PTSD, neurodynamics and memory

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PTSD has gained considerable attention over the past twenty-five years, and important questions of interest to the mental health community have arisen over this time. What makes some people develop Posttraumatic Stress Disorder (PTSD) while others do not? What factors contribute to the vulnerability of some and the resiliency of others? How have therapeutic approaches to PTSD changed? Is there a coherent understanding of how to help people who have been traumatised that goes beyond particular theoretical schools? Can new insights be gained from neuroscience and memory research that look for common denominators? Working within a three-phase approach as best practice for the treatment of PTSD, the focus of this article is on Phase Two—*Focused work on traumatic memories*. This phase forms the heart of the therapeutic effort and involves the integration of the dysregulated memory systems—both implicit and explicit. Prior to a consideration of the processes of memory, JOHN ARDEN examines the complex neurodynamics of PTSD, including the roles of the amygdala and the hippocampus, in order to identify the most efficacious therapeutic approaches.

The effects of trauma have always interested me, in part due to the traumatic experiences of my Armenian relatives. Those who escaped the genocide brought with them traumatic memories such as watching relatives murdered in front of them, narrowly escaping death with one eye, or being brutally raped. Why did some develop Posttraumatic Stress Disorder (PTSD) while others did not? What contributed to the vulnerability of some and the resiliency of others?

PTSD has gained considerable attention over the past twenty-five years and this has intensified since the Afghan and Iraq wars. Important questions of interest to the mental health community have arisen over this time. How have therapeutic approaches to PTSD changed? Is there a coherent understanding of how to help people who have been traumatised that goes beyond particular theoretical schools? Can new insights be gained from neuroscience and memory research that look for common denominators?

In order to consider these questions it is necessary to move beyond theoretical schools, and the vast marketplace of therapeutic 'clubs' and brand names, where much of the language used by each school is meaningful only to 'initiated members'. It is important to shed this clubhouse language, and my primary interest is in finding common factors (Arden & Linford, 2008a; 2008b).

During the past few years I have taken part in a 'Best Practices' research group for the Kaiser Permanente Medical Centers. Like many groups, we recommend a phased approach to helping people with PTSD. A three-phase approach for the treatment of PTSD has been endorsed by the *International Society for the Study of Trauma and Dissociation*. While Phases One and Three are not the focus of this article it is important to note what constitutes each phase.

Phase One, *Establishing safety stabilisation and symptom reduction*, is employed immediately after a traumatic event when victims are provided with support through a crisis intervention model (Slaikeu, 1990). Therapists can provide 'psychological first aid' to stabilise, calm and orient the victim. It is important to note that Critical Incident Debriefing administered immediately after the trauma is now considered counter-therapeutic (Barlow, 2010). Best practice in Phase One is viewed as providing some initial psychoeducational support. Support for this model comes from research with trauma victims who were administered the drug propranolol immediately after the trauma. Compared to controls, this group was less prone to develop PTSD due to a lowering of elevated stress hormones that allows for less traumatic memory to be encoded (Vaiva et al., 2003; Pitman, Sanders, & Zusman, 2002).

Once elevated, stress hormones drop to a new baseline, albeit higher than before the trauma. Phase Two of therapy, *Focused work on traumatic memories*, is the focus of this paper. It involves the integration of the dysregulated memory systems—both implicit and explicit. Emotional engagement with traumatic memories

is a critical part of recovery, whereas avoidance of engagement serves to maintain PTSD symptoms (Zoellner, Fitzgibbons, & Foa, 2001).

Phase Three of therapy is *Integration and rehabilitation*. This phase involves the movement toward long-term recovery and the development of resilience that can contribute to what Tedeschi (1999) refers to as 'posttraumatic growth'. The development of a sense of meaning and direction in life is paramount. The experience of traumatic event(s) necessitates posttraumatic growth—a broadening of one's sense of self, relationships, and philosophy of life. This 'self' organisation (borrowed from Complexity Theory) involves helping the client to acknowledge his or her connection to the world and depth of meaning.

Phase Two forms the heart of the therapeutic effort. The foundation for this phase is based on the integration of implicit and explicit memory systems. Before launching into a discussion on memory and therapeutic approaches let us first examine the neurodynamics of PTSD. From this framework we are able to identify the most efficacious therapeutic approaches.

Neurodynamic aspects

Over the past 15 years neuroimaging studies involving PTSD symptom provocation have identified some consistent findings including *reduced activity* in the left hemisphere, the dorsolateral prefrontal cortex (DLPFC), the hippocampus, and Broca's area. These studies have also shown *increased activation* in the parahippocampus gyrus, the posterior cingulate, and the amygdala (Bremner, 2002; Nutt & Malizia, 2004; Rauch, 1996).

