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Combat Related Posttraumatic Stress Disorder – History, Prevalence, Etiology, Treatment, and Comorbidity

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1. Introduction

This chapter seeks to provide a better understanding of combat related posttraumatic stress disorder. Some of the information presented in this chapter may apply broadly to all populations affected by posttraumatic stress disorder, but should not be used as a primary reference for the disorder as a whole. This chapter will first provide a brief history of the diagnosis and discuss the current diagnostic criteria including potential changes that have been suggested for the Diagnostic Statistical Manual – V. Next, the chapter will present the prevalence of posttraumatic stress disorder and explain potential gender differences in soldiers affected by the disorder. Theories of how an individual obtains posttraumatic stress disorder will be discussed and current and novel treatments will be explained. A brief discussion on traumatic brain injury will also be presented, as it is a common comorbidity of combat related posttraumatic stress disorder.

2. History of posttraumatic stress disorder

Posttraumatic stress disorder was not officially recognized as psychological disorder until the Diagnostic Statistical Manual -III, which was published in 1980 (American Psychiatric Association, 1980; Lasiuk & Hegadoren, 2006). Posttraumatic stress disorder was known by an array of different labels previous to 1980, such as combat neurosis, railway spine, shell shock, soldier's heart, and stress response syndrome. Although it has been speculated that posttraumatic stress disorder has existed in all trauma stricken populations throughout history,the occurrence has been documented primarily in soldiers who experienced combat related trauma. (Jones et. al., 2003; Lasiuk & Hegadoren, 2006). One exception to this pattern is the historical concept of hysteria. Hysteria has also received much attention, but the symptoms associated with this term have evolved throughout history and therefore the term can only be loosely associated with posttraumatic stress disorder. This chapter will make reference to numerous historical figures that noted the similarities of hysteria to the symptoms that they were observing, but it must be noted that this concept is loosely defined.

2.1 Railway spine, soldiers' heart, and hysteria

Another non-military population who exhibited posttraumatic stress disorder-like symptoms is seen in the documentation of a phenomenon called *railway spine*. This is the

first time where a cluster of symptoms that resembled posttraumatic stress disorder was documented on within medical literature. Railway spine was observed in London in the late 1700's in railway passengers and workers who were in train crashes (Lasiuk & Hegadoren, 2006; Micale, 1990; Ray, 2008). They experienced the physical effects of the crash such as whiplash, but more importantly they were said to have born the psychological effects of the trauma from the crash. Some of the symptoms of railway spine that resemble posttraumatic stress disorder include: nightmares about the crash, avoiding trains as a means of transportation, and difficulty sleeping. At the time, these symptoms were seen by some as being consistent with hysteria, which was believed to more commonly occur in females.

Since some of the individuals who suffered from railway spine believed that the railway companies should be legally liable for their passenger's and worker's well being, there was much debate as to whether a train crash could cause chronic psychological impairment (Lasiuk & Hegadoren, 2006). At the time, some believed that the individuals were faking their symptoms in order to receive financial gain from the railway companies. Others believed that the symptoms were legitimate which inspired a debate about what caused the symptoms. An English surgeon named John Eric Erichsen believed that hysteria should not be associated with railway spine and that its cause was rooted in an organic illness (Lasiuk & Hegadoren, 2006; Erichsen, 1866 & 1886 as cited in van der Kolk, 2007). Another English surgeon, Herbert Page opposed Erichsen's belief and argued that fear could be a sufficient cause for the symptoms (Lasiuk & Hegadoren, 2006; van der Kolk, 2007). Herman Oppenheim argued that railway spine could result from slight molecular changes in the central nervous system and renamed it *traumatic neurosis* (Lasiuk & Hegadoren, 2006; Oppenheim, 1889 as cited in van der Kolk, 2007). This is the first time where the title of the disorder implies that trauma is implicated in the development of the disorder. Kraepelin later used the term traumatic neurosis in reference to a reaction that was seen in those who survived through accidents or other disasters (Kraepelin, 1899 as cited in Ray, 2008).

During the late 1800's, many theorists became interested in the etiology of hysteria. Individuals such as Charcot and Janet contended that an individual must experience trauma in order to develop hysteria-like symptoms (Ray, 2008). Both individuals also agreed that hysteria was not solely a female disorder and pointed out many male populations that experienced symptoms that mimicked hysteria. One population Janet highlighted was males who had suffered from railway spine. Janet developed the term *neurasthenia* which encompassed a number of reactions to emotional trauma (Ray, 2008). Neurasthenia included symptoms such as headaches, fatigue, sleep issues, and emotional and somatization disorders.

Disorders that are similar to combat related posttraumatic stress disorder emerged once again during the Boer, Crimean, and American Civil Wars (Ray, 2008). Terms such as *soldiers' heart* and *DaCosta syndrome* were developed to describe symptoms that were frequently seen in soldiers after being exposed to combat situations. Some of the symptoms associated with soldiers' heart included "extreme fatigue, tremors, dyspnea, palpitations, [and] sweating" (Ray, 2008, p. 218). The central focus when providing a soldiers' heart diagnosis was the abnormality of the soldier's heartbeat. Little attention was paid to their emotional response to the trauma. Since soldiers were expected to be courageous, when a soldier showed any kind of fatigue they were only briefly sent to the back of the battle lines, so that they could recoup (Ray, 2008). After they received some time in the back, they were believed to have recovered and were sent back to the front lines. As a result, soldiers were likely exposed to multiple traumas during war.

2.2 World War I – Shell shock

A British military psychologist named Charles Samuel Myers was the first to use the term *shell shock* in medical literature (Myers, 1915 as cited in van der Kolk, 2007). Previous to his writings, the term was used in reference to British soldiers during World War I who had been exposed to a detonation or explosion, but had not sustained a visible head injury (Jones et. al., 2007). Some of the symptoms soldiers exhibited included tremors, dizziness, increased sensitivity to noise, headaches, difficulty concentrating, and amnesia (Turner, 1915 as cited in Jones et. al., 2007). Frederick Mott, a British neuropathologist suggested that shell shock impacted the tissue in the brain and spinal chord and could be fatal in extreme cases (Mott, 1917 as cited in Jones et. al., 2007). He also believed that some of the symptoms could be attributed to the gases that soldiers were exposed to during an explosion and that the gases could cause damage to the central nervous system (Mott, 1919 as cited in Jones et. al., 2007). Myers later conducted research on shell shock and suggested that the disorder may also result from psychological distress. He believed this because many of the soldiers who showed symptoms that were consistent with those of shell shock, had not been anywhere near an explosion (van der Kolk, 2007). The British Army was compelled to accept Myers' hypothesis because it enabled them to force soldiers to return to combat since the problem was psychological and they were not physically injured (Jones et. al., 2007). Subsequently the army declared two subtypes of shell shock, those who had been exposed to an explosion and those who were said to suffer from "nervousness" due to their anxiety about combat (Sloggett, 1916 as cited in Jones et. al., 2007).

