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Post-traumatic stress disorder and vision

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Abstract Post-traumatic stress disorder (PTSD) can be defined as a memory linked with an unpleasant emotion that results in a spectrum of psychological and physical signs and symptoms. With the expectation of at least 300,000 postdeployment veterans from Iraq and Afghanistan having PTSD, optometrists will be faced with these patients' vision problems. Complicating the diagnosis of PTSD is some overlap with patients with traumatic brain injury (TBI). The estimated range of patients with TBI having PTSD varies from 17% to 40%, which has recently led the Federal government to fund research to better ascertain their relationship and differences. As a result of the sensory vision system's interconnections with the structures of the limbic system, blurry vision is a common symptom in PTSD patients. A detailed explanation is presented tracing the sensory vision pathways from the retina to the lateral geniculate body, visual cortex, fusiform gyrus, and the hypothalamus. The pathways from the superior colliculus and the limbic system to the eye are also described. Combining the understanding of the afferent and efferent fibers reveal both feedforward and feedback mechanisms mediated by nerve pathways and the neuropolypeptides. The role of the peptides in blurry vision is elaborated to provide an explanation as to the signs and symptoms of patients with PTSD. Although optometrists are not on the front line of mental health professionals to treat PTSD, they can provide the PTSD patients with an effective treatment for their vision disorders.
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According to a recent Rand Corporation report,[1] it is estimated that there will be 300,000 military personnel with post-traumatic stress disorder (PTSD) after deployment from Iraq and Afghanistan. The U.S. Department of Veterans Affairs (VA) has acknowledged that this will be a tremendous burden on the VA health care system.[2] On June 4, 2008, the Department of Health & Human Services announced an agreement between the Department of Defense and the U.S. Public Health Service to increase services for postdeployment military personnel and their families.[3] A more recent message regarding the urgency for immediate implementation of coordinated efforts by the Departments of Defense and Veterans Affairs was delivered by the Military Officers of America Association.[4] If the number (300,000) of postdeployed military is added to the number of civilians in the United States with PTSD diagnosed annually (estimated at 6.8%),[5] a potential health crisis is on the horizon. This report places emphasis on the postdeployment aspects of PTSD as it represents enormity and urgency. A literature search revealed that no articles concerning this specific potential crisis or PTSD in general have appeared in major optometric publications. Accordingly, the purpose of this article is to present the history, prevalence, treatment considerations, and specific vision problems that can be addressed by the optometrist. To make the description of PTSD more comprehensive, the relationship between PTSD and traumatic brain injury (TBI) along with concomitant neurophysiologic manifestations will be discussed.

History of PTSD

When a diagnosis such as PTSD is discussed, a definition or guideline for the diagnosis must be presented. The American Psychiatric Association's lengthy criteria for PTSD

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diagnosis (DSM-IV 309.81) is found in Appendix A (a shorter definition is somewhat problematic because of the many factors involved). Arbitrarily, a description of PTSD from the Civil War will be the starting point from a historical and chronological perspective. A recent report reflects the interest in the Civil War and PTSD with an estimate of 44% of Union Army soldiers having medical or mental disorders after that war.[6] Not known as PTSD at that time, the condition was named *irritable* or *soldier's heart*. During World War I the condition was called *combat fatigue* or *shell shock* and during World War II was known as *battle fatigue* or *gross stress reaction.*[7]

According to Sargant,[8] Breuer and Freud first used the term *abreaction* in their studies about hysteria. Abreaction used in their context means catharsis, and they reported that many cases of hysteria would be eliminated by just "talking it out."[9] Abreaction was the foundation of Sargant's, as well as today's, treatments for PTSD. As noted by Jones et al. in a historical review, Salmon, in 1917, was successful in the treatment of psychiatric casualties of World War I.[10] Salmon and Fenton[11] described the itemized clinical protocols and the facilities that the U.S. Army developed to diagnose and treat World War I veterans. Some of the more salient points were that patients were to be considered sick individuals and should undergo a thorough medical evaluation; occupational therapy was effective as a treatment; and psychiatric social work was a necessary factor in the successful treatment of PTSD.

In his book, Conditioned Reflexes, Pavlov[12] described the effects on behavior as a result of trauma. During a storm in Petrograd in 1924, the dogs' kennel became flooded accompanied by loud sounds of waves of water against the walls and collapsing trees. There is a description of the procedure of retraining one dog in particular who had previously learned a number of *conditioned reflexes*. After the trauma, the dog did not display these reflexes. A program of gradual retraining was presented, including creating a pool of water in the laboratory where the training was conducted. Expanding on Pavlov's research, Sargant developed a technique for soldiers suffering from battle fatigue in World War I and World War II. The technique, known as *abreaction*, is defined by Sargant as "… a process of reviving the memory of a repressed experience and expressing in speech and action the emotions related to it, thereby relieving the personality of its influence."[8] In other words, the technique brings the patient to a crescendo of emotional excitement followed by emotional collapse. After the emotional collapse, the memory of the traumatic event still remains, but the associated emotional component is decreased or eliminated. Pavlov[12] gave the explanation for the emotional inhibition as cortical extinction, which was termed *transmarginal inhibition*. He further proposed that a psychological session inducing the emotional state, transmarginal inhibition, can be achieved by dancing, singing, or other activity that produces a state of emotional excitement.

