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Traumatic Stress And Attachment

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Traumatic stress in the normal individual results in activation of the sympatho-adrenal system causing a rise in noradrenaline and adrenaline, stimulation of the thyroid system causing increased secretion of thyroid hormones and activation of the hypothalamic-pituitary-adrenocortical (HPA) system resulting in elevated levels of cortisol. Studies in animals and in humans with posttraumatic stress disorder indicate that chronic traumatic stress can result in dissociation of the sympatho-adrenal medullary and the HPA system resulting in sustained elevations of the former system but suppressed or altered ACTH-corticoid responsivity.

As reviewed by Henry, self preservative behavior with its emphasis on power and control, is associated with catecholamines, thyroid hormones and left hemispheric functioning while species preservative behavior, with its emphasis on attachment, familiarity, reverence and synchronicity, is associated with cortisol, oxytocin and right hemispheric functioning. Traumatic stress seems to disturb this hemispheric balance which is reflected in the suppression of cortisol and loss of attachment behavior and other species preservative right hemispheric functions.

Keywords: stress, attachment, posttraumatic stress disorder, cortisol, thyroid, catecholamines,

Introduction

After reading Jim Henry's book *Stress, Health and the Social Environment* in 1981, I was deeply moved. At the time, I was a young mother, guided strongly by my instincts in caring for my children, and I found the book extremely supportive. I was also a young scientist and the book, being an exquisite integration of psychology and physiology, kindled a variety of questions and interesting ideas about how our feelings, thoughts, attitudes and relationships profoundly affect our health. I wrote to Jim to ask him some of the questions I had and to express my appreciation for such a great contribution to science through his book. He responded to that letter and over the next 15 years he became a cherished mentor, colleague and friend.

In the years I knew Jim Henry, most of our conversations centered around the physiologic mechanisms related to an organism's loss of capacity for attachment behavior when confronted with the consequences of chronic traumatic stress.

This article will focus on the changes in some of the hormonal systems as a result of traumatic stress, such as the catecholamine, cortisol and thyroid systems and their possible relationship to attachment behavior. Hormonal findings in combat veterans with posttraumatic stress disorder (PTSD), including very recent thyroid data, will be presented and will be related to Henry's idea that the *self-preservative* physiological state associated with chronic stress, i.e., elevated catecholamines, elevated thyroid hormones and an altered hypothalamic-pituitary-adrenal cortical (HPA) system are associated with the inability to access the feelings, behaviors and cognitions associated with *species preservative* behavior including attachment, love, empathy, compassion, trust, reverence and joy.

Hormonal Profiles in Combat-related Posttraumatic Stress Disorder

At the Neuroscience Division of the National Center for Posttraumatic Stress Disorder (PTSD) in West Haven CT, USA, I have had the honor of working with John Mason studying the biological components of chronic, severe PTSD in combat veterans. These veterans report 1) re-experiencing symptoms including intrusive recollections of traumatic events such as nightmares, intrusive thoughts and flashbacks (feeling and acting as if the trauma is reoccurring) when presented with trauma related stimuli; 2) avoidance symptoms including emotional withdrawal and detachment, emotional numbing, avoidance of trauma related stimuli, lack of interest in significant activities, restrictive range of affect, a sense of a foreshortened future; and 3) hyperarousal symptoms including irritability, difficulty sleeping, anger outbursts, difficulty concentrating, hypervigilance and exaggerated startle. Many of these men suffer from extremely

disrupted lives and cannot maintain healthy attachment relationships, a job, or a stable, safe living environment.

Fig 1 shows a comparison of the mean hormonal profile in PTSD patients compared to other diagnostic groups. For all hormone measures with the exception of free T4, the PTSD group differs markedly from the major depressive disorder group, even though over half of the PTSD patients also met criteria for major depression. The classification accuracy in the differential diagnosis of PTSD versus major depressive disorder moves from of maximum of about 60% with any single hormone to 78% with the use of two hormones (the noradrenaline/cortisol ratio) to 95% when three or more hormones are used in stepwise discriminant analysis or multidimensional scaling procedures (Mason, Kosten, Southwick & Giller, 1990).

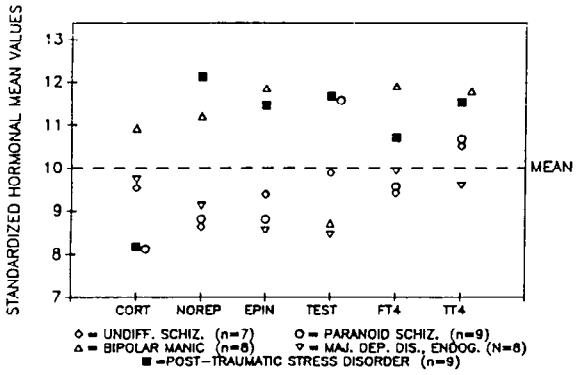

Figure 1. Comparison of the mean hormonal profile in PTSD with other diagnostic groups (from Mason et al., 1990).