By 1917, shell shock was said to have accounted for one in seven discharges from the British Army (Salmon, 1917 as cited in Jones et. al., 2007). Many doctors at the time believed that having shell shock was synonymous to being a coward (van der Kolk, 2007). Since numerous soldiers sought pensions for the effects of shell shock, the British Army became much more conservative with the diagnosis (Jones et. al., 2007). They intended that only the soldiers who were actually exposed to an explosion receive the diagnosis. Consequently, soldiers who were still serving and were dismissed from their duties for symptoms that resembled shell shock were said to be "not yet diagnosed nervous" (Jones et. al., 2007). Of those soldiers, individuals that did not have visible wounds and did not recover from their symptoms were labelled as "neurasthenic" (Jones et. al., 2007). Soon after the United State entered World War I, similar symptoms were observed in American soldiers.

Following World War I, many psychiatrists attempted to translate the clinical skills they gained during the war to working with the general public. Although the majority of psychiatrists were unsuccessful in impacting the field, Abram Kardiner was able to incite some changes (van der Kolk, 2007). Kardiner was one of Freud's students, and after treating veterans of World War I he tried to develop a theory on *war neurosis* that fit with psychoanalysis. Many of the symptoms that he made note of to characterize war neurosis are still highly relevant to the diagnostic criteria that we use for posttraumatic stress disorder today. Kardiner documented on symptoms that he labelled as "physioneurosis," which is nearly synonymous to the current symptom of physiological hyper-arousal (Kardiner, 1941 as cited in van der Kolk, 2007). He also made note of many of the re-experiencing and numbing or avoidant symptoms of posttraumatic stress disorder. Some of the symptoms which he acknowledged include irritability or proneness to anger, becoming withdrawn or detached, and individuals feeling as if they were re-experiencing the trauma when triggered by a neutral stimuli (Kardiner, 1941 as cited in van der Kolk, 2007). He continued to conceptualize war neurosis based upon psychoanalytic theories and he

believed that those with the disorder were fixated on the trauma (Kardiner, 1941 as cited in van der Kolk, 2007). Despite psychiatrists working extensively with those with shell shock, much of the public was still sceptical of the diagnosis and believed soldiers were malingering (Ray, 2008).

2.3 World War II – Combat neurosis

During World War II, numerous names developed for what was previously labeled as shell shock even though each label was describing a very similar set of symptoms (Ray, 2008). Although having numerous names for one disorder could potentially result in confusion, it was seen as positive growth because it showed that multiple clinicians and researchers were coming to the same conclusion, that combat neurosis was a valid diagnosis. A new population of individuals suffering from similar symptoms – those who had survived the Nazi concentration camps, also expanded the professional understanding of combat neurosis. Observing concentration camp survivors brought Harry Abram to expand the concept of combat neurosis to a number of other trauma stricken populations which included: those under stress and those experiencing a life-threatening illness or an emergency situation (Abram, 1970 as cited in Ray, 2008). In his description, he suggested that the syndrome was comprised of both physical and psychological factors. An equal integration of both components was a novel argument because all past theories had put the primary emphasis on only one aspect without realizing the interplay between both components (Ray, 2008).

2.4 The diagnostic statistical manual, vietnam war, and posttraumatic stress disorder

In 1952, the first Diagnostic Statistical Manual included a diagnosis known as *stress response syndrome* (American Psychiatric Association, 1952 as cited in Lamprecht & Sack, 2002). The diagnosis was conceptualized as transient personality characteristic and was considered to be a normal reaction to extreme stress. Furthermore, with treatment, the symptoms were believed to subside once the ego regained balance (Lamprecht & Sack, 2002). The belief that people commonly recover from the syndrome was maintained despite multiple case examples to the contrary. The second Diagnostic Statistical Manual retained a very similar definition of stress response syndrome despite evidence demonstrating the need for adjustments (American Psychological Association, 1968 as cited in Lamprecht & Sack, 2002). It became a common belief among professionals that everyone had a breaking point, and that stress response syndrome was a normal response to an extreme stressor (Lamprecht & Sack, 2002).

Eventually the prevalence of soldiers who suffered from the chronic effects of stress response syndrome following the Vietnam War became undeniable. Vietnam veterans lobbied for compensation from the government for the trauma that they suffered (Lasiuk & Hegadoren, 2006). This forced the American Psychiatric Association to reconsider their conceptualization of the disorder, and in 1980 the term *posttraumatic stress disorder* was officially adopted into the Diagnostic Statistical Manual - III (American Psychiatric Association, 1980). In this version, posttraumatic stress disorder was defined by its' overt symptoms so that the characterization was not biased to a particular theory (Ray, 2008). In the revision of the Diagnostic Statistical Manual – III, they further refined the criteria for posttraumatic stress disorder. A distinction was made between common life stressors and a traumatic event, which was considered outside of the realm of normal human experience. Posttraumatic stress disorder was defined as having experienced a traumatic event, causing

marked distress and fear, helplessness, or horror (American Psychiatric Association, 1987 as cited in Lasiuk & Hegadoren, 2006). Civilian populations such as those who suffered child abuse, sexual abuse, and intimate partner violence were also included under the diagnosis. Extreme changes were made in the diagnostic criteria of posttraumatic stress disorder in the Diagnostic Statistical Manual – IV, which closely resembles the diagnostic criteria that we follow today in the revised version.

2.5 Current definition of posttraumatic stress disorder

Posttraumatic stress disorder is currently defined by the Diagnostic Statistical Manual – IV Text Revision as an anxiety disorder resulting from exposure to a traumatic event involving personal or secondary threat to life or wellbeing and causing intense fear, helplessness, or horror (American Psychiatric Association, 2000). Posttraumatic stress disorder is characterized by physiological hyper-arousal, avoidance of stimuli that would provoke anxiety or general emotional numbing, and recurrence of psychologically re-experiencing aspects of the trauma.

2.6 Potential diagnostic statistical manual – v changes

It is apparent that the definition of posttraumatic stress disorder has evolved throughout the years. As such, it is to be expected that the Diagnostic Statistical Manual criteria will continue to be adapted as we learn more about the disorder. Some of the proposed changes for the Diagnostic Statistical Manual - V criteria will be presented in this section. Currently, none of the changes presented here have been officially accepted. Upon publication of the Diagnostic Statistical Manual - V, readers should reevaluate the proposed changes that have been presented in this chapter. It is not expected that the current proposal will severely alter the prevalence rates of posttraumatic stress disorder or severely impact how clinicians evaluate or treat the disorder (Frueh et. al., 2010).

The Diagnostic Statistical Manuel – IV – TR criterion for posttraumatic stress disorder specifies three symptom clusters: re-experiencing, avoidance or emotional numbing, and hyperarousal (American Psychiatric Association, 2000). These symptoms arise from primary or secondary exposure to a traumatic event that evokes feelings of extreme horror, fear, or helplessness. Re-experiencing is described as having nightmares, intrusive memories, feeling as if the event were reoccurring, and experiencing psychological and/or physiological distress when encountering internal or external reminders of the trauma. Avoidance or emotional numbing is defined as trying to avoid thoughts or feelings about the trauma, trying to avoid people or places that serve as reminders of the trauma, impaired memory for the trauma, feeling detached from others, having a sense of a foreshortened future, restricted affect, and anhedonia. Finally, hyper-arousal is defined as difficulty sleeping, irritability or anger, difficulty concentrating, hyper-vigilance, and exhibiting an exaggerated startle response. In order to receive a diagnosis of posttraumatic stress disorder, an individual must exhibit one re-experiencing symptom, three avoidance or emotional numbing symptoms, and two hyper-arousal symptoms. The symptoms must be present for over one month following the traumatic event and must cause impaired functioning or distress. If the symptoms have been apparent for less than three months the posttraumatic stress disorder is labeled as acute, but if present for over three months, the label is then changed to chronic posttraumatic stress disorder. The criteria for the Diagnostic Statistical Manual – IV – TR (current edition) and the proposed changes for the Diagnostic Statistical Manual – V can be seen in the table below (figure 1).