A vivid memory associated with an emotional charge is known as an *eidetic image*. Basing treatment for hysteria on this definition, Ahsen[13] developed a technique of having the patient relive the emotional experience and then change the negative outcome. Subsequently, Bandler and Grinder[14] popularized the technique, naming it *neuro-linguistic programming* (NLP), without giving credit to Ashen.

Beginning in 1991, and continuing until today, a number of studies have reported successful treatment of PTSD, as measured with the *Minnesota Multiphasic Personality Inventory* with alpha-theta training, which has subsequently been named the *Peniston-Kulkosky Protocol* (P-KP).[15,16] Peniston provided a historical perspective of their training protocol, crediting earlier researchers, particularly with regard to alpha training.[17] Further description of the P-KP technique is given in the Treatment for PTSD, Neurotherapy section below.

In 2003, Rizzo introduced a virtual reality technique derived from Pavlov's experience with traumatized dogs, as described above.[18] Within the immersive environment, smell and hearing were added to the visual stimuli to aid the veteran to relive an emotional experience with the goal of retaining the memory of the event but disassociating the emotional factor. On the U.S. Department of Defense Mental Health System Web site, a new program titled *afterdeployment.org* originated on August 5, 2008.[19] The Web site contains self-help guidelines to reduce stress and deal with other emotional issues for the returning war veteran.

Tests for PTSD

Tests for PTSD are based on DSM-IV diagnostic criteria (*see* Appendix A). Although there are many tests for PTSD, 4 in particular will be discussed: the *Minnesota Multiphasic Personality Inventory* (MMPI),[16,20,21] the PTSD Checklist—Military Version (PCL-M) (*see* Appendix B),[22,23] and 2 Department of Defense Tests, DD 2795 and DD 2900.

The current MMPI test for PTSD is the Minnesota Multiphasic Personality Inventory–PTSD known as the MMPI–PTSD.[24] Three forms of the MMPI (MMPI, MMPI-2, and the MMPI-PTSD) are used in the diagnosis of PTSD. Lyons and Wheeler-Cox[25] discussed the various issues and preference among these 3 forms of the MMPI. The MMPI-PTSD is the most frequently used of the 3 MMPI tests for evaluating PTSD, although some still prefer the MMPI-2. The MMPI-PTSD test contains 49 items and takes 15 minutes to complete. A cutoff score of 26 or more leads to the diagnosis of PTSD.[26] The PCL-M is a 17-item questionnaire for PTSD as shown in Appendix B.[27] The test takes 5 to 7 minutes to complete, with a cutoff score of 50 or more indicating PTSD.[28] The evidence of consistency for the MMPI is 0.95 and for the PCL-M is 0.97; the evidence for validity for the MMPI is moderate–strong, and for the PCL-M, strong.[29]

The remaining 2 tests, which have been developed within the last 10 years, are administered before and after military deployment. Military personnel are required to complete form DD 2795 before deployment and form DD

2900 after deployment. These tests are performed before and after deployment to obtain a comparative assessment of an individual's mental state when returning from combat. The main concerns associated with the DD 2795 and DD 2900 forms are the following: (1) Because of the extremely large number of personnel expected to have PTSD after deployment, it has been suggested that the analysis of DD 2795 and 2900 forms be filed online. With 300,000 potential veterans, the online scoring would result in incredible time and financial savings. (2) There are currently tests for PTSD, such as the MMPI-PTSD and the PCL-M, which have been extensively studied. To make the diagnosis of PTSD from the DD 2795 and 2900 forms more meaningful, research studies are suggested to correlate their results with those of the MMPI-PTSD and PCL-M tests.

Certainly, with any new test, the established criteria for PTSD will have to be biased to minimize the number of missed diagnoses. This can be accomplished by using a well-known procedure of Signal Detection Theory (SDT), which is used as an aid in decision-making under conditions of uncertainty.[30] For example, SDT can be used to bias decision-making to determine a minimal number of missed diagnoses, with an allowance for overdiagnosis.

