TRAUMA, ATTACHMENT, & DISSOCIATION

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There is an accumulation of data over the past two decades suggesting a strong association between childhood physical and sexual abuse and the development of dissociation (Brown, Sanders, McRoberts, & Tollefson, 1989, Sanders & Giolas, 1991, Terr, 1991) which has also been reported in Vietnam veterans with chronic Post-Traumatic Stress Disorder (Brende, 1987)

"Dissociation" has been viewed as a theoretical construct or defense mechanism to explain the absence of certain mental contents from consciousness in spite of the fact that the concept of 'repression' has been used predominantly for many years. (Frankel, 1990). Dissociation has been defined in at least three distinct ways:

> "First, dissociation is used to characterize semi-dependent mental modules or systems that are not consciously accessible, and/or not integrated within the person's conscious memory, identity, or volition. Second, dissociation is viewed as representing an alteration in consciousnes wherein the individual and some aspects of his or her self or environment become disconnected or disengaged from one another. And third, dissociation is described as a defense mechanism that effects such disparate phenomena as nonorganic amnesia, the warding off of current physical or emotional pain, and other alterations of consciousness, including a chronic lack of personality integration, such as with Dissociative Identity Disorder (DID)" (Cardena, 1994).

Many professionals in this field do not include drug induced or mood related 'state dependent learning' (Overston, 1991) as manifestations of dissociation since lost memories associated with a certain mood can usually be retrieved when that mood is reinstated (Singer & Salovey, 1988). On the other hand, Rossi & Cheek (1988) compared the phenomenon of state-dependent learning to both traumatic dissociation and therapeutic hypnosis, as Brown (1994) has described:

> "Intrusive memories, feelings, and flashbacks can be conceptualized as state-bound patterns of information, that are released whenever arousal reaches a certain critical level. This could account not only for the vividness of intrusive imagery and affect at certain times also for the way in which such material can emerge in subsequent periods of arousal. This model also parsimoniosulsy accounts for the frequency of both affective disturbance and somatic symptoms (either as the direct consequence of autonomic nervous system (ANS) disturbance or as a physical reexperiencing of the intrusion of traumatic memories), and for the decreased threshold for subsequent dissociation (Spiegel et al, 1989)."

Some types of 'dissociative' behaviors may be considered relatively normal, as found in: 1) Individuals who engage automatically in certain tasks without full conscious

awareness, even when there may be several tasks performed at the same time, such as may occur in assembly line work; (2 Individuals whose behaviors or perceptions are inconsistent with external appearances or verbal reports, such as may occur when an individual in a socially embarrassing situation may break out with 'hives' although maintaining very calm speech and demeanor (Erdelyi, 1985).

Pathological dissociation, first described by Pierre Janet a century ago as he studied patients suffering from Hysteria, is the prevelent defense mechanism found in traumatized individuals. Pathological dissociation is associated with the 'disintegration' of personality and the loss of all or some memory of events and all or some aspects of identity. In fact, it may lead to the development of separate, coexisting mental systems - consciousness, memories, or identities - in victims unable to retrieve certain traumatic or distressing events from the past, particularly adults traumatized as children. Thus, pathological dissociation may lead to the development of separate, coexisting mental systems - consciousness, memories, or identities leading to the formation of Multiple Personality Disorder (now called Dissociative Identity Disorder (DID) (DSM-IV, 1995).

Individuals with a loss of memory caused by dissociative fugue states are likely to be temporarily confused about who or where they are and can't recall basic personal information that they should be able to remember. Individuals with DID suffer from extraordinary lapses of memory associated with changes in consciousness, explaining their inability to recall traumatic events. They are unable to integrate various experiences and memories into a single identity and suffer from the coexistence of diverse identities, each of whom has either a differing memory or no memory at all of certain events. In fact, there may be a complete inability to retrieve certain traumatic or distressing events by the host personality or certain other alternate personalities in DID in spite of the fact that one or more victim personalities may spontaneously or with triggering phenomena (sensory stimulation and/or a therapist's suggestion) recall or relive the orginal traumatizing experience. Strangely there is also an alternate personality, commony demonstrating aggressive or narcissitic traits, with knowledge of the other alternates although not necessarily of the traumatic memories.

