Flexion to pain	3
Extension to pain	2
No motor response	1

to direct light. She was noted to have a small forehead laceration with surrounding ecchymosis. She was sedated, paralyzed, and intubated by the emergency department staff to ensure airway protection. A computerized tomography (CT) scan of the patient's head was performed, which revealed a large subdural hematoma. The patient's neurologic status continued to deteriorate, and, therefore, the local air medical service was immediately dispatched to transport the patient to a level I trauma center. She was transported to a level I trauma center for further evaluation of her head injury. Upon her arrival to the trauma center, her trauma evaluation was completed, and she was transferred to the surgical intensive care unit. Her pupils were found nonreactive on her initial trauma evaluation at the trauma center. In the surgical intensive care unit, a neurosurgical consultation for the treatment of an acute subdural hematoma and possible surgical evacuation was obtained.

IDENTIFYING TBI

Identifying an acute brain injury in patients with trauma requires serial neurologic examinations, including pupillary assessments, a CT scan, and possibly a magnetic resonance imaging. As depicted in the case study, evidence of TBI may be present in the initial neurologic examination performed at the scene. Victims may have altered GCS scores or impaired level of consciousness ranging from subtle to severe. The initial pupillary examinations may appear benign, and pupillary changes may not surface until hours after the occurrence of the injury. Performing serial neurologic examinations, including pupillary assessments, aids in identifying subtle changes in a patient's neurologic status. These subtle changes can indicate the presence of TBI and/or an acute deterioration in a patient's condition.[4] The presence of TBI is confirmed with a CT and/or a magnetic resonance imaging once a patient arrives in a healthcare facility. The CT scan can quickly aid in differentiating between a focal and a diffuse brain injury.

Subdural hematomas are one type of focal brain injury occurring in approximately 30% of patients with TBI.[4] An acute subdural hematoma occurs usually after a deceleration injury in which cerebral veins rupture because of either a linear or rotational shearing force.[5] Rupture of these vessels causes bleeding into the subdural space. This bleeding may exert additional pressure onto the surrounding brain tissue, causing shifting of the tissue or mass effect. Because the subdural space is not limited by the cranial sutures, this bleeding can potentially spread along the entire cerebral hemisphere.[6] As the size of the bleed and the mass effect on the brain increases, the patient's neurologic status declines, and pupillary changes often appear. As bleeding continues, the likelihood of a poor prognosis also increases: mortality rates as high as 50% have been reported among the patients who present with a subdural hematoma requiring immediate surgical evacuation.[6]

THE PUPILLARY EXAMINATION

The standard of care for any patient with TBI includes serial neurologic examinations. These examinations include a pupillary assessment and are often performed by trauma nurses at the scene, in the emergency department, and in the acute and critical care units.[7] Unlike other components of the neurologic examination that require patients to be conscious, the pupillary examination is one of the few neurologic signs that can be assessed in an unconscious patient or in a patient receiving neuromuscular blocking agents and sedation. The pupillary examination is a minimally invasive assessment, which provides valuable information about the severity and progression of the brain injury, as well as brainstem function. Thus, it is important for trauma nurses to perform pupillary examinations thoroughly and understand the physiologic basis of the pupillary response.

When performing a pupillary examination in patients with TBI, a trauma nurse should make the conscious patient focus on a distant object straight ahead. In the comatose patient, pupils are assessed per the position they are found in. A pupil assessment should include the examination of size and equality of pupils, pupillary shape, and reactivity to light.

Pupil Size and Equality

Pupil size is reported as the width or diameter of each pupil in millimeters. A standardized pupil gauge should be used to report the pupil size in millimeters. The use of this gauge aids in decreasing subjectivity, particularly when serial assessments are performed. The normal diameter of the pupil is between 2 and 5 mm, with the average pupil measuring 3.5 mm. Although both pupils should be equal in size, a 1-mm discrepancy is considered a normal deviation. This condition is known as *anisocoria* and is present in 15% to 17% of the population without any known clinical significance.[6] Pupil size should be assessed both before and after the pupil responds to direct light.

Pupil Shape

Pupil shape is reported as round, irregular, or oval. The normal shape of the pupil is round. An irregular-shaped pupil may be the result of ophthalmological procedures such as cataract surgery or lens implants, and this should be noted on the initial assessment and confirmed with the patient or family. A pupil that is oval in shape may indicate the early compression of cranial nerve III due to increased intracranial pressure (ICP), and thus should be addressed immediately. If an oval pupil is detected, measures should be taken to decrease ICP. As ICP is reduced, the oval-shaped pupil should resolve. However, if ICP continues to rise or is not treated, the oval-shaped pupil will become further dilated and will eventually become nonreactive to light.