Prevalence of PTSD

Based on earlier and current tests, the prevalence of PTSD can be estimated. The current prevalence of PTSD among Vietnam veterans is approximately 15%,[31] and, according to Thompson et al.,[32] between 12.2% and 15.8%. Israeli soldiers in the 1982 Lebanon War had a 62% incidence of PTSD.[31] The prevalence of PTSD in World War II veterans has been reported to be 37%, for Korean War veterans 80%, and for Gulf War veterans (broken down by gender) 9.4% of men and 19.8% of women.[31] Reger and Gahm[22] report the incidence of PTSD in returning active duty Iraqi soldiers at 17% and for reserve soldiers 24%. In addition to actual combat, other issues were found to be important in the etiology of PTSD: premilitary factors, war-zone exposure, and postmilitary factors.[31]

Neurophysiology of PTSD

van der Kolk et al.,[33] Morris et al., [34] Davidson, [35] and Voss and Temple[36] suggested that the lateral nucleus of the amygdala is involved with memories of fear and the anterior cingulate cortex with memory.[32,34,36,37] Much of the feedback loops to and from the amygdala and the anterior cingulate cortex are controlled by the hypothalamus. It is important to note that the hypothalamus is involved with the regulation of bodily functions, including memory, emotion, the pituitary gland, the immune system, cardiac function, and vision.[38] Hence, this can explain many of the nervous system symptoms related to PTSD and, as van der Kolk[37] described, the beneficial effects of relaxation, i.e., the effects on heart rate variability (HRV) by hatha yoga. Further evidence for hypothalamic activity involved in the symptoms and treatment of PTSD was reported by Yehuda et al.,[39] where they found that negative feedback inhibition of adrenocorticotropic hormone (ACTH) levels by cortisol was likely related to hypothalamic regulation of pituitary function. In their article on stress and irritable bowel disorder (IBD), Mawdsley and Rampton[40] reported a similar stress mechanism, which may account for the high proportion of patients with PTSD who also suffer from IBD.[41] In addition to the innumerable functions and connections discussed above, the hypothalamus has further wide-ranging physiologic influence via the hypothalamic-pituitary-adrenal axis (HPA). The importance of the role of the HPA axis in relation to stress was emphasized in 1955 by Nobel Prize winners Guillemin[42] and Schally[43] (*see* Figure 1).

Treatment for PTSD

A review of the various treatment methods that have been used for PTSD for the last 20 years is presented.

Cognitive behavioral therapy

Cognitive behavioral therapy (CBT) includes trauma-focused cognitive therapy, exposure therapy, neurotherapy, and eye movement desensitization and reprocessing (EMDR). These techniques are used individually or in combination with each other.[44]

Trauma-focused cognitive therapy

Trauma-focused cognitive therapy modifies beliefs related to the traumatic experience and the individual's behavior during the trauma, for example, guilt and shame.[45]

Exposure therapy

One of the first articles that described the rationale for exposure therapy emphasized the importance of exposure to the initial trauma in reducing fear and anxiety related to that initial trauma.[46] Later, Minnen and Foa[47] reported that there was no difference in the effectiveness of treatment between a 30-minute (exposure) and a 60-minute (prolonged exposure) therapy.

Important aspects of exposure therapy are the repeated confrontation of the traumatic memories and the avoidance of situations that recall the emotions associated with the trauma.[45] Four components of exposure therapy were given by Cahill et al:[44] (1) education regarding the trauma, especially a rationale for using exposure therapy; (2) controlled breathing training; (3) the use of imagery of the trauma, both during therapy and as a home exercise; and (4) real-life reminders of the trauma, usually given as a home

