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Vicarious Trauma: Possible Biopsychological Effects

John Newman Raymond

Walden University

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Abstract

Vicarious trauma (VT) affects many professions in our society (Trippany, White Kress, & Wilcoxon, 2004). There is no research, this author could find, as to the biopsychological effects of VT on patients. There is a body of research on the biopsychological effect of posttraumatic stress disorder (PTSD). This report indicates some of the biopsychological effects on the brain and the emotional response from PTSD. Since VT symptoms are related to PTSD (Byrne, Lerias, & Sullivan, 2006), a comparison of VT affects to posttraumatic stress syndrome affects might indicate similar structure and function deficits of the brain. This report discusses some of the research conducted concerning the above listed issues.

Vicarious Trauma: Possible Biopsychological Affects

Posttraumatic stress disorder (PTSD) is a well-defined symptom pattern of poor memory and hyperarousal. This paper will discuss the biopsychological implication of PTSD, the affect from PTSD, and affect reports of vicarious trauma to suggest that vicarious trauma may have similar brain structure and function deficits as found in PTSD.

Biopsychological Implications of Post Traumatic Stress Disorder

The hippocampus “sea horse” (Pinel, 2006, p. 69) is well known as an essential component of memory (Tonegawa, 2005), in particular, spatial, declarative and temporal memory.

Tonegawa suggests the hippocampus also distinguishes “pattern separation”. Pattern separation is being able to separate patterned events from time of event. If a person gets an apple every morning, pattern separation assists in the ability to distinguish today’s apple acquisition from yesterday’s apple acquisition. The hippocampus is also responsible in the memory of location. Instruments used in assessing the functioning of the hippocampus are the Morris water task and the virtual Morris water task (Astur et al., 2006; Tonegawa, 2005). The Morris water task places a subject in water that has a hidden submerged platform—situating the room with cues such as lights, desk, and doors. The subject swims until locating the platform. When the subject returns the next day, swim time is reduced in normal hippocampal function. With an impaired hippocampus, the subject will not find the platform as quickly.

Many studies suggest a size reduction of the hippocampus in patients with post-traumatic stress disorder (PTSD). Li et al. (2006) studied 24 Hunan, Chinese who experienced a recent horrific fire in November of 2003. Using magnetic resonance imaging (MRI) and proton magnetic resonance spectroscopy (H-MRS), Li et al. determined in the left hippocampus a decreased ratio of N-acetyl aspartate (NAA) to creatine (Cr) in participants with PTSD. This

reduction is indicative of tissue deficit in the hippocampus. Li et al. suggest the reduction is due to the experience of trauma from the fire. If what Li et al. suggest is true, one would imagine a substance that if taken may reverse the damage. Paroxetine (Paxil) may be that substance.

Patients experiencing PTSD do have an increase in declarative memory and hippocampal volume while taking Paxil (Vermetten, Vythilingam, Southwick, Charney, & Bremner, 2003). It is interesting to note that the United States Food and Drug Administration cautions patients taking paroxetine to be aware of increase suicidation (Paroxetine hydrochloride (marketed, 2007)). This author suggests that with the increase in declarative memory comes with it an increase in awareness and exposure to overwhelming experiences. If the hippocampus does decrease in size from a traumatic event, this author suggests it may decrease to protect the organism from an overwhelming experience. Mifepristone is another drug that has been reported to reduce the effect of glucocorticoid (Mayer et al., 2006). Mifepristone is approved as an abortive drug and can pose a serious treat to life if purchased from anyone other than a health care provider (Mifeprex (mifepristone) Information, 2006). Another supporting factor in the reduced hippocampus volume with PTSD discussion is the issue of increased levels of glucocorticoids. Nelson (2000) presents an explanation of hippocampal cell death:

When GR [glucocorticoid receptors] are stimulated, this reduces NE [norepinephrine] uptake by neural cells, alters the capacity of the neuron to use glucose, increases the activity of excitatory amino acids (e.g. glutamate), and facilitates the action of Nmethyl-D-aspartate (NMDA) receptors that result in actions that are highly energydemanding. The result is to increase the vulnerability of the neuron to overstimulation that can result in cell death. These actions of glucocorticoids are seen most prevalently in the hippocampus. Although elevated glucocorticoids may initially help foster hippocampal

activity involved in memory storage, high or prolonged elevations in glucocorticoids have been associated with cell death, altered dendritic growth, and impairment in the cognitive functions served by the hippocampus and its related structures. (p. 170)