The decreased activity in Broca's area appears to correspond with the difficulty in expressing a coherent narrative of the traumatic event (Hull, 2002). And the dysfunction of the DLPFC may be associated with problems in working memory, language, cognition and integration of verbal expression with emotions.

Failure to regulate the activity of the amygdala has been one of the most consistent findings reported for PTSD. There appears to be a range of hyperactivity of the amygdala based on the type of trauma. For example, the general pattern of amygdala activation for PTSD indicates that combat-exposed (PTSD) individuals experience the most heightened amygdala activity and for other types of trauma, less activation of

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Photo: A man is comforted after his home is destroyed by bushfires in the USA © Justin Sullivan, Getty Image News, Getty Images.

the amygdala than combat exposed individuals but much more than non-traumatized individuals (Britton, Phan, Taylor, Fig, & Liberzon, 2005).

Consistent with these findings are reports that PTSD is associated with a failure to activate the anterior cingulate cortex (ACC). At the same time, there is diminished medial prefrontal cortex (mPFC) activity in response to traumatic memories. Studies that utilised Positron Emission Tomography (PET) scans to measure patterns of neural activity associated with traumatic images and sounds, have shown decreased activity in the left prefrontal cortex (L-PFC) and anterior cingulate cortex (ACC) (Bremner et al., 1999).

Given that the ACC is important in monitoring emotional experience, and there is generally greater intensity of negative emotions associated with PTSD, this neurodynamic pattern may represent a failure of this region to exert appropriate top-down inhibition of the amygdala. Also, since the L-PFC is associated with positive emotion, there is a breakdown in monitoring and maintaining positive emotion. This picture, coupled with the studies that show increased amygdala and R-PFC activity (associated with negative emotion), reflects a failure to inhibit negative emotion. Accordingly, these dynamics underlie the hyperarousal symptoms of PTSD.

Hippocampal volume update

Beginning in the 1990s, studies of adults with PTSD have found reductions in hippocampal volume, i.e., the size of the hippocampus. Using MRI-based measurement of hippocampal volume, Bremner has shown reduced volumes for combat-related PTSD (Bremner, Krystal, Southwick, & Charney, 1995) and childhood physical and sexual abuse related PTSD (Bremner et al., 1997).

The initial explanatory model for reductions in hippocampal volume described how high levels of cortisol have been associated with a range of separate neuroanatomical effects on the hippocampus, including the impairment of synaptic plasticity, the inhibition of neurogenesis, and ultimately neuronal death (Sapolsky, 2003). In general, the hippocampal deficits occurring as a result of excitotoxicity and the cortisol cascade—retraction of dendritic processes, reduced synaptic plasticity, and the inhibition of neurogenesis—are thought to be potentially reversible (Sapolsky, 2003; Alderson & Novack, 2002).

The aversive effects of high stress are in contrast with the effects of moderate stress. This variation involves different types of receptors in the hippocampus. At low levels of stress there is a heavy occupancy of mineralocorticoid receptors in the hippocampus. Classic memory impairment appears to be associated with high levels of stress and occupancy of cortisol receptors.

The old model—where PTSD is associated with hippocampal volume reductions—changed with reports that traumatised children did not suffer from hippocampal shrinkage (De Bellis et al., 1999; Karl, Malta, & Maercker, 2006). Indeed, the hippocampal size differences between adults and children with PTSD have been a topic of much discussion. This discrepancy may reflect a gradual adverse effect on the structure of the hippocampus so that it may not be evident until postpubertal development, or that it may be an inherent vulnerability for chronic PTSD that persists into adulthood (Gilberson et al., 2002).

Though the reduction in hippocampal volume does not appear at the time of the trauma, it does with the passage of time. Additionally, in contrast to people without PTSD (who have hippocampal asymmetry, with the left larger than the right), adults who experienced childhood abuse have been reported to have near symmetry. Woon and Hedges (2008) found that maltreatment resulting in adult PTSD may disrupt normal hippocampal development manifested as hippocampal symmetry. They also found evidence for bilateral hippocampal volume reductions for adults who had experienced abuse during childhood; but this reduction was not evident in children who had experienced abuse. This study supports the view that the hippocampal volume reduction occurs sometime between childhood and adulthood and those reductions are associated with symptom severity of PTSD.