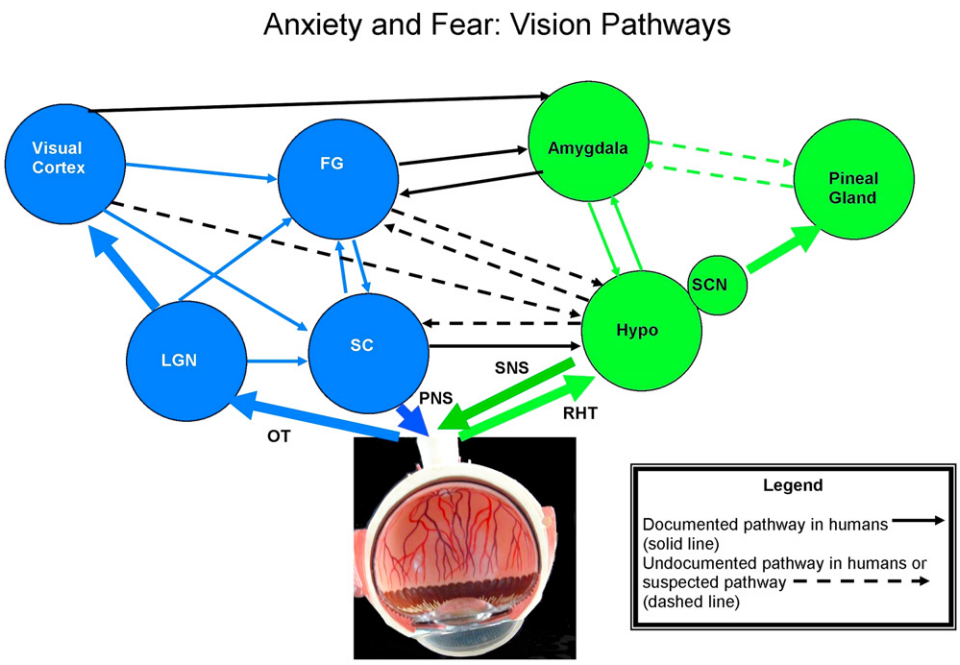

Figure 1 Anxiety and fear: vision pathways. A diagrammatic representation of the multiple interconnections involving the vision and emotional systems. FG, fusiform gyrus; Hypo, hypothalamus; LGN, lateral geniculate nucleus; OT, optic tract; PNS, parasympathetic nervous system; RHT, retino-hypothalamic tracts; SC, superior colliculus; SCN, suprachiasmatic nucleus; SNS, sympathetic nervous system.

exercise. According to a review article, there have been numerous studies, with a wide range of traumatic events that have reported that approximately 80% of the patients receiving between 9 and 12 sessions have significant improvement, with approximately 40% completely eliminating their symptoms.[48]

Neurotherapy

Neurotherapy, in the context of this article, can be defined as the treatment of neurological disorders via brain wave biofeedback. There are 4 spontaneous brain waves that are measured in the electroencephalogram. The 4 waves are measured in cycles per second (Hz). The slowest of the brain waves is the Delta wave, which ranges from 0 to 3 Hz, and is most prominent during sleep. The next fastest brain wave is the Theta wave, 4 to 7 Hz, and is related to creative activity, loss of concentration, and daydreaming. The Alpha wave is from 8 to 12 Hz and is the dominant brain wave during activities such as Zen meditation. The fastest of the brain waves is 13 Hz and greater and is known as the Beta wave, which is related to everyday awake activity. The basic P-KP technique is designed to increase the alpha–theta brain wave amplitudes, while decreasing delta brain waves.

A Web site that addresses the neurotherapy treatment of PTSD is Homecoming for Veterans.[49] The neurotherapy technique that is utilized and described is based on the P-KP as previously described.[17] One of the studies cited, Peniston and Kulkosky,[20] reported a 50% decrease in the PTSD subscale of the MMPI-2. Their organization has volunteer neurotherapists throughout the United States providing courtesy treatment to veterans with PTSD. A recent report by Putman[50] noted the efficacy of eyes-open Alpha wave training in contrast to eyes-closed Alpha wave training; the latter being the usual means of training Alpha to increase a performance task. His subjects were 77 U.S. Army Reservists. These data later contributed to additional documentation at Homecoming for Veterans on the use of the P-KP.[49]

Applying a comparable technique to the P-KP, Rosenfeld[15] reported improvement in MMPI-2 scores of 122 patients with a diagnosis of a psychotic or personality disorder. Similarly, Scott et al.[16] reported improved MMPI-2 scores for 121 patients after a protocol much like that described by Rosenfeld.[15] (A recent review of EEG biofeedback by Sokhadze et al.[51] generally supports previous findings with the P-KP for patients with substance abuse as well.)

In a review of neurofeedback, Hammond[52] cited several studies that successfully treated PTSD via brain wave biofeedback (neurofeedback). The rationale was based on the understanding that the left hemisphere is associated with memory and the right hemisphere is associated with negative emotion, with an activation difference between the 2 hemispheres leading to depression.

Eye movement desensitization and reprocessing

Eye movement desensitization and reprocessing (EMDR) is a standardized procedure that stimulates bilateral eye movements, bilateral tapping, or binaural tones, while the patient imagines associations and memories related to the

trauma.[45] However, in a review of 34 studies of the treatment of PTSD, EMDR was found to be no more effective than other exposure techniques, with the eye movement component of the technique noted to be unnecessary.[52,53]

Virtual reality

Ten Vietnam veterans, diagnosed with PTSD, were treated by VR exposure therapy from 8 to 16 sessions. Clinician-rated PTSD symptoms decreased as a result of the treatment for 3 months, but not for 6 months.[54] (Kenny et al.[55] described in detail how to train a novice therapist to interview PTSD patients in a VR setting.) A review of 21 VR studies treating anxiety and specific phobia reports large effects on all affective domains.[56] The affective domains are categories of emotional feelings relating to one's self both in independent and social settings.