Diagnostic Statistical Manual – V	Diagnostic Statistical Manual – IV – TR
A. The person was exposed to one or more of the following event(s): death or threatened death, actual or threatened serious injury, or actual or threatened sexual violation, in one or more of the following ways:	A. The person has been exposed to a traumatic event in which both of the following were present:
1. Experiencing the event(s) him/herself	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 self or others
2. Witnessing, in person, the event(s) as they occurred to 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
3. Learning that the event(s) occurred to a close relative or close friend; in such cases, the actual or threatened death must have been violent or accidental	
4. Experiencing repeated or extreme exposure to aversive details of the event(s) (e.g., first responders collecting body parts; police officers repeatedly exposed to details of child abuse); this does not apply to exposure through electronic media, television, movies, or pictures, unless this exposure is work related.	
B. Intrusion symptoms that are associated with the traumatic event(s) (that began after the traumatic event(s)), as evidenced by 1 or more of the following:	B. The traumatic event is persistently reexperienced in one (or more) of the following ways:
1. Spontaneous or cued recurrent, involuntary, and intrusive distressing memories of the traumatic event(s). **Note:** In children, repetitive play may occur in which themes or aspects of the traumatic event(s) are expressed.	1. Recurrent and intrusive 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 in which the content and/or affect of the dream is related to the event(s). **Note:** In children, there may be frightening dreams without recognizable content.	2. Recurrent distressing dreams of the event. **Note:** In children, there may be frightening dreams without recognizable content.
3. Dissociative reactions (e.g., flashbacks) in which the individual feels or acts as if the	3. Acting or feeling as if the traumatic event were recurring (includes a sense of reliving

traumatic event(s) were recurring (Such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings.) **Note:** In children, trauma-specific reenactment may occur in play.	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 or prolonged psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event(s)	4. Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
5. Marked physiological reactions to reminders of the traumatic event(s)	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 traumatic event(s) (that began after the traumatic event(s)), as evidenced by efforts to avoid 1 or more of the following:	C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:
1. Avoids internal reminders (thoughts, feelings, or physical sensations) that arouse recollections of the traumatic event(s)	1. Efforts to avoid thoughts, feelings, or conversations associated with the trauma
2. Avoids external reminders (people, places, conversations, activities, objects, situations) that arouse recollections of the traumatic event(s).	2. Efforts to avoid activities, places, or people that arouse recollections of the trauma
D. Negative alterations in cognitions and mood that are associated with the traumatic event(s) (that began or worsened after the traumatic event(s)), as evidenced by 3 or more of the following: **Note**: In children, as evidenced by 2 or more of the following:	
1. Inability to remember an important aspect of the traumatic event(s) (typically dissociative amnesia; not due to head injury, alcohol, or drugs).	3. Inability to recall an important aspect of the trauma
2. Persistent and exaggerated negative expectations about one's self, others, or the world (e.g., "I am bad," "no one can be trusted," "I've lost my soul forever," "my whole nervous system is permanently ruined," "the world is completely dangerous").	4. Markedly diminished interest or participation in significant activities
3. Persistent distorted blame of self or others about the cause or consequences of the traumatic event(s)	5. Feeling of detachment or estrangement from others

4. Pervasive negative emotional state -- for example: fear, horror, anger, guilt, or shame	6. Restricted range of affect (e.g., unable to have loving feelings)
5. Markedly diminished interest or participation in significant activities.	7. Sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span)
6. Feeling of detachment or estrangement from others.	
7. Persistent inability to experience positive emotions (e.g., unable to have loving feelings, psychic numbing)	
E. Alterations in arousal and reactivity that are associated with the traumatic event(s) (that began or worsened after the traumatic event(s)), as evidenced by 3 or more of the following: **Note**: In children, as evidenced by 2 or more of the following:	D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:
1. Irritable or aggressive behavior	1. Difficulty falling or staying asleep
2. Reckless or self-destructive behavior	2. Irritability or outbursts of anger
3. Hypervigilance	3. Difficulty concentrating
4. Exaggerated startle response	4. Hypervigilance
5. Problems with concentration	5. Exaggerated startle response
6. Sleep disturbance -- for example, difficulty falling or staying asleep, or restless sleep.	
F. Duration of the disturbance (symptoms in Criteria B, C, D and E) is more than one month.	E. Duration of the disturbance (symptoms in Criteria B, C, and D) is more than 1 month.
G. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.	F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
H. The disturbance is not due to the direct physiological effects of a substance (e.g., medication or alcohol) or a general medical condition (e.g., traumatic brain injury, coma).	
Specify if: *With Delayed Onset:* if diagnostic threshold is not exceeded until 6 months or more after the event(s) (although onset of some symptoms may occur sooner than this).	*Specify* if: *Acute***:** if duration of symptoms is less than 3 months *Chronic***:** if duration of symptoms is 3 months or more *With Delayed Onset:* if onset of symptoms is at least 6 months after the stressor

Fig. 1. DSM IV – TR (American Psychiatric Association, 2000) and Proposed Criteria for the DSM – V (American Psychiatric Association, 2010 obtained 7.6.11 from http://www.dsm5.org/ProposedRevisions/Pages/proposedrevision.aspx?rid=165#)

The first change that was proposed is meant to give more clarity to what qualifies as a traumatic event. The current criteria states that the person must have both "experienced, witnessed, or [been] confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others" and "the person's response involved intense fear, helplessness, or horror" (American Psychiatric Association, 2000). In order to provide more clarity, it has been proposed that a traumatic event should be constituted by actual or threatened: death, serious injury, or sexual violation. In addition, the individual must have either personally experienced the traumatic event, witnessed it in person, heard about it happening to a close friend or relative (where death or threatened death must be either violent or accidental), or have had one extreme exposure or repeated exposures to the unpleasant details of the event (American Psychiatric Association, 2010). A criticism of the current definition is that it could be interpreted that witnessing the media's portrayal of a situation would qualify as a traumatic event, although not all clinicians would endorse this interpretation. Therefore, it has been proposed that the Diagnostic Statistical Manuel - V prohibit that a media portrayal of an event qualify as traumatic unless the exposure is work related. The new definition also removes the qualifier that the person must react with intense fear, helplessness, or horror. One of the arguments for this change is that the current definition does not allow for individual differences in how people respond to trauma. A contrary argument for removing this qualifier is that most people respond to trauma in a manner that is consistent with the criteria.

In a worldwide sample of 28,490 participants who experienced a potentially traumatic event, only 1.4% of participants did not respond to the event with intense fear, helplessness or horror while meeting all other criteria for a diagnosis of posttraumatic stress disorder (Karam et. al., 2010). This study bolsters the argument that those who experience an event which results in them meeting all of the other criteria for the disorder will almost always respond with intense horror, fear, or helplessness. For that reason, including this additional criterion does not provide meaningful information. Those that do not meet the criteria may for some reason respond differently to trauma, but this would be something to be explored in therapy rather than addressed in their diagnosis. In addition, excluding this qualifier from the diagnostic criteria could reduce the amount of time it takes to assess for posttraumatic stress disorder.