The failure to find the reductions in hippocampal volumes with children with PTSD that have been found in adults with PTSD, led some researchers to wonder if smaller hippocampal volumes represent a predisposing factor instead of a result of PTSD. In an interesting counterpoint to the 'gluccocorticoid-cascade hypothesis', whereby cortisol is thought to lead to hippocampal volume reductions in PTSD patients, Gilbertson and colleagues explored a 'vulnerability hypothesis' which suggests that people with premorbid smaller hippocampi are more vulnerable to PTSD (Gilbertson et al., 2002). In response to trauma, these people overreact to traumatic events because they have a less viable internal thermostat to shut down cortisol release.

In an effort to determine if the reduction of hippocampal volume is a cause or a risk factor for PTSD Gilbertson and colleagues compared monozygotic twin brothers, one who had combat related PTSD and the other who never went to war (Gilbertson et al., 2002). These researchers found a concordance between the twins regardless of combat status. Both had smaller hippocampal volumes. These results suggest that the vulnerability hypothesis may be viable as a possible partial explanation for increased risk. On the other hand, Bremner and colleagues reported a 9% reduction in hippocampal volume in twins with combat related PTSD

Overall, there is a positive relationship between the degree of amygdala activity and anxiety symptom severity.

relative to their non-combat exposed twin brother (Bremner, 2000). These studies support the notion of a combined genetic and environmental contribution to smaller hippocampal volume in PTSD.

Amygdala activity

The general model of PTSD envisions amygdala hyperactivity not inhibited by the mPFC. This includes heightened amygdala activity even in the resting state. Thus, while high levels of stress appear to be corrosive to the hippocampus, the same stress can produce enhanced dendritic arborization in the amygdala (Vyas, Mitra, Rao, & Chattarji, 2002; Vyas, Bernal, & Chattarji, 2003). Overall, there is a positive relationship between the degree of amygdala activity and anxiety symptom severity.

Further illustration of heightened amygdala activity comes from researchers who have found increased blood flow in the amygdala, reflecting over-activity in patients with PTSD (Pissiota et al., 2002; Semple et al., 2000). Correspondingly, there is reduced grey matter volume in the mPFC (Carrion et al., 2001; De Bellis et al., 2002). The reduction in the mPFC represents the inability to inhibit the overactivity of the amygdala.

As with hippocampal studies, it is not clear whether these findings are related to chronic exposure to neurohormones and/or genetic factors. It could be that individuals born with smaller hippocampal/ amygdala volume are at greater risk of PTSD/ DID. Consistent with this hypothesis, abused subjects without DID were found to have larger hippocampal and amygdala volumes than non-abused subjects (Vermetten, Schmahl, Lindner, Loewenstein, & Bremner, 2006). This suggests that large hippocampal and/or amygdala volumes may be protective in the face of early trauma (or another factor allowed those structures to develop more and promoted the capacity of the individual to cope better).

During the past several years there has been an accumulation of evidence that extinction does not erase or undo fear learning. Indeed, after extinction a conditional fear can return in a range of circumstances, including the simple passage of time via spontaneous recovery, exposure to the unconditioned stimulus, or exposure to the conditioned stimulus in a novel context. The recovery or reappearance of fear indicates that extinction training actually involves new learning via the mPFC, hippocampus and the amygdala to inhibit the expression of conditioned fear rather than eliminating the underlying representation of conditioned fear (Phelps, 2009). In other words, the development of new cognitive skills and coping strategies can serve to inhibit hyperarousal and re-experiencing (i.e., flashbacks), but not erase the traumatic memories.

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The role of norepinephrine

Norepinephrine (NE) plays a major role in the pathophysiology of PTSD. Not surprisingly a hyperactive amygdala can trigger elevated levels of NE through its rich connections with the locus coeruleus (LC), which is the primary source of NE in the brain. Built into the pre-synapatic (releasing) membranes are alpha-2 auto receptors that provide a breaking function for NE. Thus, when NE is released by the pre-symptomatic neuron it makes contact with both the post-synaptic and pre-synaptic neurons. When it makes contact with the post-synaptic neuron it can trigger an action potential. When NE interlocks with the alpha-2 auto-receptor on the pre-synaptic neuron that released the NE, it acts to slow down the release of NE.

However, if the alpha-2 pre-synaptic NE auto-receptor is agonised (blocked) the braking action does not occur. Yohombine (a herb used for treatment of sexual dysfunction) and caffeine are alpha-2 agonists. When a person with PTSD consumes excessive amounts of yohombine or caffeine they may experience excessive NE, which may precipitate a panic attack. Alternatively, a person may have hyposensitive alpha-2 NE auto receptors, which are insensitive to NE, and do not act to shut down NE. As a result of their insensitivity NE is not shut down, but rather, is increased.