How does dissociation take place? It has been decribed as an unusual disconnection or disengagement of the self from the painful aspects of a traumatic experience, taking place as follows:

> "Such disconnection may occur in a number of ways. A rape victim, for example, may disengage from the ongoing event by seemingly having no sensory experinces or emotions during it, by 'observing' it from a perspective at a distance form the physical body (i.e., an out-of-body experience), by becoming fully immersed in an imaginal event, and so forth" (Cardena (1994).

Is there a neurological basis for pathological dissociation? Putnam (1988) has found that the shifts between different alter personalities in DID patients are mediated by alterations in psychobiological states.

Some neurological syndromes simulate dissociation, i.e. organic amnesia, temporal lobe epilepsy, and commissurotomy. Patients with striate cortex damage but an otherwise intact visual system report 'blindness' on the contralateral side of the lesion although they can sense the presence of a flash of light coming from the blind side in a remarkably accurate way. Gazzaniga (1985) described a commissurotomized (severence of the corpus callosum connecting both hemispheres) individual who could not 'see' the picture of a horse presented to the left visual field but still drew the picture of the horse with his left hand.

Brown, P. Toward a Psychobiological Model of Dissociation and Post-Traumatic Stress Disorder, in Ed: Lynn, S.J. & Rhue, J.W., DISSOCIATION: Clinical and Theoretical Perspectives. Guilford Press, NY & London.

There have been several studies of brain function in dissociative disorders, however it is a complex problem.
Brown suggests two related areas for examination: 1) the psychophysiological studies of PTSD and 2) the studies of similarities and differences between temporal lobe epilepsy and dissociation.

PTSD patients report significantly higher levels of dissociative symptoms than do matched controls both at the time of the original trauma and subsequently (Bremner, Southwick, Brett, et al, 1992; Bremner, Steinberg, Southwick et al, 1993).

In control subjects, the administration of the alpha-2 receptor antagonist yohimbine results in mild stimulation, wheras PTSD patietns experience anxiety, panic attacks and dissociative experiences including flashbacks and increased norepinephrine turnover (Krystal, Southwick, & Charney, 1990; Southwick, Krystal, Morgan, et al., 1993).

PTSD patients have greater increases in heart rate, blood presssure, and anxiety (Pitman et al, 1987) and have a fourfold decrease in sensitivity to norepinephrine compared with controls (Perry, Southwick, & Giller, 1989).
Giller and co workers (Giller, Perry, Southwick, et al, 1990) suggest that there is more sensitivity to the noradrenergic system and a lostened ability to 'damp down,' input from stimulation so that there is a heightened response to stimuli and difficulty in maintaining consistent levels of arousal.

In explaining the relationship between the symptom of emotional numbing and detachment, Brown states:

> "Emotional numbing may be related to catecholamine depletion as a result of hyperreactivity to conditioned stimuli, behavioral constriction (with the consequent loss of curiosity and involvement resulting in a further dampening of responses), immunosuppression, and analgesia related to the release of endogenous opioids

during conditioned fear responses (Pitman et al., 1990; van der Kolk et al., 1985; van der Kolk, 1987)."

There are recent findings that suggest an analgesic response as a component of the acute dissociative response in severe traumatic events (Cardena & Spiegel, 1993).

Suppression of pain is caused by stress-induced endogenous opiods in animals (Akil, 1985) and can be blocked by the administration of the opiod antagonist naloxone (Calcagnetti,Helmstetter, & Fanselow, 1987). Pitman et al (1990) also found that pain responses in Vietnam vetrans with PTSD exposed to stressful video tapes of combat, were blocked when naloxone was administered.

Similarities between chronic PTSD and naracotic withdrawal suggest opioid depletion affects in PTSD patients (van der Kolk et al., 1985). An attempt to rais endogenous opiod levels amy account for the need of many PTSD patients to overstimulate themselves - which occurs as a result of their provocative and almost addictive like behaviors to repeated trauma and stress.