Another relatively significant change that is being proposed is to use four symptom clusters to diagnose posttraumatic stress disorder instead of three. More specifically, it has been proposed to divide the avoidance and emotional numbing cluster into an avoidance cluster and separate cluster focusing on distorted thinking and negative emotions. In order to obtain a diagnosis using the divided clusters, an individual would need to avoid either internal or external reminders of the trauma. In addition they would need three symptoms from the distorted thinking and negative emotions cluster. This cluster would be comprised of seven symptoms which include: inability to remember the trauma, anhedonia, feeling detached, restricted affect, pervasive experience of negative emotions, distorted blame of self or others for the trauma, and persistent distorted negative thoughts about one's self, others, and the world. This change is being proposed because factor analysis has suggested that the current model does not account for all of the dimensions of posttraumatic stress disorder (Frueh et. al., 2010). More specifically this means that avoidance and emotional numbing are distinct concepts.

A problem that has emerged with the current diagnosis is that posttraumatic stress disorder symptoms overlap with many of the symptoms from major depressive disorder. Some of the

symptoms that overlap include: anhedonia, sleep problems, irritability, and difficulty concentrating. Due to the high comorbidity of posttraumatic stress disorder and major depressive disorder, some have suspected that counting the same symptom for both disorders accounts for much of the comorbidity rather than the disorders actually co-occurring. A study by Elhia and colleagues (2008) demonstrated that the disorders do in fact frequently co-occur because when the symptoms that overlap with depression and anxiety are removed, the lifetime prevalence figure for posttraumatic stress disorder only decreases from 6.81% to 6.42%. Removing the overlapping symptoms would cause some individuals to reach a subclinical level, but it is valuable to be aware that the disorders are in fact distinct. Concern about the overlap in criteria may become even more common because the proposed changes will make posttraumatic stress disorder less distinct from depression. It is being proposed that the current symptoms, which are very similar to those of major depressive disorder remain in the criteria. Furthermore, it has been proposed that additional criterion that also overlaps with major depressive disorder be added to the diagnostic criteria. It is unclear how this change will impact future comorbidity of posttraumatic stress disorder and major depressive disorder, which may prove to be a problem.

Additional changes that are being proposed include adding an extra symptom to the hyper-arousal cluster and removing the distinction between chronic and acute posttraumatic stress disorder (American Psychological Association, 2010). Other minor changes are being proposed to reword some of the criteria in order to provide clarity. Removing the distinction between chronic and acute posttraumatic stress disorder is being proposed because there is not enough evidence to show that they are separate concepts rather than just two separate time points on the same continuum. The additional symptom that may be added to the hyper-arousal cluster includes engaging self-destructive or reckless behavior. With the additional symptom, individuals would need to have three of six symptoms from the hyper-arousal cluster to receive a diagnosis.

We have clearly come a long way in our understanding of posttraumatic stress disorder throughout the years, but there are many aspects of the disorder that we still do not understand. As we learn more about the mechanisms of the disorder, the definition will continue to be adapted within the Diagnostic Statistical Manual.

3. Epidemiology

Prevalence estimates of posttraumatic stress disorder are important because they can be used to determine how to allocate resources for those affected by the disorder (Ramchand et. al., 2010). This section will present prevalence figures for the general population and the figures for the current and past wars, and will conclude with a discussion about the gender differences in posttraumatic stress disorder.

3.1 Prevalence

In the general population of the United States, posttraumatic stress disorder has been found to have a lifetime prevalence of 6.8 percent (Kessler et. al., 2005). Posttraumatic stress disorder is frequently seen in military personnel due to their elevated potential for exposure to trauma during combat. In the current war in Iraq and Afghanistan, the prevalence of posttraumatic stress disorder in soldiers post-deployment is believed to be between 10.3% and 17% (Sundin et. al., 2009). The prevalence of posttraumatic stress disorder for Vietnam Veterans ranges from 8.5% to 19.3% and between 1.9% and 24% for soldiers in the Persian

Gulf War (Sundin et. al., 2009). Prevalence figures vary widely in the military based upon a number of variables, such as how posttraumatic stress disorder was assessed, how much time has elapsed since the trauma, the level of combat exposure, the number of completed tours, gender, and the unit the individual was assigned to during deployment.

3.2 Gender differences in posttraumatic stress disorder in the military

Posttraumatic stress disorder has been known to develop following a broad range of traumatic situations. Due to this chapter's focus on combat related posttraumatic stress disorder this section will only present traumatic situations that are commonly experienced by those in the military. Some of the more common trauma experiences include: combat situations where the soldier felt as though their life was in danger or witnessed the death or threatened death of another person, seeing dead bodies or mutilated body parts during an assignment, or sexual assault while in the military. Men experience posttraumatic stress disorder as a result of combat situations more frequently than women because women are not permitted to have infantry positions. Women are more likely to experience sexual assault, which is unfortunately a frequent occurrence in the military (Williams & Bernstein, 2011).

3.2.1 Men in the military

Men in the military are vulnerable to an array of traumatic situations during combat. Individual differences exist and dictate whether a person has a heightened likelihood for developing posttraumatic stress disorder and how severe the stressor must be in order for them to develop the disorder (See the section on etiology for a more in depth discussion of individual differences in vulnerability for developing posttraumatic stress disorder). Some soldiers may be traumatized by just hearing the sounds of explosions due to a fear of being harmed by an explosive device. More resilient soldiers may obtain posttraumatic stress disorder from being involved an automobile accident while deployed or by being exposed to an explosive device that detonated near them or injured someone around them. Furthermore, they could be traumatized from engaging in hand-to-hand combat or in a firefight with the enemy. Finally, soldiers could be traumatized while retrieving severely injured soldiers or collecting bodies or body parts of soldiers who were killed in combat. Women in the military are also at risk for being involved in the previously mentioned traumatic situations, but have a decreased likelihood because their job assignments are intended to keep them away from direct combat. The list of potential combat scenarios provided is not meant to be all-inclusive, as there are a number of unpredictable situations in war that can cause a soldier to develop posttraumatic stress disorder.

3.2.2 Women in the military

Women in the military are thought to have an increased probability of experiencing a traumatic event during their service because of their ability to be sexually assaulted. Female soldiers and male soldiers placed in non-combat positions experience the same level of risk for encountering a traumatic event during deployment. Female soldiers are additionally at risk for being sexually assaulted by other soldiers (Williams & Bernstein, 2011). Although men are also sexually assaulted while in the military, women are more frequently assaulted. Lipari and Lancaster (2003) found that in active duty personnel, 3% of women have been sexually assaulted while in the military as compared to 1% of men. Furthermore, Sadler and colleagues (2003) found in a sample of 558 female veterans, 28% had experienced a rape or

an attempted rape while in the military, 8% experienced some form of sexual coercion, and 27% experienced unwanted sexual attention. The Department of Defense (2004) found that 71% of the women seeking treatment for posttraumatic stress disorder, who had served in the Vietnam War and subsequent wars, had been raped while in the military. Some of the risk factors that increase a female soldier's chance of being sexually assaulted include being between the ages of 17 to 24 years old, using alcohol, and past history of sexual assault (Williams & Bernstein, 2011).