Several factors impair the PFC of which elevations of NE is part. These include: 1) low serotonin and the corresponding reduction of the OFC's capacity to inhibit the overactivity of the amygdala; 2) elevation of NE, through activation of the alpha1-adrenergic receptors; 3) increases in cortisol and its interactions with catecholamines.

These factors ramp-up anxiety through unleashing the amygdala and further trigger the release of NE through the LC, and dopamine via the ventral tegmental area, as well as acetylcholine via the dorsolateral tegmental nucleus. All these factors further dampen the PFC's capacity for problem solving and rational behaviour. As a consequence, there is a heightened tendency toward startle response, vigilance, insomnia, flashbacks, intrusive memories, and increased fear conditioning.

Though NE, when excessive, can act to take the PFC off-line through the alpha-1 adrenergic receptors, moderate levels can act to enhance PFC activation through the alpha-2A receptors. This underscores the utility of a moderate degree of activation. It is also a moderate degree of activation that is critical for neuroplasticity through the Glutamate circuits and specifically the NMDA receptors. These dynamics underscore the efficacy of exposure and the 'safe emergency' addressed below.

Complexity in the HPA activity

By the mid-1990s researchers were reporting that victims of trauma and chronic stress experienced wide variations in elevations of cortisol levels. Yehuda and colleagues found that some combat veterans, Holocaust

survivors, and other trauma victims actually had reduced cortisol secretion as well as other factors indicating abnormal HPA activity (Yehuda, Boisonuane, Iowy, & Giller, 1995; Yehuda, McFarlane, & Shalev, 1998). The most robust evidence comes from patients who experienced chronic and intractable PTSD. By 2000, stress-related hypo-cortisolism gained considerable attention (Gunnar & Vazquez, 2001; Fries, Hesse, Hellhammer, & Hellhammer, 2005).

threatened a person is physically there tends to be a higher and flat cortisol output. Though morning cortisol can be lower, afternoon/evening output is higher. If the stress is of a social nature (i.e., divorce), the overall cortisol output is higher, including morning, afternoon and evening.

The issue of when cortisol levels are high or low may be correlated with vulnerability to develop PTSD. For example, some studies have shown that initially low urinary cortisol

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Yehuda (2001) has suggested that while PTSD is associated with hyperactivity in the HPA axis, it leads secondarily to reduced cortisol levels through negative feedback. In a meta-analysis of 107 studies on hypocortisolism in the morning, Miller, Chen, and Zhou (2007) found that the dysregulation in cortisol reflected greater concentrations of afternoon/evening cortisol, (especially if the person was subjected to shame), a flatter diurnal rhythm, and a higher daily volume of cortisol output. They note that collectively these findings suggest that chronic stress is accompanied by a deregulated pattern of secretion through lower than normal morning output, a higher than expected secretion across the rest of the day, and a flattened diurnal rhythm. Also, there was evidence that cerebrospinal fluid concentrations of corticotropin-releasing hormone (CRH) were significantly increased.

There seems to be a time factor operative here. The more months that had elapsed since the stress first emerged, the lower a person's morning cortisol, daily volume, and ACTH. On the other hand, when chronic stressors were still present in the person's environment, morning, afternoon/evening, and daily cortisol output were significantly higher.

Miller, Chen and Zhou (2007) also examined whether HPA activity varies based on the nature of the threat. For example, if stress involves how

concentrations immediately after the trauma predict subsequent PTSD diagnosis in adults (Yehuda et al., 1998; Delahanty, Nugent, Christopher, & Walsh, 2005).

It appears that a marked elevation in cortisol in the evening is a more reliable predictor of later PTSD than measures of cortisol during other times of the day. Also, elevations of pro-inflammatory cytokines such as interleukin (IL)-6 soon after the trauma, is predictive of PTSD development six months later.

Elevated cytokines have shown to be not only indicators of the inflammatory response to physical and emotional stress, but also the disruption of the HPA axis (Chrousos, 1995). It also appears that an elevation in cytokines is significantly correlated with evening cortisol. Several studies have examined the immune system function of adults with PTSD and found increased levels of pro-inflammatory cytokines circulating or in the cerebral spinal fluid (Maes et al., 1999; Baker et al., 2001; Rohlder et al., 2004).

The secretion of inflammatory cytokine interlukin-6 (IL-6) can also be triggered by activation of beta-adrenergic receptors, with increases in NE. Inflammation can also occur through other aspects of acute stress including the corticotropin releasing hormone (CRH)/P-histamine axis and by the combination of elevated cortisol and IL-6 (Elenkov, Iezzoni, Daly, Harris, & Chrousos, 2005). It has been shown that children who experienced trauma and also had elevations of IL-6 and evening cortisol were more likely to develop PTSD than children who experienced trauma and did not have those elevations (Pervanidou, 2008).