Using the PCL-M and the Behavior and Symptom Identification Scale–24 (a 24-item self-report using a 5-point frequency or severity scale that assesses treatment outcome) as pre- and post-treatments measures, Reger and Gahm[22] reported on the successful use of VR in the treatment of a 30-year-old soldier who had been deployed in combat in Iraq for more than 1 year. The soldier received six 90-minute sessions during a period of 4 weeks. Another veteran of the Iraqi war was given four 90-minute VR sessions over a course of 4 weeks.[57] According to a comparison of the results of a battery of pre- and post-treatment rating scales, there was a decrease in both patients' self-reported symptoms. The authors hold hope for short-term treatment for PTSD. Detailed descriptions of a VR application for Iraqi war veterans were also given by Rizzo et al.,[58,59] including visual scenarios, patient interactions, and man–machine interfaces. Rizzo et al.[23] report on the successful treatment of 2 U.S. Army Iraqi veterans with virtual reality. The treatment protocol was 10 VR sessions. Success was measured by pre- and post-treatment measures on the PCL-M and other PTSD rating scales.

Pharmacology

In a comprehensive review of the various treatments for PTSD, van Etten and Taylor[60] reported that selective serotonin reuptake inhibitors (SSRIs) and carbamazepine had the greatest effect during a single trial on PTSD symptoms, anxiety, and depression. A wide range of psychotropic pharmaceutical agents have been used in the treatment of PTSD. Friedman et al.[61] suggested the following: SSRIs sertraline, paroxetine, fluoxetine, fluvoxamine, and citalopram; tricyclic antidepressants (TCAs); monoamine oxidase inhibitors (MAOIs); antidepressants nefazadone, venlafaxine, and bupropion; anti-adrenergic agents carbamazepine, valproate, lamotrigine, and gabapentin; atypical antipsychotic agents risperidone and olanzapine; stimulants dextroamphetamine and methylphenidate; and alpha-2 agonists clonidine and guanfacine. Another article reported that the following pharmaceutical agents are also used in the treatment of PTSD: fluoxetine, paroxetine, mirtazapine, olanzapine, fluphenazine, and sertraline (the only drug that has U.S. Food and Drug Administration approval for PTSD treatment).[62] Interestingly, in combination with exposure therapy, sertraline was not found to be more effective than exposure therapy 5 weeks after treatment for PTSD.[63] In general, pharmaceutical agents should be used judiciously, especially the SSRIs. For a summary of the side effects of drugs used in PTSD treatment see Table 1.

Additionally, Ballenger[64] and Gelenberg[65] list a number of drugs and classes of drugs that are used in the treatment of PTSD. As can be seen in Table 1, benzodiazepines (BZDs), MAOIs, SSRIs, and TCAs all have ocular side effects, with abnormal and blurred vision as the most common symptoms. Additionally, Risperdal is prescribed for PTSD treatment, with abnormal vision being reported as a common side effect.[66] Topamax is the drug with the most side effects, including acute myopia, secondary angle-closure glaucoma, maculopathy, nystagmus, diplopia, and abnormal vision.[67] Likewise, one should have an awareness of the visual impact of some of the toxic agents as well. (Toxicity to veterans from Gulf War exposure to nerve agents and pesticides is described by Golomb.[68]) The use of acetylcholinesterase inhibitors have been found to produce multisymptom health problems, including the following visual side effects: miosis, lacrimation, blurred vision, accommodative spasm, and diplopia.[69]

PTSD and TBI

There is considerable difficulty in making a differential diagnosis between PTSD and TBI. PTSD has been defined as a memory linked with an unpleasant emotion that results in a spectrum of psychological and physical signs and symptoms. TBI, however, occurs after a fall, assault, explosion, or motor vehicle accident. TBI is classified as severe if there is an extended period of unconsciousness and mild if the period of unconsciousness is brief.[70] There is no loss of consciousness associated with PTSD. In other words, the signs and symptoms related to PTSD are a result of the dynamics of the emotional system and other components of the central nervous system (CNS) and autonomic nervous system (ANS), whereas TBI is a result of physical or physiological damage to the CNS and/or the ANS. Moreover, an underlying difficulty in neuropsychological testing is described in detail by Dikmen et al.[71] The Halstead-Reitan test is a standardized test used in the diagnosis of PTSD and TBI. The authors reported a coefficient of correlation for the subsets to range from 0.60 to 0.92, or a coefficient of reliability of 0.36 and 0.85, respectively. For example, a coefficient of reliability of 0.36 accounts for only 36% of the relationship between the test and TBI, with 64% being related to other factors.