Individuals suffering from chronic Post-traumatic and Dissociative Disorders are prone to having vacillating episodes of 'highs' and lows' by seeking stimulation, excitement and risk taking followed by periods of denial?numbing and withdrawal from stimulation. Brown (1994) has described the findings of several researchers studying the physiological relationship with these phasic symptoms:

"Periods of heightened arousal, increased sensitivity to specific stimuli, and the phasic reexperiencing of trauma-related imagery alternating with emotional numbing and the development of dissociative symptoms might be associated with a persistent imbalance between the systems that regulate both the catecholamines and the endogenous opiates (Burgess, Watson, Hoffman, & Wilson, 1988; Spiegel, Hunt, Dondershine, 1988). A biological dynamic instability appears to underlie the psychological changes, in which heightened activity in the noradrenergic systems of the hippocampus and amygdala (Margulies, 1985) is accompanied by surges in endogenous opiate release during reexposure to traumatic situations (Friedman, 1988; van der Kolk et al., 1985; van der Kolk, 1989). This brief storm is followed by periods of emotional numbing in which amine and opioid depletion are accompanied by parasympathetic dominance (van der Kolk et al., 1985).

Hormonal levels are disturbed in individuals with PTSD. Acute elevations of epinephrine and norepinephrine in response to stressful experiences are associated with elevations of cortisol and lowering of testosterone levels. Lowered testosterone levels are often seen in individuals who have been in stressful situations, particularly those with high preexisting levels of anxiety, hostility, or depression (Mason et al, 1990). Elevated testosterone levels may be associated with repressed anger, a sense of mastery, impulsivity, suspicion, and general aggression, (Mason et al, 1986, 1990).

Patients with chronic PTSD and Dissociative symptoms frequently display episodic

aggressive outbursts, found to be linked with abnormally high norepinephrine levels (also associated with hypervigilance and feelings of competence) (Mason et al, 1990).

Brown (1994) suggests that in patients with DID, various 'alters' with different feeling tones (e.g., paranoid, aggressive, fearful, or submissive) could reasonably be expected to show sgnificant differences in the norepinephrine/epinephrine ratio and cortisol and testosterone levels during the fluctuating presence of different 'alters'.

Physiological measurements - heart rate, EMG, and skin conduction - were completed on subjects who had been traumatized and then re-exposed to traumatic stimulation. All subjects reponded significantly differently compared to a control group. They had increased heart rates and EMGs while female subjects had 1/3 higher responses to imagery for personal traumatic events compared to males. The traumatized subjects also demonstrated greater reductions in skin conductance responses during positive imagery (Shaler, Orr, & Pitman, 1993).

REFERENCES

Bremner, Southwick, Brett, et al (1992) Dissociation and Post-traumatic Stress Disorder in Vietnam combat Veterans. American Journal of Psychiatry, 149 (3), 328-332.

Bremner, Steinberg, Southwick et al (1993) Use of the Structured Clinical Interview for DSM-IV Dissociatived Disorders for Systematic Assessment of Dissociative Symptoms in PostTraumatic Stress Disorder. American Journal of Psychiatry. 150(7), 1011-1014.

Brende, J.O. (1987) Dissociative Disorders in Vietnam Combat Veterans, Journal of Contemporary Psychotherapy, vol. 17, No. 2.

Brown, P. (1994) Toward a Psychobiological Model of Dissociation and Post-Traumatic Stress Disorder, In (Ed) Lynn, S.J. and Rhue, J.W., DISSOCIATION: CLINICAL AND THEORETICAL PERSPECTIVES, Guilford Press, N.Y.

Burgess, P., Watson, I.P., Hoffman, L. & Wilson, G.V. (1988) The neuropsychiatry of Post-Traumatic Stress Disorder. British Journal of Psychiatry. 152, 164-173.

Cardena, E. , (1994)The Domain of Dissociation, in Ed: Lynn, S.J. & Rhue, J.W., DISSOCIATION: CLINICAL AND THEORETICAL PERSPECTIVES, Guilford Press, N.Y.