Further complexity of the stress response systems occurs when there is hypersecretion of CRH by the hypothalamus that may lead to an adaptive down-regulation of CRH receptors in the anterior pituitary (Bremner et al., 1997). This down-regulation may be adaptive to regulate pituitary hypertrophy, because without it, the elevated CRH would result in higher cortisol levels and damage to multiple systems including the hippocampus. This mechanism, therefore, involves a feedback system that is an attempt to put on the brakes. But it is not without adverse consequences. Pituitary volumes were found to be significantly larger in pubertal/postpubertal maltreated pediatric subjects with PTSD (Thomas & De Bellis, 2004). The duration of the abuse correlated negatively with pituitary volume.

Ramping up of this amygdala-HPA system involves the process known as 'priming' or sensitisation that occurs when responses to repeated stress increase in magnitude. This phenomenon reflects the chronic compensatory adaptation of the amygdala-HPA axis long after trauma exposure so that adrenocorticotropic hormone (ACTH) and cortisol are set at lower 24-hour levels. Other hormones such as arginine, vasopressin, and the catecholamines act synergistically with CRH. Thus, when a new emotion stressor is experienced the amygdala-HPA axis functions are enhanced through higher ACTH and higher 24-hour cortisol concentrations in response to stress. Thus, this primed system hyper-responds to stress (De Bellis, Hooper, & Sapia, 2005).

Finally, one last note about the breakdown of this system. The interaction with the noradrenergic system and CRH results in dysregulation of serotonin and increases the risk of depression. This has lead theorists to note that people suffering from PTSD are at risk for developing a major depression. Indeed, Breslau, Davis, Peterson, & Schultz

(2000) showed that onset of major depression is increased significantly for people who are exposed to trauma compared to those not exposed.

Based on this background of neurodynamics we can move on to a discussion of how a person suffering from PTSD may experience dysregulated memory systems. Since our memory systems form the fabric of who we are, a coherent therapeutic approach necessitates putting the memory systems in perspective.

Memory and neurodynamics

People with PTSD typically remember the traumatising event, but describe blank periods or gaps in the details of what occurred. Their recollections of details are often vague, unclear, and disorganised (Harvey & Bryant, 1999). These symptoms are largely related to the differing effects of explicit and implicit memories corresponding to the hippocampus and the amygdala respectively.

The reported damage to the hippocampus among PTSD patients, via both cortisol and excitotoxicity, has shown to be associated with verbal memory deficits (Bremner et al., 1995). Consistent with these findings, it is reported that PTSD patients also have declarative and autobiographical memory problems. For example, inpatient adolescents who had experienced trauma were reported to have reduced autobiographical memory with the degree of memory loss correlated with the number and severity of traumatic events (de Decker, Hermans, Raes, & Eelen, 2003).

Yehuda and colleagues found deficits in semantic memory in adult veterans with PTSD compared to controls (Yehuda et al., 1995). Clark and colleagues explored working memory of people with PTSD utilising PET technology and found significantly less activation of the left dorsolateral prefrontal cortex (DLPFC), which is associated with working memory, in patients with PTSD compared to controls (Clark et al., 2003).

The dynamics of memory and asymmetrical hemisphere activation has been explored with people with PTSD. Teicher (2000) found that people with a childhood history of abuse tend to use their left hemisphere when thinking about neutral memories. When they recall early upsetting memories they use their right hemisphere. Control subjects tended to have more integrated and bilateral response to recalling both neutral and traumatic memories. The abused subjects had hyperactivated right hemispheres, but their left hemispheres appeared developmentally arrested. Abused subjects appeared to fail to integrate traumatic memories into left hemisphere narrative networks.

Recent work on resilience by Morgan and colleagues (2000a, 2000b, 2002, & 2004) suggest those who experience greatest cognitive disturbance during combat (confusion, dissociation) are the most likely to suffer PTSD. These findings correspond to dampened frontal lobes activation which could regulate affect. Interestingly, they did not find that those subjects with better ability to deal with stress were less 'stressed' during the event. In fact, they found the best performers in combat had the highest levels of cortisol. They appeared to make better use of the surge of noradrenaline and cortisol. They also had higher heart rates, which might explain better physical performance.