Interestingly, Madison (2005) reports the plasticity of postsynaptic receptors. Madison suggests the NMDA receptors retreat or diffuse from the membrane when the synapse is silenced. Any attempt to bring the NMDA receptor back on line has failed. Only by waiting about 20 minutes, will the NMDA receptors appear in the membrane again. This research suggests that it takes a lot of “stress” to bypass the protective nature of the NMDA receptors to cause cell death. One such case is in Cushing’s syndrome where there are marked increased levels of glucocorticoids (Newell-Price, Bertagna, Grossman, & Nieman, 2006). The size of the hippocampus is proportional to the levels of glucocorticoids: As Cushing’s symptoms increase so does the level of glucocorticoids, inversely as Cushing’s is treated and glucocorticoids level decrease, so increases the volume of the hippocampus (Starkman, Gebarski, Berent, & Schteingart as cited in Van De Kolk, 1996).

Several other studies suggest the identification of reduced hippocampal size from PTSD is not a result of trauma but a preexisting condition. Astur et al. (2006) assessed participants with PTSD using a “hippocampal-dependent task—the virtual Morris Water task” (p. 234) while monitoring brain function with a functional magnetic resonance imaging (fMRI) device. Astur et al. found the control group and the PTSD group to perform the task similarly—there was no significant difference. The researchers did find a significant difference in the hippocampus activation. Where the PTSD group had little activation, the control group had significantly more activation. Astur et al. reports this research may be limited in power with too few participants. Since the PTSD group could perform the task as well as the control group; Astur et al. suggest the

hippocampus may not be reduced in size because of PTSD. Yehuda (as cited in De Kloet, Joëls, & Holsboer, 2005) suggest that persons with PTSD may have increase sensitivity to cortisol thus suppressing the hypothalamus-pituitary-adrenal (HPA) function. Wright, Lightner, Harman, Meljer, and Conrad (2006) report a similar response to stress. What may be happening is that the elevated stress and hyperarousal may be a result of the amygdala. Amygdala activation suppresses the hippocampus. Moreover, Gilbertson et al. (2002) studied monozygotic twins. One of each of the twin pairs was a Vietnam combat veteran. The combat veteran who was diagnosed with PTSD had a smaller hippocampus. So did his twin brother. The other pair showed a significant larger hippocampus. Even with the small sample size of this study, it does add to the confusion about the effects of trauma on the hippocampus or the effects of a reduced hippocampal volume on trauma.

Knowledge of whether hippocampus volume reduction because of trauma or, inversely preexisting smaller hippocampus causes PTSD symptoms does not matter to the patient suffering from it. The fact that there is an issue with the hippocampus gives hope in that researchers can begin to study how to effectively bring the hippocampus to a optimal level concurrently with the patient being able to handle what memories may arise.

Working memory deficits seems to be another aspect of PTSD (Veltmeyer et al., 2006). PTSD slows down working memory. Veltmeyer et al. attributes this issue to underlying brain instability. However, reduced cerebral blood flow (CBF) does not seem to be the cause. Pagani et al. (2005) assessed cerebral blood flow during a memory presentation to train workers. The memories presented were actual events that happened with the participants. One event was seeing a person go under the train while the train was moving. The other memory presented was being assaulted by another person. “There was a significantly higher relative CBF distribution in

subjects experiencing assaultive trauma(A) as compared to those exposed to person-under-the-train accidents (Pagani et al., p. 363).” CBF was more prevalent in the right hemisphere where emotions are reported being processed. Schore (2002) also implicates the right hemisphere in processing emotions: “Early abuse negatively impacts the developmental trajectory of the right brain, dominant for attachment, affect regulation, and stress modulation...” (Schore, p. 9).