This confusion has led to the inaccuracy of using ICD-9-CM codes to diagnose mild TBI versus PTSD, which led to a significant number of false–positive and false–negative

Table 1 Side effects of drugs used in PTSD treatment[130]

Drug used in PTSD treatment (alphabetic order)	Vision side effects
Benzodiazepines (BZDs)	
Valium	Blurred vision, diplopia
Buproprion	Blurred vision, diplopia, accommodation abnormality, dry eye, increased intraocular pressure, mydriasis
Buproprion SR	Blurred vision, diplopia, accommodation abnormality, dry eye, increased intraocular pressure, mydriasis
Mirtazapine	Eye pain, accommodation abnormality, conjunctivitis, keratoconjunctivitis, lacrimation disorder, glaucoma, blepharitis
Monoamine oxidase inhibitors (MAOIs)	
Eldepryl	Blurred vision, diplopia
Nefazodone	Abnormal vision (scotomata and visual trails), blurred vision, eye pain, dry eye, accommodation abnormality, diplopia, conjunctivitis, mydriasis, keratoconjunctivitis, photophobia, glaucoma, night blindness
Risperdal	Abnormal vision, accommodative disturbances, xerophthalmia, diplopia, photophobia, abnormal lacrimation
Selective serotonin reuptake inhibitors (SSRIs)	
Prozac	Abnormal vision, conjunctivitis, dry eyes, mydriasis, photophobia, blepharitis, diplopia, exophthalmos, eye hemorrhage, glaucoma, iritis, scleritis, strabismus, visual field defect
Serotonin- norepinephrine reuptake inhibitors (SNRIs)	
Topamax	Nystagmus, diplopia, abnormal vision, eye abnormality, abnormal lacrimation, myopia, conjunctivitis, accommodation abnormality, photophobia, strabismus, mydriasis, iritis
Venlafaxine	Blurred vision, mydriasis, accommodation abnormality, nystagmus, abnormal vision, cataract, conjunctivitis, corneal edema, diplopia, dry eyes, eye pain, photophobia, visual field defect, blepharatis, chromatopsia, corneal edema, exophthalmos, glaucoma, retinal hemorrhage, subconjunctival hemorrhage, keratitis, miosis, papilledema, decreased pupillary reflex, scleritis, uveitis
Tricyclic anti-depressants (TCAs)	
Amitriptyline	Blurred vision, disturbance of accommodation, increased intraocular pressure, mydriasis

findings in making the differential diagnosis.[72] Acknowledgement of this problem is noted in a recent Centers for Disease Control and Prevention (CDC) report listing the specific inadequacies of the current ICD-9-CM criteria for diagnosis of TBI.[73] Recognizing the variation in the percentage of veterans with both PTSD and TBI, the University of California, San Diego (UC-San Diego) School of Medicine will lead a $60-million, 5-year, 10-site clinical consortium funded by the Department of Defense Psychological Health/Traumatic Brain Injury Research Program (DOD PH/TBI) to better understand the relationship between these 2 disorders.[74]

Neurophysiologic aspects of vision, PTSD, and TBI

Figure 1 and Table 2 show the myriad connections between the vision system and the brain in relationship. It is important to note that the connections have been limited to certain structures that have been deemed the most important and relevant to the current discussion.

Figure 1 shows that fibers from the optic tract (OT) make their connection in the lateral geniculate nucleus (LGN) and the hypothalamus (HYPO) via the retinohypothalamic tracts (RHT). Fibers from the LGN make

connections with the visual cortex, the superior colliculus (SC), the amygdala, and the fusiform gyrus (FG). Fibers from the hypothalamic structure of the suprachiasmatic nucleus (SCN) go to the pineal gland, and hypothalamic fibers make connection with the amygdala. The visual cortex has connections with the amygdala, the SC, and the FG. The SC makes connections with the amygdala, the FG, and the HYPO. The FG makes connections with the amygdala and the SC. A number of suspected connections help explain vision function as well as pathways found in nonhuman species.[75,76] The suspected connections include: from the visual cortex to the HYPO, to and from the HYPO and the FG, the amygdala to the SC, and to and from the pineal gland and the amygdala. All the connections are shown with their relevant references in Table 2. Completing the feedback and feedforward connections are the parasympathetic nervous system (PNS) and motor input: SC to Edinger-Westphal nucleus to ciliary ganglion to short ciliary nerves; and the sympathetic nervous system (SNS): HYPO to the ciliospinal center of Budge to the superior cervical ganglion to short and long ciliary nerves.[77]

With the above background, the vision disorders related to PTSD will now be explained. To demonstrate the strength of the relationship between vision and emotional state is a recent article relating visual impairment to a high risk of suicide.[78] Visual impairment was defined as including visual acuity, contrast and glare sensitivity, visual fields, and stereoacuity. The authors suggest that an improvement in vision function may decrease the risk of suicide. A basic example of the intimacy between the vision processing system and the emotional input into vision functioning is noticed upon seeing a sad event, with accommodative fluctuations, hysterical visual fields, tearing, and pupil size.[77]

From a literature review, the main vision complaint of patients with PTSD is blurry vision.[79-82] The studies reporting the complaint of blurry vision were not conducted by optometrists or ophthalmologists; accordingly, there is no elaboration as to the nature or cause of the blurry vision. Because the term *blurry vision* is vague, the specific factors that can cause blurry vision can include tear film irregularities, corneal pathology, accommodative dysfunction, uncorrected refractive error, intraocular pressure, and retinal pathology. For comparison in a series of publications, a group of researchers at the State University of New York, State College of Optometry (SUNY) reported on vision disorders related to TBI.[83-85] They reported that the most common vision disorders in patients with TBI are accommodative (41%), versional (51%), vergence (56%), strabismus (26%), and visual field defects (39%). (Based on the work of the SUNY group, Padula et al.[86] brought out the need to include optometric rehabilitation in federal legislation for veterans with TBI. As a result of these and other endeavors, Public Law 110-181 was passed.[87] This law specifically includes optometric care in its provisions.