Having a 'clearer head' seems to relate to higher levels of at least two other neurohormones:

1) elite performers in combat were found to have elevated levels of the hormone dehydroepiandrosterone (DHEA), which seems to buffer the brain against the negative effects of stress—although this mechanism is not yet fully understood (Morgan, et. al., 2004);

2) the most potent protective factor appears to be a neurotransmitter called neuropeptide-Y (NPY) which binds to receptors on neurons in the prefrontal cortex and alters their response to noradrenaline, and acts as a break to its accelerator pedal. The best performers had NPY levels that were about one-third higher than in their peers and quickly returned to a healthy baseline once the stress was over. Less resilient individuals seem to have a lower capacity for NPY production and NPY levels dropped to below baseline 24 hours afterwards.

Overall, this diverse body of research indicates that the neurodynamics underlying the impairment in the memory systems result in memory gaps, and deficits in working and semantic memory. This cognitive disturbance impedes the development of a sense of purpose and cognitive clarity, which are essential components of resilience.

Meaning and significance of memory is complex, multilayered, and beyond the reductionism that words provide.

Implicit and explicit memory systems

The 'single representation theory' of memory that has gained influence with many CBT-oriented anxiety researchers envisions networks involving many thousands of nodes with dense interconnections between them. A person, feature, shape, concept, or emotion is represented by a node. The representation consists of patterns of interconnections between the nodes. Building on this theory, Lang (1985) proposed that a frightening experience creates a 'fear network' in memory, consisting of information about the traumatic event, meaning information, and the response information about emotional and physiological reactions. Reactivation of the fear network automatically occurs when the person with PTSD encounters a situation that matches cues or features of the original fear network, which then produces the same physiological responses and interpretation of being in danger.

Based on these ideas, Edna Foa and her colleagues (1998) developed the *Emotional Processing Theory*. They have proposed a variety of ways that memories from frightening events differ from memories from a traumatic event. For example, large numbers

of stimulus danger interconnections between stress-related nodes become stronger than their connections to non-trauma related nodes. The memory networks contain large numbers of response elements associated with negative self-appraisals that include thoughts of being vulnerable and weak. Also, they argue that the severity of the traumatic event leads to disrupted cognitions and fragmented, disjointed fear structures.

Teasdale and Barnard (1993) were some of the first to point out the limits of the single-representation network model based on the premise that it only represents one node for each emotion. A single level would not account for how an emotionally laden 'hot' way of remembering trauma on one occasion, can be remembered in a more detached 'cool' way on another occasion. These extra dimensions are critical to recognise and integrate in therapy. For example, a client can understand that even though she feels like she is a bad person, she can be a good person.

Owing perhaps to its adaption to the CBT perspective where language is paramount, the single network model cannot account for levels of meaning beyond that of words or sentences. Meaning and significance of memory is complex, multilayered, and beyond the reductionism that words provide (Dalgleish, 2004). Moreover, memory of a traumatic event(s) involves implicit as well as explicit memory.

Addressing some of the limitations of this Single Representation Network Theory, Brewin (2003) proposed a Dual Processing Theory that accounts for such phenomena as flashbacks and other non-verbally assessable memories. Brewin (2005) has highlighted the dynamic relationship between the two memory systems— situationally accessible memories (SAMs) and verbally accessible memories (VAMs), which are essentially implicit and explicit memory systems respectively. Accordingly, the VAM system involves the hippocampus, while the SAM involves the amygdala.

The heavy emphasis on explicit memory leads to a limitation of the 'fear network' model—conceived as composed of semantic and highly organised memories—but it does not comprehensively account for implicit memory. According to the Dual Representational Theory, trauma information can be stored as VAMs on the conscious memory level or SAMs, which are largely nonconscious. Where VAMs can be accessed in therapy through deliberate recall, SAMs are only accessible through cues that activate the nonconscious networks (Brewin, Dalgleish, & Joseph, 1996).

Consistent with the dimensions of explicit memory that are cortically and hippocampally driven, the VAM system comprises the narrative memory of the trauma. These memories are autobiographical and can be deliberately retrieved. VAM system memories of the trauma comprise the context, including the past, present, and future.

'These memories are available for verbal communication with others, but the amount of information they contain is restricted because they only record what has been consistently attended to. Diversion of attention to the immediate source of threat and the effects of high levels of arousal greatly restrict the volume of information that can be registered during the event itself' (Brewin, 2005, p. 139).

A traumatised individual uses the VAM system to evaluate the trauma both at the time it is happening, and afterwards, as she asks herself how the event could have been prevented and/or the consequences and implications of the experience on the future. Brewin and colleagues refer to the emotions that accompany VAM system memories as 'secondary emotions' because they were not experienced at the time of the trauma itself. They are directed at the past (regret or anger about careless risks taken), or the future (sadness at the loss of cherished plans and hopelessness at the thoughts of not finding fulfilment). These secondary emotions involve guilt and shame over the perceived failure of having not prevented the event.