To further the discussion of severity implications of traumatic events, Diseth (2005) did a literature review of PubMed (1502 found) and PsychInfo (127 found) on *children, conversion disorder, and dissociative disorder*. Diseth suggests from the review that traumatized children “demonstrated some permanent neurochemical as well as functional and structural abnormalities in brain areas that are involved in the integrative process of cognition and memory” (p. 79). Furthermore, Pat-Horenczk et al. (2007), after surveying “695 Israeli high school students” (p. 76), report from the 7.6% PTSD probable rate that “girls reported greater severity of [PTSD] symptoms, whereas boys exhibited greater functional impairment in social and family domains” (p. 76). Gaining more insight into the prevalence of PTSD symptoms, Kastelan et al. (2007) presents a positive correlation of severity of hyperarousal symptoms with psychotic symptoms reporting a 20% rate of hallucinations and delusions from combat-related PTSD symptoms in 91 Bosnian males. It seems reasonable that while children may be presented with daily traumatic events such as the children in the Israeli study, it is not until there are severe conditions of hyperarousal, such as a personal threat (Pagani et al., 2005), that psychotic symptoms appear. In addition, combining past trauma with secondary trauma, Jenkins and Baird (2002) suggest that “counselors with interpersonal trauma histories scored higher on [Compassion Fatigue Self-Test]” (p. 423). The combination of past trauma with recurrent experience of other’s trauma is more difficult than therapists who do not report past personal trauma working with other’s

trauma. The preceding research indicates that PTSD may be a function of life's accumulation of different traumatic events and not just a single episodic traumatic event.

The discussion to this point has been the biopsychological implications of PTSD and possible effects of accumulations of trauma. It is appropriate; now to identify what constitutes PTSD affect. The *Diagnostic and Statistical Manual* (2000) (DSM4TR) specifies PTSD's affect:

The person's response to the event must involve intense fear, helplessness, or horror (or in children, the response must involve disorganized or agitated behavior) (Criterion A2). The characteristic symptoms resulting from the exposure to the extreme trauma include persistent reexperiencing of the traumatic event (Criterion B), persistent avoidance of stimuli associated with trauma and numbing of general responsiveness (Criterion C), and persistent symptoms of increased arousal (Criterion D). (p. 463)

The DSM4TR furthers the affective implications: "For children, sexually traumatic events may include developmentally inappropriate sexual experiences without threatened or actual violence or injury" (p.465). In addition, reexperiencing can be in the form of dissociating while reliving the event, having dreams of the event, and experiencing flashbacks. Avoidances can be in form of "numbing" or removing self from others who actively or passively remind them of the trauma. In addition, the DSM4TR list other conditions that may be associated with PTSD:

Avoidance patterns may interfere with interpersonal relationships and lead to marital conflict, divorce, or loss of job. Auditory hallucinations and paranoid ideation can be present in some severe and chronic cases. The following association with an interpersonal stressor (e.g. childhood sexual or physical abuse, domestic battering): impaired affect modulation; self-destructive and impulsive behavior; dissociative symptoms; somatic complaints; feelings of ineffectiveness, shame, despair, or hopelessness; feeling

permanently damaged; a loss of previously sustained beliefs; hostility; social withdrawal; feeling constantly threatened; impaired relationships with others; or a change from the individual's previous personality characteristics. (p.465)

Chronic pain may also be another affect from PTSD. Villano et al. (2007) reports that psychiatric outpatients with PTSD and severe chronic pain “have higher ratings of psychiatric distress and addiction severity, report more stressful life events, and have a lower ratings of PA [positive affect], social support, and confidence to cope with mental illness...” (p. 172). The co-occurrences of these two disorders seem logical. With the diagnosis of PTSD, one must behave in a withdrawn, shameful manner. Having little social support and under chronic stress may eventually portray as chronic pain. Even though Villano et al. may just be verifying criteria for a PTSD diagnosis, the researchers draw attention to possible missed diagnosis by practitioners focusing on one issue such as chronic pain. The application of Villano et al. to this report is the affect of chronic pain prevalence in patients with PTSD.

With the presentation of the affects from PTSD and the previously reported biopsychological implications, this author posits that when the conditions of experiencing and internalizing traumatic events as reported by other (vicarious trauma) may have the same biopsychological effects as a self-experienced traumatic event. Indicating a link from vicarious trauma exposure to biopsychological effects may be similar affect as a self-experienced traumatic event.

Vicarious Trauma's Affect

Vicarious trauma (VT) and secondary trauma are two different constructs, yet with similar components (Jenkins & Baird, 2002). Secondary trauma has been called “compassion fatigue” and involves a component of job fatigue. Jenkins and Baird suggest that a therapist

would score high on a burnout scale, such as the Maslach Burnout Inventory (MBI). Since this paper is focusing on trauma and not burnout, any further discussion will attempt to distinguish between trauma's effect and burnout.