Reactivity to Light

Pupil reactivity is reported as the response or reflex of each pupil to direct light. Reactivity is assessed by shining a low-beam flashlight inward from the outer canthus of each eye. Each eye should be checked separately. The light should not shine directly into the pupil because the glare or reflection may obscure visualization.[6] The reaction that each pupil has to the light stimulus should be recorded. The speed of pupillary reactivity is recorded as brisk, sluggish, or nonreactive. Normally, pupils should constrict briskly in response to light. A sluggish or slow pupillary response may indicate increased ICP, and nonreactive pupils are often associated with severe increases in ICP and/or severe brain damage.

A complete pupillary reactivity examination also includes assessment of the consensual pupillary response and accommodation. The consensual pupillary response is the constriction that normally occurs in a pupil when light is shown into the opposite eye.[6] Because of this response, the trauma nurse should wait for several seconds before assessing pupillary light reflex in the second eye, as that pupil may be temporarily constricted. Accommodation is the constriction of pupils that occurs when a conscious patient is focusing on a close object. Pupils should normally constrict bilaterally when an object is held within 4 to 6 inches of a patient's nose.[6]

PHYSIOLOGICAL BASIS OF THE PUPILLARY RESPONSE

The pupil is a small hole in the center of the iris, which allows light to enter in the lens of the eye. The diameter of the pupil is controlled by smooth muscle within the iris; and pupillary size, shape, and reactivity to light are regulated by the autonomic nervous system.[6] The diameter of the iris is controlled by 2 muscles: the pupilloconstrictor, which is a sphincter muscle controlled by the parasympathetic nervous system; and the pupillodilator, which has sympathetic nervous system control.[8]

Parasympathetic Response: Pupillary Constriction

Normally, when light is shone into the eye, the rods and cones of the retina serve as sensory receptors and send the impulse via afferent nerve pathways through axons in the optic nerve, optic chiasm, and optic tract.[6] This impulse reaches the Edinger–Westphal nuclei in the midbrain. The parasympathetic response originates in these nuclei, and efferent nerve pathways transmit the impulse via cranial nerve III to the pupilloconstrictor muscle.[4] Both the ipsilateral and the contralateral Edinger–Westphal nuclei receive identical afferent input, which causes the consensual light reflex.[8] The neurotransmitter responsible for the synaptic response in pupillary constriction is acetylcholine.

Sympathetic Response: Pupillary Dilation

The sympathetic pathway responsible for pupil dilation originates in the posterior-lateral hypothalamus and involves 3 neurons.[4] The first ipsilateral preganglionic central neuron travels through the lateral brainstem to the spinal cord. The second preganglionic neuron crosses the lung apex and ascends the neck where it synapses in the cervical ganglion. The third postganglionic neuron accompanies the internal carotid into the skull base to the trigeminal ganglion and joins the nerve fibers of the abducens as it passes through the inferior orbital fissure. This neuron then continues through the trigeminal nerve until it reaches the pupillodilator muscle in the iris. The

TABLE 2

Abnormal Pupils Observed in Patients With TBI[a]

Pupillary Abnormality	Pupillary Response/Assessment Findings	Causes
Unequal pupils	*Mydriasis*: One pupil is dilated and nonreactive to light. Other pupil is of normal size and reacts to light.	Defect in efferent pathway Compression of cranial nerve III Uncal herniation Compression of posterior communicating artery Damage to nerve endings in sphincter muscle of iris from direct trauma
	Adie's pupil: One pupil is larger than the other. The affected (larger) pupil does not immediately respond to direct or consensual light reflex; however, if light stimulus is prolonged, this pupil will slowly constrict to light and slowly dilate in the dark. Accommodation in the affected pupil is sluggish. The other pupil is of normal size and reacts to light.	Defect in efferent pathway Loss of parasympathetic nerve supply to sphincter muscle in iris (frequently caused by viral infection) Sensitivity to cholinergic drugs
	Horner's syndrome: One pupil is smaller than the other. The affected (smaller) pupil has an impaired response to light and accommodation. There is ptosis of the eyelid on the side of the affected pupil. There is a loss of sweating on the affected side.	Defect in efferent pathway Total or partial loss of sympathetic innervation to pupil (due to lesion in sympathetic fibers in brainstem or spinal cord) Damage to hypothalamus
Constricted pupils	*Miosis*: Pupils are pinpoint bilaterally and too small to observe reaction to light.	Defect in efferent pathway Disruption in sympathetic pathway (can be due to intraocular inflammation from trauma/direct orbital injury) Irritation of parasympathetic pathways Pontine hemorrhage Drug induced: opiates/narcotics, pilocarpine, acetylcholine

(continues)