4. Etiology

This section will discuss the mechanisms through which an individual develops posttraumatic stress disorder. Brief attention will be given to the nature of traumatic stressors and the linear progression from acute stress disorder to posttraumatic stress disorder. The primary emphasis of this section will be on the psychological and biological theories regarding what makes a person vulnerable to posttraumatic stress disorder and what maintains it after symptoms arise.

4.1 From trauma to acute stress disorder to posttraumatic stress disorder

Not every individual who experiences a traumatic event will subsequently develop posttraumatic stress disorder. As the name implies, posttraumatic stress disorder results from an experience with a traumatic stressor. Some of the stressors that can cause the disorder include: natural disasters, combat, sexual assault, physical assault, abuse or neglect as a child, car accidents, surgery, and witnessing something life threatening happen to a loved one. A person can develop posttraumatic stress disorder from a single stressor or may encounter multiple traumatic situations. An individual who encounters multiple events may either develop the disorder after the first event and the subsequent events then exacerbate their symptoms or they may develop the disorder only after experiencing multiple traumatic events.

As previously discussed, an individual must experience their symptoms for at least a month in order to receive a posttraumatic stress disorder diagnosis (American Psychiatric Association, 2000). Individuals with symptoms lasting less than a month are given an acute stress disorder diagnosis. Although everyone who has posttraumatic stress disorder has also had acute stress disorder, not everyone who experiences acute stress disorder will go on to develop posttraumatic stress disorder. The diathesis-stress model helps explain why some individuals do not develop the disorder after a traumatic experience (Elwood et. al., 2009). This model refers to the interaction between a person's environment (the severity of the stressors that they encounter) and their biological and psychological predispositions, which can create vulnerability for developing the disorder. Those with high diathesis only require a minimal stressor in order to develop the disorder, whereas someone with no diathesis may never develop the disorder even when presented with an extreme stressor. The next section will present the psychological and biological theories on the characteristics that may act as a diathesis for developing posttraumatic stress disorder.

4.2 Theories

It is important to understand some of the basic theories on posttraumatic stress disorder in order to appreciate how these theories have then been integrated into the current theories that are far more complex. This section will provide a brief introduction to stress response

theory, theory of shattered assumption, conditioning theory, and information-processing theory. This section will be followed by a discussion about some of the current psychological theories, including: emotional processing theory, dual representation theory, and Ehlers and Clark's (2000) cognitive theory on posttraumatic stress disorder. This section will conclude with a discussion on the biological correlates of posttraumatic stress disorder. It must be noted that the majority of the research on the biological aspects of posttraumatic stress disorder comes from correlational studies. Inferences cannot be made as to whether the biological abnormalities existed before the trauma and acted as a vulnerability for acquiring the disorder or developed after being exposed to the trauma.

4.2.1 Basic psychological theories

Stress response theory posits that a person develops posttraumatic stress disorder when they are unable to reconcile their beliefs about the world with what happened during the trauma (Horowitz 1976 & 1986 as cited in Brewin & Holmes, 2003). People have an internal working model of how the world operates and a traumatic experience often violates some of those core beliefs. When the individual is unable to logically integrate what happened to them within their world-view, defense mechanisms become activated to repress the trauma. The defense mechanisms at play are said to mimic many of the avoidance and numbing symptoms of posttraumatic stress disorder. Since a drive to reconcile the trauma with one's world-view still unconsciously exists, the person will experience intrusive reminders of the trauma to force them to cope with what happened. The individual will continue to experience these symptoms until they resolve the discrepancy, which is said to explain why some suffer from chronic posttraumatic stress disorder. Clearly, this theory is highly rooted in psychodynamic principles. Although it does not explain the full range of symptoms in those with posttraumatic stress disorder, stress response theory provided a framework for the theories that followed it.

The *theory of shattered assumptions* is very similar to stress response theory in that it places an emphasis on the individual's assumptions about the world. According to this theory, the assumptions that are said to be the most important to how a person responds to trauma include believing that: the world is a good place, what happens within the world makes sense, and that they are generally a good person and worthy of having good things happen to them (Janoff-Bulman, 1992). One of the initial assumptions of this theory was that those with the most positive beliefs about the world would also be the most severely impacted by trauma. Since this belief was disproved by the fact that previous trauma serves as a risk factor for developing posttraumatic stress disorder, the theory was revised to say that those who have previously been exposed to trauma have already had their view of the world shattered. Having this negative outlook makes them vulnerable for developing posttraumatic stress disorder in the future. Similar to stress response theory, this theory provides an incomplete rationale for all of the symptoms associated with posttraumatic stress disorder.

The *conditioning theory* of posttraumatic stress disorder is based upon Mowrer's two-factor learning theory (1960 as cited in Brewin & Holmes, 2003). The process of fear acquisition occurs when a traumatic experience is paired with a neutral stimulus, resulting in a fear response to the previously neutral stimuli. Once the neutral stimulus becomes a conditioned stimulus, the person begins to generalize their fear to other situations (Keane, et. al., 1985 as cited in Brewin & Holmes, 2003). Using a behavioral framework, individuals with posttraumatic stress disorder should habituate to their feared stimuli due to the re-

experiencing symptoms of the disorder. Individuals with the disorder do not habituate because once they begin re-experiencing the trauma they then engage activities that are consistent with the avoidance or numbing symptoms of the disorder. Since their distress subsides, they are then reinforced to continue engaging in avoidance and numbing tactics to cope with the trauma. Although this theory is highly useful for explaining posttraumatic stress disorder in many ways, it has been criticized because it is missing the cognitive component of the disorder. The cognitive component is important because it is often necessary for explaining individual differences in acquisition of the disorder (Brewin & Holmes, 2003).

Information processing theory integrates the cognitive components of the disorder into conditioning theory (Lang et. al., 1979 as cited in Brewin & Holmes, 2003). The general assumption of this theory is that when a person has a traumatic experience, the memory is stored differently than those from normal experiences. Posttraumatic stress disorder is then the result of a memory not being processed correctly. Information processing theory focuses solely on the cognitive components of the trauma and does not broadly integrate the social and personal context of the event. The memory of the trauma is comprised of: the surroundings during the trauma, other concrete aspects of the event, the person's physical and emotional reactions, and their assessment of the event. The consolidation of the experience, including all of the aforementioned components into a memory is called a *fear network.* Subsequently, when an individual is exposed to something that resembles an aspect of the fear network, the entire network then gets activated which triggers the same emotional response that was experienced during the trauma. An example of the fear network is a soldier ducking to the ground in fear when he hears a balloon pop because he was traumatized after witnessing an explosion while in combat. In this example, a loud noise, feeling fearful, and ducking to the ground, all are a part of the soldier's fear network. Simply hearing a sound that was similar to an explosion was sufficient to trigger the entire fear network.

Edna Foa added to this theory by explaining that what separates posttraumatic stress disorder from other anxiety disorders is that a traumatic event causes the person to question their basic assumptions about their personal safety in a global manner (Foa et. al., 1989 as cited in Brewin & Holmes, 2003). Since their assumptions about safety have been violated, their threshold to activate the fear network is low. In addition, because the individual does not feel safe, they are much more aware of their surroundings causing a reciprocal relationship between the decreased threshold and their sense of safety. An individual can reintegrate the different components of their fear network back into a normal memory if they are exposed to those components in a way that teaches them to that they are not actually in danger. This concept will re-visited and elaborated upon in the therapy section regarding Prolonged Exposure.

