The Role of Hyperarousal in PTSD

By Lindsey Smaka

In psychotraumatology, the term ‘hyperarousal’ seems to get thrown around a lot. For those suffering with Posttraumatic Stress Disorder, this term might hold a myriad of meanings; it may represent a label for their fear, their inability to sleep, or their jumpiness. For counselors, hyperarousal might mean very little besides serving as a set of diagnostic criteria for clinical assessment. However, when examining the role of the hyperarousal from a neurological lens, this symptom cluster begins to take on a more profound meaning. Having a working knowledge of the role which hyperarousal plays in the development of PTSD may help inform clinicians in intervention and treatment; and a scholarly emphasis on research in this area may hold the key to preventing some clients from even developing PTSD in the first place.

Posttraumatic Stress Disorder is a largely heterogeneous condition, varying widely in its symptomatology from one individual to the next. Because so many symptoms can manifest with PTSD, symptomatology is generally organized into four criteria groups, or ‘clusters’. These clusters include: avoidance, intrusion/re-experiencing, negative mood and/or cognitions, and hyperarousal; each cluster houses a set of corresponding symptoms (APA, 2022). Among the symptom clusters, hyperarousal holds particular clinical significance.

The hyperarousal cluster includes, but is not limited to, symptoms of: irritability, aggression, impulsive behavior, hypervigilance, an exaggerated startle response, concentration difficulties, and insomnia/difficulty sleeping (Miles, et al., 2022). The DSM 5-TR explains: “PTSD is often characterized by a heightened vigilance for potential threats… Individuals with

PTSD may be very reactive to unexpected stimuli, displaying a heightened startle response, or jumpiness, to loud noises or unexpected movements" (APA, 2022). The effects of hyperarousal can be debilitating. Hypervigilance sufferers may exhibit fearful behavior, such as rarely leaving home, constantly scanning surroundings, or always sitting near an exit in order for a quick escape (Kaplan et al., 2022). Consistently emerging as the predominant symptom of PTSD, it's estimated that around 70% of PTSD cases are considered to be within the "hyperarousal subtype" (Weston, 2014).

In order to fully understand PTSD and the role which hyperarousal plays, one must first examine the biological underpinnings of the disorder. When working with a traumatized client, a counselor might find their client as seemingly 'stuck'. Neuroscientist, Peter Afford (2019) explains that when trauma is followed by extended periods of stress, neurochemical disruption occurs; serotonin levels drop, while noradrenaline and dopamine surge. He posits that this leads to, "the inhibition of the default mode network, which means that even the resting brain is in a more stressed state" (Afford, 2019). In fact, the neurological changes that can occur from psychological trauma are so profound, they often mirror those of traumatic brain injury. Dr. Dawn-Elise Snipes explains this phenomenon: "We actually see physiological changes; shrinkage of the hippocampus, and other physiological changes, as the result of environmental, situational, emotional trauma– not just actual traumatic brain injury, like from a concussion" (Snipes, 2022).

The human body's threat-response system is known as the hypothalamic pituitary adrenal axis, or, the HPA axis. When a threat is detected, the amygdala raises the alarm, activating the HPA axis, and kickstarting cortisol production. Cortisol and glucose flood the bloodstream, to aid in fight-or-flight reactions and suppress bodily systems (Snipes, 2022). This is the body's

normal response to threat, which has evolved to promote survival. However, when the HPA axis is in perpetual activation due to traumatic stress, the body goes into a state of hypocortisolism, which Snipes, (2022), describes as, "the body blocking cortisol from going through", which, "is actually a protective mechanism designed to conserve energy during threats that are beyond the organism's ability to cope". This blockage of cortisol causes other neurotransmitters and hormones to become imbalanced, particularly, norepinephrine, which exacerbates hyperarousal. Within the research literature, it's been shown that norepinephrine imbalance is linked with "hyperarousal, increased startle responses, and increased coding of memories" (Snipes, 2022).

Studies have suggested the use of specific anti-anxiety medications, such as the beta blocker, Propranolol, as an immediate, peritraumatic intervention may be successful in preventing the development of PTSD. (Marshall, et al., 2006). Research conducted by Ronzoni et al., (2016) saw that psychotropic drugs can prevent the amygdala from signaling the HPA axis to activate, thus preventing subsequent fight-flight response, and cortisol and norepinephrine imbalance. While certain medications may aid in ameliorating PTSD symptoms, experts warn that others should be avoided. Studies show that early use of benzodiazepines can contribute to chronic PTSD and is generally considered "unwise" (Patterson, 2006).

Outside of medication, several modalities have been tested for targeting hyperarousal symptoms. Cognitive Processing Therapy (CPT) is an evidence-based, manualized treatment for PTSD. Several studies have compared the efficacy of CPT and Prolonged Exposure (PE). In a study by Cox et al., (2021), it was found that, "overall differences between PE and CPT were minimal". While PE has shown impressive results in many studies, some experts warn against the use of PE, especially for those with severe hyperarousal, "If the brain becomes excitotoxic during stress, it inhibits learning and memory, so exposure therapies for these particular clients

may or may not be super helpful. If it's increasing the excitotoxic environment in their brain too much it can be dangerous, which is why exposure therapies need to be taken very seriously" (Snipes, 2022).

Additional therapies have also shown limited effectiveness in treating hyperarousal symptoms. One of which is the oft-celebrated cognitive behavioral therapy. Citing a 2004 study, Crawford et al., (2019), reports that, "41% of patients completing cognitive behavioural therapy (CBT) for PTSD continued to manifest clinically significant insomnia, anger and irritability, which may be explained by the persistence of hyperarousal". To treat lingering symptoms of hyperarousal, following an intervention for PTSD, adjunctive therapies may be necessary. This is where psychoeducation and collaboration may prove especially beneficial for clients. Educating clients on hyperarousal symptoms, and explaining how different modalities may help to treat specific symptoms, may serve to provide clients with the guidance necessary to inform their approach to treatment, and expectations for recovery.

A clinician's patience is invaluable when working with a traumatized client, and it's important to remember that, "it may take clients who have experienced trauma, who have high levels of cortisol, more time to master new skills, because it's harder for them to focus. That norepinephrine is focused on fight or flee, not learning, memory and concentration" (Snipes, 2022). This implies that PTSD interventions may require a longer duration than what is initially proposed or suggested by practitioners.

From what we've seen in the existing literature, hyperarousal is often the most influential and neurologically harmful component of PTSD. This cluster doesn't just represent symptoms, but radical biological changes to the human body and brain. By collaborating with professionals

in adjacent fields and specifically targeting the neurobiological mechanisms of hyperarousal, together, we may be able to effectively stop PTSD in its tracks.

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