The SAM system, by contrast, contains information that has been obtained from lower-level processing of the traumatic scene which includes; sights, sounds, and bodily sensations such as the changes in heart rate, temperature, or pain that were too briefly apprehended to be bound together in conscious memory required for the VAM system. The SAM system is consistent with implicit memory that is largely amygdala driven and accounts for flashbacks that can be triggered involuntarily by situational reminders of the trauma, which may include sights, sounds, physical feelings or emotions.

The interplay between VAMs and SAMs can be influenced by the degree of intensity of emotion. Since VAMs are highly dependent upon the hippocampal-PFC memory system, during periods of intense emotion they can be superseded by lower level systems that involve the SAMs—the hyperactivity of the amygdala. In such situations the SAM system tends to become hyperactive.

Thus, during these periods of intense emotion associated with traumatic experiences, there tends to be a reduction of hippocampally dependent processing of information (underlying the VAMs—explicit memory) and the formation of SAMs due to heightened amygdala reactivity. The increase in SAMs and the decrease in VAMs correspond to increased trauma reminders in the form of flashbacks triggered by sights, sounds, and smells that are experienced as a sense of timeless threat.

Because they are amygdala driven, and part of the implicit memory system, the non-hippocampally dependent memories (SAMs) appear to be more resistant to change and the passage of time. In contrast, VAMs, are far more vulnerable to distortion/modification over time. Thus, the SAM system is not restricted to verbally coded memories and is more extensive. Because they are difficult to communicate to a therapist, SAMs (implicit memories) are also difficult to update by a purely verbally-based approach. Flashbacks, for example, are spontaneous and difficult to control because it is awkward to attempt to regulate exposure to the sights, smells, and sounds, etc., that act to trigger them. The emotions triggered have been referred to as 'primary emotions', consisting of fear, helplessness, and horror (Grey, Holmes, & Brewin, 2001). The more drawn out and extended the traumatic experience, the more the tendency to experience a range of emotions.

Since the SAM system is largely non-conscious and can involve the fast track to the amygdala, sensory information (i.e., smells or sounds etc.) go directly to the amygdala from the thalamus. Meanwhile, high levels of stress result in elevated levels of cortisol, NE, and cytokines which result in impairments to the VAM system through over reactivity of the amygdala.

According to Dual-Representation Theory, when the client with PTSD deliberately maintains attention on the implicit memories (i.e., the sounds, smells etc.) content of the flashbacks, and no longer tries to suppress them, the memories encoded in the SAM system become reconsolidated in the VAM system. The timeless qualities of the SAM images and sensations thus become linked with a spatial and temporal context. Through the reconsolidation in the VAM system, when SAMs are triggered, they are put into the context of time and place so that the individual can remind himself that he is now safe and the trauma or threat is in the past.

The exposure and reconsolidation process must be repeated multiple times not only because there generally exists a lot of information in the SAM system to be re-encoded, but also because neuroplasticity necessitates repetition. Rehearsing the new VAM memories long enough will promote easier access to the SAM memories. Thus, Dual Processing Theory envisions the creation of new memories through the VAM (explicit system) that compete with SAM (implicit system memories of the trauma). This perspective acknowledges that original SAM based memories of the trauma are indelible, and that the VAM system memories put the SAM system memories in perspective. This process calms down the activation of the sympathetic nervous system, including how the PFC inhibits the overactivity of the amygdala.

The interaction between the SAM and VAM system can be promoted through exposure and cognitive restructuring processes. Peres and colleagues (2007) have demonstrated cerebral blood flow changes during retrieval of traumatic memories before and after psychotherapy. The narrative organisation of memory can be modified by associated experiences of emotional context and the state of consciousness during the recall process (Peres, Mercante, & Nasello, 2005).

The therapeutic integration of SAMs and VAMs is consistent with the Expert Consensus Guidelines series of treatment of PTSD (which states that) exposure-based therapy is the treatment of choice for intrusive thoughts, flashbacks, trauma related fears, and avoidance (Expert Consensus Panel for PTSD, 1999). Many CBT approaches to PTSD combine cognitive restructuring methods with exposure (Brewin, 2001). Exposure to the traumatic memories is a well-constructed process that incorporates the emotional content (Littrell, 1998).

The re-interpretation and reconsolidation of traumatic memories, combined with exposure and cognitive restructuring, can alleviate some of the distressing symptoms by challenging the nature of the representations of the traumatic event (Peres, McFarlane, Nasello, & Moores, 2008). The affect shift, combined with new explicit, hippocampally driven memories (VAMs), allows the traumatic and fragmented memories (SAMs) to transform into an integrated implicit and explicit memory that can be more easily managed and available for narrative expression.