PTSD alone, as an emotional reaction, can cause blurry vision because it interferes with the function of tears, cornea, the ciliary muscle, and the retina. The neurologic mechanisms involved in these disorders involves ocular input from PNS, motor nuclei, and SNS primarily via neuropolypeptide, cytokine, and nitric oxide activities as controlled by the HYPO either directly or via its numerous interconnections.[88] An abbreviated review of the literature revealed the following relating peptide function to the ocular components responsible for clear vision: tears and lacrimal gland function,[89-96] the cornea[89,97-99] ciliary muscle function,[100-104] and retinal activity.[105-113] Besides the direct effects from peptides and other agents, pharmaceutical agents prescribed for PTSD also have side effects on vision function (*see* Table 1). (For more information on the relationship between hypothalamic function and vision see Trachtman[114] and Table 2.)

Table 2 Anxiety and fear: vision pathways

From	To*				
Amygdala	Fusiform Gyrus (FG): 131, 132	Hypothalamus (Hypo): 133, 134, 135, 136	Pineal Gland (Pineal): 137, 138, 161	Superior Colliculus (SC): 139	Visual Cortex: 135, 140, 141
FG	Amygdala: 131, 132	Hypothalamus (Hypo): 142, 161	Superior Colliculus (SC): 143		
Hypo	Amygdala: 144, 145, 146	Fusiform Gyrus (FG): 142, 161	Pineal Gland (Pineal): 147	Superior Colliculus (SC): 148, 149, 150	
LGN	Amygdala: 151	Fusiform Gyrus (FG): 152	Superior Colliculus (SC): 153, 154		
Pineal	Amygdala: 137, 138	Hypothalamus (Hypo): 147			
SCN	Pineal: 147				
SC	Amygdala: 155, 156, 157	Fusiform Gyrus (FG): 133, 143, 158	Hypothalamus (Hypo): 159		
Visual Cortex	Amygdala: 140, 141	Fusiform Gyrus (FG): 160, 161	Hypothalamus (Hypo): 145	Superior Colliculus (SC): 140, 141	

* Numbers represent references.

One of the earliest studies on the relationship between PTSD and TBI reported that 33% of the TBI patients also had PTSD diagnosed according to the DSM-III-R criteria.[115] According to Klein et al.,[116] an important factor in TBI patients with PTSD was memory of the trauma, with the incidence of PTSD in TBI patients 5 times higher in patients who had a memory of their trauma. They also found in their study of 120 patients that 22% of TBI patients also had PTSD diagnosed. In a relatively small sample, (n=15) Glaesser et al.[117] reported that 27% of patients with mild TBI also had PTSD diagnosed. A later report by Bombardier et al.[118] noted between 17% and 30% of patients with TBI also met the DSM-IV criteria for PTSD. A more recent review of the literature on PTSD and TBI notes the possibility that there is an overdiagnosis of PTSD in patients with TBI because of overlapping symptoms, and there is no current treatment for patients with the dual diagnosis.[119] In a study of Iraq and Afghanistan veterans, 39.6% with mild TBI also had PTSD diagnosed.[120]

Summary

There appears to be general agreement that CBT is the most effective method for the treatment of PTSD.[60,62,121-125] Bisson and Andrew[126] in a review article noted that CBT and EMDR were equally effective in reducing the symptoms of PTSD. Important considerations in the treatment of PTSD include pretrauma health, the nature of the trauma event, and events immediately and subsequently occurring after trauma.[127] Spinazzola et al.[128] expressed their concern for the external validity of studies on the treatment of PTSD. The problem that the authors described is that demographic information is not reported, therefore, making generalization from the study sample to the general population difficult to interpret.

As a result of decades of war, the Israeli military[129] suggests that the treatment for PTSD should be given as soon as possible after the causative trauma, bureaucratic channels should be streamlined to decrease or eliminate delays in treatment and follow-up care, and patients and therapists should be made aware of their own possible symptoms of PTSD, as many therapists experience PTSD by providing treatment.