4.2.2 Contemporary psychological theories

Emotional processing theory is based on information processing theory, but takes into consideration individual perceptions before, during, and after the trauma (Foa & Riggs, 1993 as cited in Brewin & Holmes; Foa & Rothbaum, 1998 as cited in Brewin & Holmes). Furthermore, this theory proposes that those with more rigid views before the trauma will have worse outcomes following the experience. Having an extremely positive view or extremely negative view pre-trauma is considered a risk factor for developing posttraumatic

stress disorder. Positive views include believing the world is very safe or the person thinking they are completely capable of dealing with stress. Negative views would include believing that the world is a bad place or that bad things always happen to them. When an individual with rigid negative views of the world encounters a traumatic situation, it confirms that their views of the world were accurate. Therefore, an individual's outlook before experiencing trauma can impact how they perceive the event while it is happening and how they reflect on what happened. This theory is clinically relevant because if during treatment, an individual can be repeatedly re-exposed to the traumatic experience they can habituate to the feared stimulus and may reevaluate and hopefully reconsider how they reflect on the trauma. A client can be re-exposed to the trauma in session by either asking the client to imagine the experience or ask them to have real life encounters with innocuous situations that remind them of the memory.

Dual representation theory, as its name implies, makes the assumption that people store memories in two distinct ways (Bewin et. al., 1996 as cited in Brewin & Holmes, 2003). More specifically, memories tied to emotionally traumatic situations are stored differently than those from every day occurrences. Memories can either be stored as *verbally accessible* or *situationally accessible*. A verbally accessible memory is one that can be intentionally retrieved. A situationally accessible memory cannot be recalled at will, and can only be triggered by perceptual reminders of the trauma, such as sights, sounds, or physiological responses. When a memory becomes pathological, it is because it has become dissociated from being verbally accessible and is only situationally accessible. In addition, only primary emotions are stored in situationally accessible memories, such as fear, hopelessness, or horror. In order to transform a traumatic memory into a normal one, the individual must learn to express the traumatic situation verbally as though it were regarding a daily occurrence. This changes the emotions associated with the situational memory from negative emotions to positive ones due to the continued pairing of positive emotions with the memory.

Ehlers and Clark (2000) proposed a *cognitive model of posttraumatic stress disorder*, which highlights the discrepancy of the disorder from other anxiety disorders. This is because individuals who develop posttraumatic stress disorder perceive a current threat from a past event instead of a future event. Furthermore, this theory suggests that what distinguishes those who develop posttraumatic stress disorder from those who experience trauma but do not develop the disorder, is whether they equate experiencing past trauma to also having an increased susceptibility to future danger.

Ehlers and Clark's model proposes that there are multiple negative appraisals that people can make after experiencing a traumatic event that may lead to the belief that there is also a current threat for danger. The content of the appraisals include an individual's beliefs regarding: the fact that the event occurred and that it happened to them, their behavior and emotions during the trauma, the meaning of initial occurrence of posttraumatic stress disorder symptoms and the chronic symptoms (such as re-experiencing, emotional numbing, and concentration problems), the positive and negative reactions of other's to the trauma, and the physical or global consequences of the trauma (Ehlers & Clark, 2000, pg. 322). Many of the negative appraisals that can lead to posttraumatic stress disorder contain themes about the individual assuming personal responsibility for the trauma, believing that others perceive the event as their fault, and assuming that their cognitive and emotional responses to the trauma are going to be permanent. Since individuals with posttraumatic

stress disorder often assume personal responsibility for the trauma by attributing it's occurrence a personal deficiency, they also overestimate the likelihood of something dangerous happening again.

Ehlers and Clark's theory adopts some of the same concepts from dual representation theory and posits that a pathological memory contains only the sensory and emotional aspects of the event. Since the individual has not integrated the memory into their autobiographical memory, they are unable to provide all of the details of the event on cue. Remembering the details of the event may buffer from having unwanted recollections by providing context for memory. The chronological details of the event are also important because those with posttraumatic stress disorder may not be consciously aware of all of the precursors of the event, but can still be triggered by a stimulus that preceded the trauma. These individuals may also show biased attention for the negative aspects of what occurred before, during, and after the trauma. Furthermore, they often engage in behaviors that cause or exacerbate their symptoms, such as avoiding reminders of the trauma. Their avoidance often causes intrusive recollections, fails to give them the opportunity to disprove their beliefs about the trauma, and inhibits them from creating an autobiographical memory of the event. This theory provides the most integrative and detailed explanation of posttraumatic stress disorder and clearly incorporates many of the theories that preceded it. The theory's multifaceted explanation of posttraumatic stress disorder provides clinicians with a complex framework for viewing their clients. Due to the complexity of the theory, clinicians can choose which aspects are the most relevant to the cognitive distortions that they are seeing in their client.

4.2.3 Biological theories

In recent years, researchers have extended the biological theories on depression to posttraumatic stress disorder due to the comorbidity of both disorders. Kilpatrick and colleagues (2007) were one of the first research teams to generalize the genetic research on the serotonin transporter gene (5-HTTLPR) from depression to posttraumatic stress disorder. Previous research established that those with two short 5-HTTLPR alleles had a higher risk of developing depression than those with two long alleles or a combination of a short and long allele (Lesch et. al., 1996). The environment also plays a huge role in whether someone develops depression despite the genetic component of the disorder. Using this framework, Kilpatrick and colleagues (2007) investigated whether having two short 5-HTTLPR alleles increased the likelihood of developing posttraumatic stress disorder in participants who were exposed to hurricane Rita, which hit Florida in 2004. They found that low social support and high hurricane exposure proved to be risk factors for developing posttraumatic stress disorder. In addition individuals who had high levels of hurricane exposure, low levels of social support, and had two short alleles had a 4.5 times greater chance of developing posttraumatic stress disorder than the rest of the sample.

Research has also looked at monozygotic twins to examine the biological differences in a twin with posttraumatic stress disorder compared to their twin who does not have posttraumatic stress disorder. Pitman and colleagues (2006) examined twin pairs, where one twin obtained posttraumatic stress disorder through involvement in the Vietnam War and the other twin did not experience combat exposure or develop posttraumatic stress disorder. They found that the twin with posttraumatic stress disorder demonstrated higher heart rate reactivity to a startling noise than his brother. This response is thought to be in part the

result of hyperactivity in the amygdala. They also discovered that high-risk twin pairs often had some level of neurological dysfunction. The study inferred that this preexisting dysfunction might act as a vulnerability for developing posttraumatic stress disorder. When examining hippocampal volume using magnetic resonance imaging, they found that the twins with more severe posttraumatic stress disorder had a smaller hippocampus than average, but their twin brothers also had reduced hippocampal volume. Since this is a correlational study, the authors caution that more research is needed to draw causal inferences from these results.

Stress hormones such as cortisol have also been examined and found to correlate with posttraumatic stress disorder. A meta-analysis by de Kloet and colleagues (2006) concluded that those with posttraumatic stress disorder have lower baseline levels of cortisol than those without the disorder. Conversely, when exposed to a stressor, those with the posttraumatic stress disorder show an elevated cortisol response in comparison to those without the disorder. Although many theories have been proposed as to why this relationship exists, there is no conclusive evidence explaining why people with posttraumatic stress disorder have deceased baseline levels of cortisol, yet have an exaggerated stress response.