This integration of implicit and explicit memory (VAMs and SAMs) via exposure takes place within the 'safe emergency' of the therapeutic relationship. The resulting promotion of a moderate degree of anxiety is critical for neuroplasticity. These dynamics include moderated levels of NE to engage the alpha-2A receptors, which engage the PFC (instead of turning it off by excessive amounts of NE which engage the alpha-1 adrenergic receptors), and a moderate increase in glutamate, which engages the NMDA receptors—important for long-term potentiation (LTP).

Indeed, consistent with the dynamics of the inverted 'U', which aid in effective neuroplasticity and memory, arousal levels must be carefully managed. For example, if arousal levels are too low, the PFC is not engaged, and traumatic images are not accessed. But if arousal levels are too high, the levels of NE are excessive and the adrenergic receptors are activated serving to take the PFC off-line. The client begins to dissociate or become so overwhelmed with the traumatic memory, at the expense of contact with the immediate surroundings, strengthening its networks without transferring information from images biased to verbal memory (Brewin, 2005).

Therefore, a graduated and incremental approach is advisable whereby the traumatic SAM system memories are dealt with in smaller units, and a hierarchy from less distressing to more distressing is developed allowing therapist and client an opportunity to breakdown the levels of distress into 'doable' chunks. Most importantly, the client, rather than waiting to feel comfortable in challenging himself to move out of his comfort zone, needs to understand that a moderate degree of anxiety is therapeutic. Through this process of incremental challenge, the newly constructed VAM system memories can be facilitated by a moderate degree of anxiety accessed through graduated exposure and integration of SAMs—implicit memory. This perspective is similar to the concept of a 'therapeutic window' that highlights the importance of the timing for effective treatment of victims of childhood trauma (Briere, 2002).

Because they are difficult to communicate to a therapist, SAMs (implicit memories) are also difficult to update by a purely verbally-based approach.

'Hot spots' are brief moments when emotions are particularly intense and correspond to flashbacks. Hot spots are important points for focus in exposure rather than the entire event (Ehlers & Clark, 2000). Hot spots may correspond to moments where there is maximal functioning separation between visuospatial and verbal processing (Brewin, 2005). This separation can lead to a large discrepancy between the contents of the respective implicit and explicit memory systems (VAM & SAM). These are moments that provide retrieval cues that need reconsolidation into explicit memory so that they do not trigger flashbacks.

It is recommended that therapeutic attention be on the fragmented SAM related flashback memory hot spots. Sustained conscious attention to these memories could potentially promote integration with the VAM system, thereby strengthening coping skills with a new, far more adaptive, narrative. This process enhances inhibitory control over the amygdala and diminishes flashbacks. The restructuring and integration of the memory systems thus promotes a new narrative based on memories of successful self-efficacy prior to the trauma and constructive lessons learned post-trauma.

Since traumatic memories are vulnerable to being triggered and re-experienced (i.e., flashbacks) because they are fragmented and disorganised, therapy should be directed toward making those memories coherent and structured to reduce the risk of unwanted intrusions (Ehlers & Clark, 2000; Conway & Pleydell-Pearce, 2000). Traumatic memories can be reactivated through exposure and reconsolidation by incorporating them into more accurate information.

Implicit memories cannot be corrected, but can be more efficiently inhibited. Therapists can promote the construction of alternative memories. Theoretically, this is a Constructivist perspective instead of a Connectionist perspective. Newly constructed memories compete with the original memory for control of behaviour and attention with the emphasis on self-organisation.

Also, through the therapeutic alliance the degrees of anxiety via NE can be managed so that only moderate levels will occur for maximum neuroplasticity. In this way, therapy reverses the breakdown of normal memory processing that initially leads to the development of PTSD.

Increasing cognitive complexity and self-complexity bolsters one's stress protective abilities (Tennen & Affleck, 1998). Self-complexity increases as we expand the number of different perspectives we have of ourselves, optimally one of those perspectives is that the self is complex enough to weather the stress.

Therapeutic approaches, therefore, should attempt to integrate implicit and explicit memory of traumatic experience by constructing an adaptive narrative promoting self-organisation. It is through this foundation that as the PTSD symptoms subside the individual may be inclined to stand back and take a wider look at his/her place in the world and try to derive meaning from that wider perspective. Tainted much less by the emotional pain of the trauma there is the potential to represent a sense of connectedness with people and the world around him/her. The old sense of self ('old me') is lost since the trauma, and the new sense of self ('new me') can be seen as more open and appreciative of the interdependence of the world.

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