TABLE 2 *(Continued)*

Abnormal Pupils Observed in Patients With TBI[a]

Pupillary Abnormality	Pupillary Response/Assessment Findings	Causes
	Argyll–Robertson: Pupils are small and irregular in shape, and may be unequal. Pupils do not respond to light stimulus, but do constrict with accommodation. May dilate minimally in the dark.	Defect in afferent pathway Usually due to viral infection (neurosyphilis, encephalitis)
Dilated pupils	*Drug-induced mydriasis:* Pupils are dilated bilaterally. Pupils may or may not have reaction to light stimulus.	Defect in efferent pathway Sympathetic stimulants or parasympathetic blocking agents cause pupillary dilation. Caused by hallucinogens, antihistamines, amphetamines, anticholinergics, dopamine, and barbiturates May be caused by ophthalmic mydriatics that are administered for intraocular examinations (atropine, scopolamine)
	Anoxia mydriasis: Pupils are dilated bilaterally and do not respond to light stimulus. There is no consensual response or accommodation.	Defect in efferent pathway Transtentorial herniation Anoxia Brain death
Equal pupils with abnormal response	*Hippus:* Pupils may be equal in size. Pupils initially react briskly to light, but then alternate between dilation and constriction.	Defect in efferent pathway Early compression of cranial nerve III Lesion/injury to midbrain Barbiturate toxicity
	Marcus Gunn pupil: Pupils are equal in size, but one pupil has an abnormal response to light. When direct light is shone into the affected eye, there is a sluggish reaction. When direct light is shone into the normal eye, the affected eye will constrict (normal consensual reaction), but when the light is then directed back to the affected eye, the pupil in the affected eye will dilate.	Defect in afferent pathway Lesion or atrophy of the retina or optic nerve causes damage to afferent nerve fibers Retinal detachment Occlusion in retinal vasculature

[a]From Hickey[4] and Barker.[6]

neurotransmitter responsible for this synaptic reaction is norepinephrine.

Cranial Nerves

The eye is innervated by 3 cranial nerves (CN III, IV, VI), which work together to provide smooth eye movements. Cranial nerve III (oculomotor) controls the parasympathetic response of the pupil, causing pupillary constriction.[6] Cranial nerves III, IV, and VI innervate the muscles surrounding the eye and are responsible for extraocular eye movements (EOMs). Assessment of EOM is appropriate in the conscious trauma patient and can be particularly important when caring for patients with facial trauma. However, assessment of EOMs in the unconscious patient with TBI is not always possible or indicated. When caring for this type of patient, it is necessary to assess only cranial nerve III by testing the pupillary response to light.

ABNORMAL PUPILLARY RESPONSE IN TBI PATIENTS

When performing pupillary examinations in patients with TBI, trauma nurses may detect abnormalities, such as an irregular pupil size, shape, or a sluggish or nonreactive pupil. When an abnormality is detected, the trauma nurse should first identify whether the abnormality was present on the previous pupillary examination. If an abnormal pupil is present on the initial pupillary examination, it should be clearly documented, and a physician should be immediately notified. Immediate notification of a physician should occur with changes in pupillary response. Comparing the current examination with the previous to provide time-oriented data for the physician is wise but should never delay immediate physician notification.

A complete neurologic examination should be performed and any changes in the patient's condition should be noted and reported to a physician. An abnormal pupil in a patient with TBI is often indicative of increasing ICP due to progression of the hematoma/hemorrhage or cerebral edema. However, the trauma nurse should be aware of other clinical factors that may cause an abnormal pupil response. Table 2 highlights abnormal pupils that may be seen in TBI patients and identifies both physiologic and clinical factors contributing to the abnormalities. Regardless of the cause of an abnormal pupil, the trauma nurse should always notify a physician immediately when an abnormal pupil is detected. A CT scan and continuous ICP monitoring will aid in definitively identifying the cause of the abnormal pupil.

CONCLUSIONS

Interdisciplinary care for patients with TBI is often extremely complex, and trauma nurses have an integral role in early detection and monitoring of changes in patient status. The pupillary examination is a crucial component of the serial neurologic assessments routinely performed by trauma nurses at all levels of care delivery. The pupillary examination can be quickly and easily performed in the unconscious or minimally responsive patient when a TBI is suspected, and can provide valuable information about the degree of initial or progressing brain injury. As evidenced in the case study, a specific type of TBI, the subdural hematoma, may cause pupillary changes, which indicate the need for rapid interventions to decrease ICP and/or cerebral bleeding and edema. By gaining a better understanding of the physiologic basis of the pupillary response and the particular components of the pupillary examination, trauma nurses are in a key position to detect early changes in a patient's condition and administer or advocate for immediate interventions.

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