In accordance with the diathesis-stress model, both the psychological and biological theories on posttraumatic stress disorder should be taken into consideration because diathesis is comprised of both components. All of the contemporary theories on posttraumatic stress disorder are valuable to help conceptualize the disorder and no one theory has become dominant within the research. Each theory can be applied based on its relevance to a particular client.

5. Treatment

A number of treatments have been shown to be effective in treating posttraumatic stress disorder. Many of the treatments that are used for the disorder are rooted in cognitive behavioral therapy. This section will focus primarily on the treatments that have proven effective with those suffering from combat related posttraumatic stress disorder. The Veterans Administration in particular, has endorsed both cognitive processing therapy and prolonged exposure therapy (Karlin et. al., 2010). This section will also address a few of the more novel treatments for posttraumatic stress disorder such as the use of virtual reality and biofeedback. Some clinicians and researchers have recently incorporated virtual reality technology into prolonged exposure therapy. In addition, with the use of biofeedback, veterans can be taught to monitor their own physiological reactions, which are often elevated due to the hyper-arousal component of posttraumatic stress disorder.

5.1 Popular treatments for combat related posttraumatic stress disorder

This section will discuss cognitive processing therapy, prolonged exposure therapy, and the medications that can be used for individuals with posttraumatic stress disorder. An array of therapies exists for treating posttraumatic stress disorder and what is covered below should not be considered an all-inclusive list of the effective treatments.

5.1.1 Cognitive processing therapy

Cognitive Processing Therapy places an emphasis on the meaning that an individual assigns to their traumatic experience (Karlin et. al., 2010; Resick & Schnicke, 1992). The treatment is

divided into three phases and is typically administered over the course of 12 sessions. In addition, the treatment can be used in individual or group therapy. The three phases are comprised of: education, processing, and challenging. During the education phase, clients learn about the symptoms of posttraumatic stress disorder, how treatment will work, and is taught about the interaction between thoughts and feelings. They are also asked to consider how the event has impacted their outlook on the world. More specifically they are asked to examine the changes that may have occurred in their beliefs about themselves, others, and how the world operates. During the processing phase, the client is asked to either write about or discuss the traumatic event and work to identify thinking patterns that may be hindering their recovery. In the final phase of therapy, the challenging phase, the therapist works with the client to help them reframe their distorted beliefs about themselves, others, and the world. In doing this, the client develops a more balanced view of their environment.

5.1.2 Prolonged exposure therapy

Prolonged exposure therapy was designed specifically for individuals with posttraumatic stress disorder. The length of treatment typically ranges from 8 to 15 sessions, although it was initially designed to be 10 sessions (Foa & Kozak, 1986; Foa et al., 2007). This treatment draws from cognitive behavioral theories and it operates on the assumption that exposure to a feared stimulus will eventually extinguish the fear. During the first and second session, the primary focus is to provide psycho-education regarding the techniques that will be used, explain the rationale for using those techniques, and discuss the ways that people typically react to a traumatic event. Subsequent sessions will be dedicated to either imagery exposure or in vivo exposure. *In vivo exposure* is where the client goes out into the real world and encounters the feared object or situation in person with the goal of habituation. The in vivo scenarios that are used during treatment are low risk and are often commonplace experiences. These scenarios are appropriate for treatment because individuals with posttraumatic stress disorder will often avoid an array of low threat situations because they trigger unpleasant memories. *Imagery exposure* involves the person imagining the feared situation. More specifically, the client is prompted to talk about the most disturbing aspects of their trauma with the therapist. This gives them the ability to reprocess what actually happened and the opportunity to reorganize how they reflect on the traumatic event. The length of treatment depends on the client and is terminated when they no longer have symptoms that inhibit them from engaging in every day activities.

5.1.3 Medication

Due to the biological component of posttraumatic stress disorder, individuals who suffer from the disorder can also receive antidepressants to help ameliorate their symptoms. Medication can be used in conjunction with psychotherapy or can be used alone. Although a number of medications are currently being investigated for the treatment of posttraumatic stress disorder, the Food and Drug Administration has only approved two medications (Friedman & Davidson, 2007). Both of the medications that they approved, Sertraline and Paroxetine, are selective serotonin reuptake inhibitors. As we learn more about the biological mechanisms of the disorder the medications that are recommended for posttraumatic stress disorder will continue to change.

5.2 New treatments

Although there is limited research on novel treatments for posttraumatic stress disorder, some treatments are showing promising results. Two of those treatments include heart rate variability biofeedback training and virtual reality exposure therapy.

5.2.1 Heart rate variability biofeedback training

As mentioned in previous sections, hyper-arousal is one of the symptoms found in those with posttraumatic stress disorder. Persistent hyperarousal has been linked to physiological abnormalities such as increased blood pressure, exaggerated heart rate response to stressors, and an elevated resting heart rate (Cohen et al. 1997; Pitman et al. 1987). This has led researchers to speculate that posttraumatic stress disorder may alter sympathetic nervous system reactivity. In addition, researcher found that between 80% to 100% of individuals with posttraumatic stress disorder can be distinguished from those without by looking solely at their physiological reactivity (Orr & Roth 2000), which can be indicative of autonomic nervous system dysfunction. Heart rate variability can be used as an indicator of how the autonomic nervous system is functioning (Appelhans & Luecken 2006). Those with posttraumatic stress disorder typically have low heart rate variability (Tan et. al. 2011). Heart rate variability is the mean value of heart rate fluctuations over a period of time and is reflective of the interplay between the sympathetic and parasympathetic nervous system (Akselrod et al. 1981; Cohen et al. 1999). Research has established that by breathing at an ideal resonance frequency (approximately 5.5 breaths per minute), an individual can increase their heart rate variability (Vaschillo et al. 2002). Ideal resonance frequency varies by person.

Clients undergoing *heart rate variability training* are asked to first meet with the therapist to determine what breathing rate will produce their greatest heart rate variability (Lehrer et. al., 2000; Tan et. al., 2011). Clients are then instructed to practice breathing at this rate at home. They may either practice with a CD that guides them through the breathing techniques or they may be given a machine that notifies them when they are not breathing at their ideal rate. In a pilot study by Tan and colleagues (2011), participants who underwent eight, 30 minute training sessions experienced a significant reduction in posttraumatic stress disorder symptoms from pretest to posttest.

5.2.2 Virtual reality exposure therapy

Virtual reality exposure therapy has been used to treat soldiers that served in Vietnam, Operation Enduring Freedom, and Operation Iraqi Freedom. Computer programs were developed for both populations containing scenes that look similar to the surroundings veterans would have experienced during combat. The Vietnam virtual reality environment contains a scene with a virtual jungle and includes sounds of the jungle, gunfire, and nearby helicopters and has a separate scene within a helicopter (Gerardi et. al., 2010). *Virtual Iraq* was developed for veterans of current war. (A. A. Rizzo, et al., 2008). Virtual Iraq contains scenes of a Middle Eastern themed city, where the person is able to travel through the city by foot or in a truck. This environment can be adapted based on the client's therapeutic needs. In addition to the virtual reality scene, the individual is also presented with auditory, tactile, and olfactory stimulation. The client sits on a platform equipped with subwoofers, and the therapist controls which sounds the client hears. Furthermore, the platform vibrates in coordination with the virtual reality environment. The

clinician also controls the smells that are emitted from the "olfaction box" which includes various scents such as: burning rubber, body odor, and gasoline. Since all of these stimuli are presented simultaneously, it increases the reality of the virtual environment (A. A. Rizzo, et al., 2010).