The contrast of the hormonal profiles obtained in patients with PTSD and major depressive disorder suggests some significant qualitative differences in the depressive syndromes associated with these two disorders.

In general, the hormonal alterations detected in veterans with PTSD were as expected. The catabolic *fight or flight* hormones urinary noradrenaline and adrenaline were elevated as was the serum thyroid hormone, total T4, but unexpectedly, the stress hormone, urinary cortisol was suppressed in PTSD patients and in paranoid schizophrenics, compared to patients with other psychiatric disorders. This was a puzzling finding, since this hormone has been so often reported to be elevated in relation to stress, anxiety, and depression, which are prominent features in PTSD. These findings were, however, consistent with previous work in monkeys by Mason et al. (1990) showing that chronic stress could lead to a sustained suppression of urinary cortisol excretion, both under basal conditions and under conditioned avoidance stimulus conditions (Fig 2), which persisted for over a year during which weekly avoidance sessions were administered.

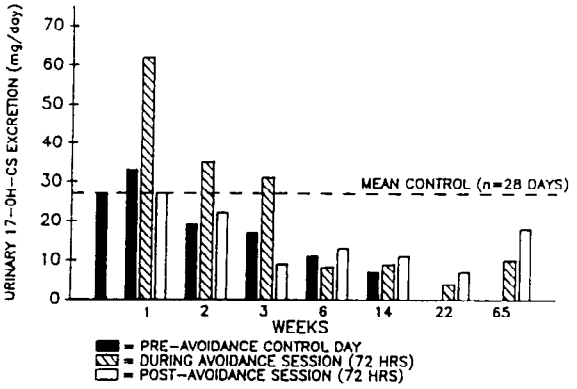

Figure 2. Lowering of urinary corticosteroid levels in a monkey during prolonged exposure to weekly 72 hour avoidance sessions (from Mason et al., 1990).

An organism's response to the initial phases of an overwhelmingly stressful situation typically involves simultaneous activation of both the sympatho-adrenal and the HPA axes (Mason et al., 1961), but these systems can operate independently: the decreased level of cortisol appears to be a result of active suppression rather than exhaustion of the adrenal glands because administration of ACTH or the presentation of other intense emotional stimuli produce substantial cortisol elevations against such a low cortisol baseline (Mason et al., 1990)

Suppression of Cortisol, Emotional Withdrawal and Lack of Attachment

The notion that suppression of cortisol is related to emotional withdrawal and chronic stress is not new, as several reports by Mason et al. over the last 30 years suggest this relationship (see e.g., Mason et al., 1990). The contribution Henry made was to expand on MacLean's description of two distinct biobehavioral modes in mammals: *self preservation*

and species preservation (MacLean, 1988), and to hypothesize that the suppression of cortisol is a physiologic mechanism associated with emotional withdrawal and self preservation that reflects the inability to access species preservative behavior including attachment, trust, empathy, spiritual experience and love (see Henry, 1997; this volume).

The cortisol system, then, can be conceptualized as one that reflects not simply stress arousal mechanisms, but rather the dynamic balance between opposing arousal and anti-arousal mechanisms (Mason et al., 1990). Low cortisol appears to be related to those coping mechanisms associated with emotional withdrawal, e.g., denial, avoidance, emotional numbing, detachment, restriction of affect, estrangement, etc. Furthermore, emotional involvement is reflected by a responsive, not suppressed, cortisol system as suggested by Price, Thaler and Mason (1957).

Suppression of Cortisol in Infants and Children

Correlations between suppressed cortisol levels and emotional withdrawal/lack of attachment behavior were reported by Tennes, Downy & Vernadakis (1977) in one year old infants during a 1 hour separation from their mother. The highest excretors of cortisol responded most emotionally to both separations and reunions (responsive), while the lowest excretors of cortisol (avoidant) showed some initial distress at separation but then became quiet and inactive, refusing social contact with the caretakers. This group ignored the mother or turned away from her upon return. (Tennes & Mason, 1982).

Hart, Gunnar and Cicchetti (1995) studied salivary cortisol in maltreated and normal children, and those who had been traumatized exhibited reduced cortisol reactivity and failed to show elevations in cortisol on days of high versus low social conflict in the classroom.