Optometrists have an excellent opportunity to apply their clinical skills to help the predicted hundreds of thousands of returning veterans from the Iraqi and Afghanistan wars as well as thousands of civilians who suffer from PTSD annually. First, the optometrist needs to recognize the signs and symptoms associated with PTSD, such as those listed in Appendices A and B. Second, the optometrist should have a general plan available to treat the visual concomitants of PTSD, such as decreased visual acuity, glare, contrast sensitivity, and stereovision issues, as well as assistance with the visual skills of accommodation, binocular vision, and peripheral awareness. Third, the optometrist should be aware of the pharmacologic implications related to the treatment of PTSD. The most common ocular side effects are accommodative dysfunction, conjunctivitis, diplopia, and mydriasis.

On another level, optometrists should become familiar with the interaction of the vision and emotion pathways to better understand ocular pathologies especially as related to the tears, cornea, ciliary body/muscle, intraocular pressure, and the retina. Much of this information has been cited with the work of McDermott[89] at the University of Houston College of Optometry, who investigated the therapeutic pharmacologic properties of the peptides. All health professionals will be facing the overwhelming number of PTSD patients in the near future, and optometrists can be on the front line of the caregivers.

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Appendix A

Diagnostic criteria for 309.81 Post-traumatic Stress Disorder

A. The person has been exposed to a traumatic event in which both of the following were present:
   (1) The person experienced witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of others.
   (2) The person's response involved intense fear, helplessness, or horror. **Note:** In children, this may be expressed instead by disorganized or agitated behavior.

B. The traumatic event is persistently re-experienced in one (or more) of the following ways:
   (1) Recurrent and distressing recollections of the event, including images, thoughts, or perceptions. **Note:** In young children, repetitive play may occur in which themes or aspects of the trauma are expressed.
   (2) Recurrent distressing dreams of the event. **Note:** in children, there may be frightening dreams without recognizable content.
   (3) Acting or feeling if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated). **Note:** In young children, trauma-specific reenactment may occur.
   (4) Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.
   (5) Physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by 3 or more of the following:
   (1) efforts to avoid thoughts, feelings, or conversations associated with the trauma
   (2) efforts to avoid activities, places, or people that arouse recollections of the trauma
   (3) inability to recall an important aspect of the trauma
   (4) markedly diminished interest or participation in significant activities
   (5) feeling of detachment or estrangement from others
   (6) restricted range of affect (e.g., unable to have loving feelings)
   (7) Sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span)

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by 2 (or more) of the following:
   (1) difficulty falling or staying asleep
   (2) irritability or outbursts of anger
   (3) difficulty concentrating
   (4) hypervigilance
   (5) exaggerated startle response

E. Duration of the disturbance (symptoms in Criteria B, C, and D) is more than one month.

F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if:

Acute: if duration of symptoms is less than 3 months.

Chronic: if duration of symptoms is 3 months or more

Specify if:

With Delayed Onset: if onset of symptoms is at least 6 months after the stressor

From American Psychiatric Association. 309.81 Posttraumatic stress disorder. In: Diagnostic and Statistical Manual of Mental Disorders, Fourth ed. American Psychiatric Association, Washington 1994:424-429.

Appendix B

PTSD Checklist – Military Version (PCL-M)

Patient's Name: _____

Instruction to patient: Below is a list of problems and complaints that veterans sometimes have in response to stressful life experiences. Please read each one carefully, put an "X" in the box to indicate how much you have been bothered by that problem *in the last month*.

No.	Response	Not at all (1)	A little bit (2)	Moderately (3)	Quite a bit (4)	Extremely (5)
1.	Repeated, disturbing *memories, thoughts, or images* of a stressful military experience from the past?					
2.	Repeated, disturbing *dreams* of a stressful military experience from the past?					
3.	Suddenly *acting* or *feeling* as if a stressful military experience *were happening* again (as if you were reliving it)?					
4.	Feeling *very upset* when *something reminded* you of a stressful military experience from the past?					
5.	Having *physical reactions* (e.g., heart pounding, trouble breathing, or sweating) when *something reminded* you of a stressful military experience from the past?					
6.	Avoid *thinking about* or *talking about* a stressful military experience from the past or avoid *having feelings* related to it?					
7.	Avoid *activities* or *situations* because they *remind you* of a stressful military experience from the past?					
8.	Trouble *remembering important parts* of a stressful military experience from the past?					
9.	Loss of *interest in things that you used to enjoy?*					
10.	Feeling *distant* or *cut* off from other people?					
11.	Feeling *emotionally numb* or being unable to have loving feelings for those close to you?					
12.	Feeling as if your *future* will somehow be *cut short*?					
13.	Trouble *falling* or *staying asleep*?					
14.	Feeling *irritable* or having *angry outbursts*?					
15.	Having *difficulty concentrating*?					
16.	Being *"super alert"* or watchful on guard?					
17.	Feeling *jumpy* or easily startled?					

From **PCL-M for DSM-IV (11/1/94)** Weathers, Litz, Huska, & Keane National Center for PTSD - Behavioral Science Division.