Individuals undergoing treatment with the Virtual Iraq technology typically come in twice a week for 90 minutes over the course of five weeks (A. A. Rizzo, et al., 2008). The initial sessions are dedicated to identifying the details of the traumatic event and teaching the client stress management techniques such as deep breathing. They are also taught how to use the technology and to rate their distress so that it can be used as a reference throughout treatment. In a study on the efficacy of this treatment modality, Reger and Gahm (2008) found that patient's PTSD Checklist score decreased by approximately 50% post-treatment and they also showed a significant functional improvement. A major criticism of this type of therapy is the cost of the technology. Although this complaint is justified, virtual reality may prove to be a very valuable tool for clinicians that can afford to use it.

6. Traumatic brain injury

Posttraumatic stress disorder has been regarded as a signature wound of the wars in Iraq and Afghanistan. Of equal importance is the fact that *traumatic brain injuries* have been called the other signature wound of the wars (National Council on Disability, 2009). This section will focus on traumatic brain injuries due the pronounced overlap of this medical condition with posttraumatic stress disorder in soldiers returning from Iraq and Afghanistan. During the current wars, it is believed that up to 23% of soldiers have obtained a traumatic brain injury during deployment (Terrio et. al., 2009). In addition, 5% to 7% of Operation Enduring Freedom and Operation Iraqi Freedom soldiers are thought to have a probable comorbidity of posttraumatic stress disorder and a traumatic brain injury (Carlson et. al., 2011). Hoge and colleagues (2008), using a sample of 2525 Army infantry soldiers, found that 43.9% of soldiers who had lost consciousness after experiencing trauma to the head met criteria for posttraumatic stress disorder three to four months after returning from Iraq. Furthermore, 27.3% of the soldiers who solely experienced altered consciousness following a trauma to the head also met criteria for posttraumatic stress disorder. Although this is a biased sample due to an Army infantry soldiers' disproportionately high levels of combat exposure, it highlights the clear overlap of posttraumatic stress disorder and traumatic brain injury.

A traumatic brain injury diagnosis is given when an individual experiences an external disturbance to the head, resulting in trauma to the brain, and causing a lack of consciousness or diminished cognitive capacity (Department of Defense, 2007). A traumatic brain injury diagnosis is categorized in terms of severity and labeled as mild, moderate, or severe. In 2009, of the soldiers diagnosed with a traumatic brain injury, 78.4% of the cases were classified as a mild traumatic brain injury (Levin, 2010). A mild traumatic brain injury is defined as experiencing trauma to the head that causes a loss of consciousness for less than 30 minutes and an alteration of consciousness or mental state and posttraumatic amnesia for less than 24 hours (Department of Defense, 2007).

Soldiers in the current war frequently come into contact with explosive devices and as a result can obtain a traumatic brain injury in three ways (Department of Defense, 2007). A

soldier is said to have a *primary blast injury* when they were close enough to an explosion to experience the extreme changes in atmospheric pressure, otherwise known as a "blast wave." A blast wave can easily permeate a combat helmet and can ultimately cause trauma to the brain. A *secondary blast injury* can be obtained when a fragment from the explosion hits the soldier on the head hard enough to cause brain injury symptoms. This type of injury can be external but may also permeate the skull. Lastly, a soldier is said to have obtained a *tertiary blast injury* when an explosion causes the soldier to either be knocked to the floor or into something resulting in trauma to the head.

Despite the high comorbidity, researchers continue to struggle to detangle the overlap of symptoms between posttraumatic stress disorder and traumatic brain injury. The residual symptoms that one experiences as a result of a traumatic brain injury are called *postconcussive symptoms*. Many of the symptoms associated with posttraumatic stress disorder overlap with postconcussive symptomology, which include irritability, memory deficits, sleep problems, and difficulty focusing attention (Kennedy & Moore, 2010). Some of the symptoms that can often be unique to a traumatic brain injury diagnosis include balance problems, dizziness, and headaches (Kennedy & Moore, 2010).

Brenner and colleagues (2010) examined the unique contribution of posttraumatic stress disorder and traumatic brain injury to a sample of injured Army personnels' endorsement of postconcussive symptoms (headache, dizziness, memory problems, balance problems, irritability). They concluded that soldiers with either posttraumatic stress disorder or a traumatic brain injury endorsed more postconcussive symptoms than those without a diagnosis. Those with both posttraumatic stress disorder and a traumatic brain injury endorsed more symptom prevalence than those with a single diagnosis. Although it is noteworthy that a comorbid posttraumatic stress disorder and traumatic brain injury diagnosis can increase postconcussive symptomology, it is also important to recognize that the co-occurrence of either disorder can reciprocally exacerbate the other (King 2008).

Researchers have speculated that standard treatments for posttraumatic stress disorder could be less effective when a comorbid traumatic brain injury diagnosis exists (Bryant, 2001; Carlson et. al., 2011). This is solely speculation because there has been limited research to explore the efficacy of current treatments for those with this comorbidity. King (2008) suggests that early education about postconcussive symptomology and an explanation of the reciprocal relationship of the co-occurrence of posttraumatic stress disorder and traumatic brain injury can aid in proper detection and treatment. It is important for further research to explore the effectiveness of treatment for those with a comorbid diagnosis due to the high prevalence of soldiers who suffer from the co-occurring disorders. In addition, it is important for clinicians to be aware that the presence of a mild traumatic brain injury in a patient with posttraumatic stress disorder may make recovery from the posttraumatic stress disorder more challenging (Chard et. al., 2011).

7. Conclusion

Throughout the years we have gained a far better understanding of posttraumatic stress disorder. We have refined our diagnostic criteria for the disorder and developed more complex theories for understanding its' etiology. With the high prevalence of soldiers who are affected by posttraumatic stress disorder, it is important that we continue to refine our

understanding of the disorder so that can continue to improve the therapeutic techniques we are using to treat veterans who suffer from the disorder. Future research should examine how the common comorbidities of posttraumatic stress disorder, such as traumatic brain injuries impact treatment outcomes.

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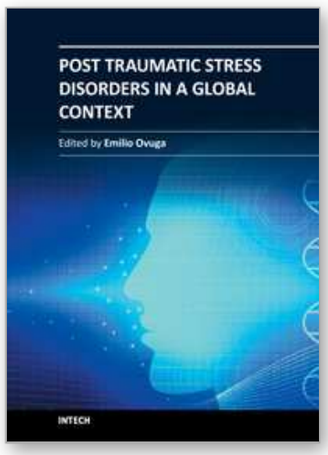

Post Traumatic Stress Disorders in a Global Context

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If, as a health care or social service provider, one was called upon to help someone who has experienced terror in the hands of a hostage taker, an irate and chronically abusive spouse or parent, or a has survived a motor vehicle accident, landslide, earthquake, hurricane or even a massive flood, what would be one's priority response? What would be considered as the most pressing need of the individual requiring care? Whatever the answer to each of these questions, people who have experienced terror, suffer considerable psychological injury. Post-Traumatic Stress Disorder in a Global Context offers some answers to meet the needs of health care and socials service providers in all settings, whether in a hospital emergency room, at the war front, or natural disaster site. The take home message is, after providing emergency care, there is always a pressing need to provide mental health care to all victims of traumatic stress.

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