Cortisol Suppression and Emotional Numbing in Adults

In a sample of Vietnam veterans (N=30), urinary cortisol measures were strongly negatively correlated with measures of emotional numbing ($r = -.65$, $p < 0.0001$) (Mason et al., unpublished). Yehuda et al. (1993, 1995) found low urinary cortisol in Holocaust survivors with PTSD, a negative correlation between severity of PTSD and cortisol and an exaggerated suppression of cortisol in response to low dose (0.5 mg) dexamethasone. This is in contrast to many patients with major depression, supporting the observation stated previously that the hormonal alterations in PTSD are distinct from those seen in depression.

Emotional withdrawal, emotional numbing and denial can anesthetize our sense of compassion, empathy and humanity. In this state, violence can be inflicted upon others without remorse or conflict. In support of this notion, lack of cortisol responsivity was reported in the most criminally deviant and cruel psychopaths and in preadolescent boys with conduct disorder (Woodman et al., 1978; Vanyukov et al., 1992).

According to Henry, this loss of the capacity for attachment means that the individual experiences the anger and fear of *self preservation* but lacks the pity and compassion of normal attachment and *species preservation*.

It is not the temporary shift into self preservation at the expense of species preservation that is problematic, because after an acute stress response, the healthy organism returns to a species preservative mode with renewed attention to family and loved ones. The dysfunction arises when early trauma or chronically stressful conditions occur which may bias the organism toward self preservation, making it difficult to return to a mode of safety and relaxation from which our instincts for species preservation and attachment can emerge (Henry 1997; this supplement).

Lateralization of Species vs Self Preservation and the Catecholamine/cortisol Ratio

Henry (this supplement) speculates that species preservation and attachment is associated with right hemispheric activity, and that self preservation and power is associated with left hemispheric activity. HPA (cortisol) responsivity may be a good indicator of which mode is dominant. He says, "This lack of HPA response may be taken as further evidence that following psychological trauma there is a lack of right hemispheric activity despite arousal of the left." The result could be an activation of hormones associated with fight or flight (catecholamines and thyroid hormones) but a suppression cortisol, reflecting emotional withdrawal, producing an elevated catecholamine/cortisol ratio. Mason et al. (1988), reported a significantly elevated urinary

noradrenaline/cortisol (NA/CO) ratio in veterans with combat related PTSD while other investigators have reported no elevations in the NA/CO ratio in individuals with PTSD (Lemieux & Coe, 1995; Pitman and Orr, 1990). These findings may not be as contradictory as they first appear. In an effort to identify hormonal shifts associated with clinical changes, we started a series of longitudinal studies of individual veterans with PTSD which yielded data which may address the discrepancies regarding the NA/CO ratio in PTSD.

Longitudinal Studies and Clinical Stages in PTSD

Our longitudinal hormonal studies of veterans with PTSD revealed discrete periods in which the 24-hr urinary noradrenaline-cortisol ratio (NA/CO) was elevated and other periods when the NA/CO was normal. High NA/CO ratios occurred when the patient was most symptomatic, feeling threatened, irritable, using more primitive defenses, emotionally withdrawn, having difficulty sleeping and interacting with others, poor affect regulation, etc. In reverse, normal NA/CO ratios were seen when the patient was least symptomatic, feeling less threatened, using more adaptive defenses, showing increased emotional involvement, increased affect regulation, improved sleep and interactions with others (Wang & Mason, unpublished). These observations led to a conceptualization of stages in PTSD in which physiology, symptoms, affect regulation, defenses, suicidality and capacity for attachment and insight may be distinct (Wang, Wilson & Mason, 1996).

Thyroid Hormone Findings in Combat-related PTSD

In contrast to the rapid increases and decreases of the catecholamine and cortisol systems in response to stimuli, the slower moving thyroid system shows changes over longer periods of time. Bram, already in 1927, found a clear history of traumatic stress in 85% or more of 3000 cases of thyrotoxicosis, particularly. extreme fear concerning biologic survival. Combat conditions easily elicit extreme fear for one's life and in chronic combat-related PTSD we have reported sustained elevations of serum total and free T3, and a moderate elevation of total T4 compared to controls. (Mason et al., 1993) In subsequent replication studies, similar alterations in thyroid hormone levels were found in three samples of 24 Vietnam veterans with PTSD from our site, one sample of Vietnam veterans with PTSD (n=24) from the Veteran's Administration Hospital in Palo Alto, CA, one sample of Israeli veterans with PTSD (n=11) (Mason et al., 1996) and one sample (n=13) of American World War II veterans with PTSD (Wang & Mason, unpublished) compared to aged matched control groups. Serum free T4 and thyrotropin (TSH) levels in all PTSD groups were not significantly different from controls.

These findings of elevated serum total and free T3 from a sample of 96 Vietnam veterans appear to be robust in terms of both the degree of statistical significance and the replicability in three successive 24 patient samples from the same inpatient ward, as well as in two regionally different patient samples from the East and West Coasts. The similar results in the Israeli sample demonstrates a cross cultural replication, and the World War II sample demonstrates not only a replication of the findings in a different age group and a different war experience, but also the fact that the nature of these physiological changes can be chronic, detectable 50 years after the original traumatic experience.