Erdely, M.H (1985) Psychoanalysis: Freud's cognitive psychology. New York: W.H. Freeman.

Friedman, M.J. (1988) Toward Rational Pharmacotherapy for Post-Traumatic Stress Disorder: An Interim Report. American Journal of Psychiatry, 145, 281-285.
Sanders, B., McRoberts, G., & Tollefson, C. (1989) Childhood Stress and Dissociation in a College Population, Dissociation, 2(1) 17-23.

Gazzaniga, M.S. (1985) THE SOCIAL BRAIN: Discovering the networks of the mind. New York: Basic books.

Giller, Perry, Southwick, et al, (1990) Psychoendocrinology of Post-Traumatic Stress Disorder. In M.E. Wolf & A.D. Mosnaim (Eds.), POSTTRAUMATIC STRESS DISORDER: ETIOLOGY, PHENOMENOLOGY, AND TREATMENT, Washington, DC. American Psychiatric Press.

Krystal, Southwick, & Charney (1990) Yohimbine Effects in PTSD Patients. Paper presented at the annual meeting of the American Psychiatric Association, New York.

Margulies, D.M. (1985) Selective Attention and the Brain: A Hypothesis Concerning the Hippocampalventral Striatal Axis, the Mediation of Selective Attention, and the Pathogenesis of Attentional Disorders. Medical Hypotheses, 18, 221-264.

Mason, J.W., Giller, E.L., Kosten, T.R., et al, (1986) Urinary free-cortisol levels in Post-Traumatic Stress Disorder Patients. Journal of Nervous and Mental Disease, 174, 145-149.

Mason, J.W., Kosten, T.R., Southwick, S.M., et al, (1990) The use of Psychoendocrine Strategies in Post-traumatic Stress Disorder. Journal of AppliedSocial Psychology, 20(21), 1822-1846.

Overton, D.A. (1991) Historical context of state dependent learning and discriminative drug effects. Behavioral Pharmacology, 1: 253-264.

Perry, B.D., Southwick, S. & Giller, E.J. (1989). Adrenergic Receptor Regulation in PTSD. In E.L. Giller (Ed.) BIOLOGICAL ASSESSMENT AND TREATMENT IN PTSD, American Psychiatric Press. Washington.

Pitman, R.K., Orr, S.P., Forgue, D.F., et al, (1987) Psychophysiologic Assessment of Post-Traumatic Stress Disorder Imagery in Vietnam combat Veterans. Archives of General Psychiatry, 44, 970-975.

Pitman, R.K., van der Kolk, B.A., Orr, S.P. et al., (1990) Naloxone-reversible analgesic response to Combat-related Stimuli in Post-Traumatic Stress Disroder: A Pilot Study. Archives of General Psychiatry, 47, 541-544.

Putnam, F.W. (1988) The switch process in multiple personality disorder and other state-change disorders. Dissociation, 1:24-32.
Sanders, B., & Giolas, M.H. (1991) Dissociation and Childhood Trauma in

Psychologically Disturbed Adolescents. American Journal of Psychiatry, 148 (1), 50-54.

Singer, J.A. & Salovey, P. (1988) Mood and memory: Evaluating the network theory of affect. Clinical Psychology Review, 84:127-190.

Southwick, Krystal, Morgan, et al (1993) Abnormal Noradrenergic Function in Posttraumatic Stress Disorder. Archives of General Psychiatry, 50, 266-274.

Spiegel, D. Hunt, T., & Dondershine, H.E. (1988) Dissociation, Hypnotizability and Trauma. Paper presented at the annual meeting of the American Psychiatric Association, San Francisco.

Terr, L.C. (1991) Childhood Traumas: An Outline and Overview. American Journal of Psychiatry, 148 (1), 10-20.

van der Kolk et al. (1985) Inescapable shock, Neurotransmitters and Addiction to Trauma: Towards a Psychology of Post-Traumatic Stress. Biological Psychiatry, 20, 314-325.

van der Kolk (1987) PSYCHOLOGICAL TRAUMA. Washington, DC: American Psychaitric Press.