Most of the research on vicarious trauma is on social workers and counselors. This report will attempt to highlight the affect of the study participants that reported trauma symptoms from working with clients who experienced trauma. While there are numerous variables that confound any affectual linkage of vicarious trauma to biopsychological implications, this author suggests studies to validate any conclusions made here.

Cunningham (2003) reports that clinicians who work with sexual abuse victims report more disturbances of personal worldview and safety than those clinicians working with cancer victims do. There were several confounding variables in this study: the years of counseling experience was high; counselors who worked with sexual abuse survivors probably treated their client more often than the counselors treating the cancer victims; sexual abuse is a human to human trauma; and clinicians treating cancer victims were seeing the trauma in real time with each treatment. There was also a high rate of sexual abuse survivors in the clinician population. The only affective conclusions from this study are that clinicians working with trauma victims do experience more negative influences and disruption of worldview.

Dunkley and Whelan (2006) study's "aim was to investigate both intrinsic (i.e. coping styles, personal trauma history) and external (i.e. access to supervision, the supervisory working alliance) influences on vicarious traumatization" (p. 454-455). Sixty-two phone counselors were surveyed. There were several high scores reported in "disruption in cognitive beliefs" (Dunkley & Whelan, p. 463). The researchers also report that the counselors who experienced

trauma in the past had higher levels of PTSD symptoms. The authors do not delineate what the symptoms were.

While most researchers found correlations of history to vicarious trauma, Schauben and Frazier (1995) did not. This study was more comprehensive than the previous two. Schauben and Frazier report that some of the emotions of the counselors working with survivors were “anger..., sadness, fear, helplessness, and powerlessness” (p. 57). They also, as the other two studies report, there was a change in worldview—less trusting. The authors further report the counselors’ experience with other community systems such as courts were frustrating which added to negative feelings. In addition, Trippany, White Kress, and Wilcoxon (2004) report that changes in beliefs is a major concern for counselors working with trauma survivors.

Several other groups of providers show some form of vicarious trauma; however, the researchers fail to give the emotional response of the providers. Nurses are also effected by others trauma (Nursing, 2007), but no affect is reported. Children of first responders are also reported to have PTSD symptoms, yet there are no specifications as to what the symptoms are (Duarte et al., 2006). Duarte et al. suggests that New York City firefighters’ children “6 months after the World Trade Center attack” (p. 310) express more symptomolgy than other first responder groups. The explanation given is the combination of living in the city and having a first responder as a parent created more symptoms. This author suggests it may be the result of immediate possible injury to self and the extreme nature of the attack that caused the increase in symptoms. Even the military reports increased rates of vicarious trauma in heath care worker from combat zones but do not give specific emotional responses (Kolkow, Spira, Morse, & Grieger, 2007). Many studies of vicarious trauma give few emotional responses but only

parameters of instruments that indicate symptomology are given. This author questions our society's ability to address this issue without discussing the feeling behind the symptoms.

Two studies do give more information behind the conclusion of vicarious trauma. Byrne, Lerias, and Sullivan (2006) report that the 2001-2002 New South Wales bush fires presented an opportunity to sample a near by population during and after viewing the event. "Participants with vicarious traumatization engaged in a larger range of precautionary behavior.... They experience greater distress when exposed to reminders of the event (Lerias & Sullivan, p. 174)." They also employed avoidance as a coping skill. In addition, Jacob and Veach (2005) report female partners of male survivors of child sexual abuse have similar affect of PTSD patients. Some of the affect listed are, social isolation, denial, escape behaviors, anger, powerlessness, impatience, numbness, loneliness, emotional disconnection, hurt, and shame. These emotions and responses to the trauma are similar to the criteria of PTSD.

Conclusion

The primary biopsychological effects of PTSD are poor spatial and declarative memory associated with the hippocampus and hyperarousal associated with the amygdala. The affects from PTSD are similar to the affects of those reporting vicarious trauma. This author suggests there may be similar brain structure and function of PTSD victims as compared to vicarious trauma victims as indicated by similar affective responses. Further research should be conducted to identify any correlations.

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