The unusual thyroid profile of marked and sustained elevations of both total T3 and free T3 levels in the face of only a moderate elevation of total T4 level and no significant change in free T4 level or TSH prompted us to hypothesize that there my be an increased rate of peripheral extrathyroidal conversion of free T4 to total and free T3 in PTSD. This peripheral conversion has been reported to be augmented by elevated levels of catecholamines (Galton, 1965) which we have identified as an especially characteristic feature of veterans in our sample (Mason et al., 1990.)

In an effort to explore the clinical implications of the thyroid alterations in PTSD, we conducted correlational studies looking at the relationship between psychometric measures and thyroid hormones. We found a significant positive relationship in one sample between total T3 and measures of hyperarousal ($r=.41$, $p < 0.001$) (Wang et al., 1995). Since T3 is two to four times more potent biologically than T4, it is not surprising that it was associated with hyperarousal symptoms which overlap to a great extent with symptoms of clinical hyperthyroidism e.g., exaggerated startle, irritability

and anger outbursts, sleep disturbance and difficulty concentrating.

Brain Lateralization and Synchronicity

Henry thought that the elevation of thyroid hormones in PTSD were consistent with increased catecholamine activity and the fight/flight self preservative machinery of the left hemisphere. If suppression of cortisol accompanies the arousal of this machinery, reflecting the absence of the right hemisphere's domain of attachment, then the left hemisphere's influence would dominate unopposed. Henry wondered whether the left hemisphere, with its emphasis on power and control, operating without the right hemispheric "awareness of the other", could be a model for the physiology of evil.

Another feature of attachment behavior that is lost when the self preservative mode dominates, is synchronicity. Attachment has been described by Elicker as the "persistent style of a neurobiologically based biobehavioral system that regulates the biological synchronicity between organisms" (Henry, in press). When the environment is safe and vigilance is not necessary, biological synchronicity between organisms is possible and species preservative behavior including grooming, sleeping, playing and sexual activity, can emerge. When there is a perception of loss of control and threat to safety, self preservative behavior begins to take over. Henry speculated that this shift significantly inhibited access to the right hemisphere's attachment system, including biological synchronicity.

Biological synchronicity is a profound phenomenon and is, of course, critical in mother-infant bonding, nursing and regulation of the physiology of both the mother and infant (Hofer, 1994). Yet, among adults, synchronicity is not traditionally considered an important factor in the regulation of physiology. But, what if the beneficial effects of healthy attachments like marriage and social support are mediated by the regulatory capacity of biological synchronicity? It is a well known observation that the menstrual cycles of young women who live together often become synchronized (McClintock, 1971).

If the capacity for biological synchronicity is inhibited when the self preservative system dominates as a result of chronic stress, could our response to chronic stress compromise our ability to be regulated by significant others? Could this be a mechanism for psychosomatic disease which is often the result of dysregulation or desynchronization of some physiologic system? (Mason, 1970; Hofer, 1994)

Lateralized Brain Function and "Eye Movement Desensitization Reprocessing" (EMDR)

Henry's idea that psychological trauma can cause a functional dissociation of emotional processing of the two hemispheres could be related to the success of a new technique in treating traumatic memories: eye movement desensitization reprocessing (EMDR) developed by Francine Shapiro. The technique involves holding a disturbing thought in mind while performing rapid lateral eye movements for about 20 seconds following a therapist's fingers (Shapiro, 1995). This technique has gained international attention for its effectiveness in reducing anxiety associated with a traumatic memory although the mechanism by which it works is still unknown. Henry thought that the rapid lateral eye movements performed during the hemispheric dissociation (while recalling a disturbing aspect of the memory) might actually reestablish integrative functioning of the hemispheres allowing reprocessing of the memory in a more balanced way, thereby providing relief from the anxiety evoked by the previous incomplete processing of the same memory.

Conclusion

In the last years of Henry's life, he came to an understanding of how traumatic stress can contribute to the development of insecure attachment and sought to describe this relationship in terms of lateralized brain function, physiology, psychology and evolutionary survival.

In his words:

"The ability to maintain personally relevant bonds is vital for our evolutionary survival. The infant's tie to the mother's voice and odor is recognized even by the newborn (Van Lancker, 1991), yet this personal relevance and recognition of the familiar can be impaired by anxious insecurity resulting from difficult early experiences or traumatic stress. The vital task of establishing a personally relevant universe and the solace derived from it depend on effective right hemispheric functioning. If this

function is indeed lost in the insecurely attached, much has been lost." (Henry, in press).

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