

# Memories of Fear

## How the Brain Stores and Retrieves Physiologic States, Feelings, Behaviors and Thoughts from Traumatic Events

Bruce D. Perry, M.D., Ph.D.

*The Child Trauma Academy*

[www.ChildTrauma.org](http://www.ChildTrauma.org)

This is an Academy version of a chapter originally appearing in “Splintered Reflections: Images of the Body in Trauma” (Edited by J. Goodwin and R. Attias) Basic Books (1999).

---

**Memory** -- the capacity to bring elements of an experience from one moment in time to another is the unique property of life forms. This remarkable property - the carrying of information across time - is the foundation of every biological process from reproduction to gene expression to cell division – from receptor-mediated communication to the development of more complex physiological systems (including neurodevelopment). To some degree, all of the organ systems in the human body have “memory.” This ability to carry elements of previous experience forward in time is the basis of the immune, the neuromuscular, and neuroendocrine systems. Through complex physiological processes, elements of experience can even be carried across generations. Elements of the collective experience of the species are reflected in the genome, while the experience of the individual is reflected in the expression of that genome.

No other biological system has developed more sophisticated capacity to make and store internal representations of the external world – and the internal world – than the human central nervous system, the human brain. All nerve cells ‘store’ information in a fashion that is contingent upon previous patterns of activity (see Singer, 1995; Thoenen, 1995). Neurons are specifically designed to modify in response to external cues (e.g., neurohormones, neurotransmitters, neurotrophic factors; Lauder, 1988). These neurophysiological and molecular neurobiological properties underlie all of the complex functions mediated by the brain (thinking, feeling and acting). During development, the cognitive, motor, emotional and ‘state’-regulating areas of the brain organize in response to experiences (see Perry, 1988; Brown, 1994; Perry 1997). And in each of the diverse brain systems which mediate specific functions, some element of previous experience is stored.

This storage involves complex neuromolecular processes – use-dependent changes in synaptic microarchitecture and intracellular alterations in various important chemicals involved in cellular communication and gene expression (see Kandel, 1989). The details – those that are known – are outside the scope of this chapter. Yet to understand that the physical properties of neurons change with experience is crucial to understanding

the concept of memory. Simply stated – the brain changes with experience – all experience, good and bad. The focus of this chapter is how the brain changes by storing elements of a traumatic experience.

The brain allows the individual to sense the external and internal environment, process this information, perceive and store elements of these sensations, and act to promote survival, and optimize our chances for successful mating – the key to survival of the species. In order to do this, the brain creates internal representations of the external world -- taking information that was once external to the organism, transforming this into patterned neuronal activity and, in a 'use-dependent' fashion, creating and storing these representations (e.g., Kandel and Schwartz, 1982; Maunsell, 1995). A further remarkable characteristic of this internal representation is that the brain makes and stores associations between the sensory information (e.g., sights, sounds, smells, positions, and emotions) from that specific event (e.g., the pairing of the growl of the sabertooth tiger and danger) allowing the individual to generalize to sensory information present in current or future events.

This chapter will discuss: 1) the process of making internal representations during traumatic events; 2) the development of associations specific to the traumatic event; 3) the generalization of these associations from trauma-specific cues to non-specific cues and 4) clinical implications of the storage and recall of trauma-related 'memories' during childhood.

## **The Brain: Development and Plasticity**

The human brain is an amazing organ which acts to sense, process, perceive, store (create memories) and act on information from the internal and external environment to promote survival. In order to carry out these functions, the human brain has evolved a hierarchical organization -- from the regulatory brainstem areas to the complex, analytical cortical regions (Figure 1). More simple, regulatory functions (e.g., regulation of respiration, heart rate, blood pressure, body temperature) are mediated by the 'lower' parts of the brain (brainstem and midbrain) and the most complex functions (e.g., language and abstract thinking) by its most complex cortical structures (see Loewy and Spyer, 1990; Goldstein, 1995). (FIGURE 1 HERE)

The structural organization and functional capabilities of the mature brain develop throughout life, with the vast majority of the critical structural organization taking place in childhood. Brain development is characterized by 1) sequential development and 'sensitivity' (from the brainstem to the cortex) and 2) 'use-dependent' organization of these various brain areas. The neurophysiological and neuromolecular mechanisms underlying this 'use-dependent' or activity-dependent neurodevelopment are the same which underlie the use-dependent development of 'memories'. Indeed, use-dependent development and the resulting organization of the brain are 'memories' - stored reflections of the collective experiences of the developing child.

For each of the hundreds of neurophysiological systems and areas of the brain, their mature organization and functional capabilities reflect some aspect of the quantity, quality and pattern of somato-sensory experience present during the critical organizational periods of development (see Bennett et al., 1964; Gunnar, 1986; Lauder, 1988; Perry; 1988; Brown, 1994; Singer, 1995; Perry et al., 1995; Perry, 1997). This use-dependent property of brain development results in an amazing adaptive malleability such that the brain develops capabilities suited for the 'type' of environment it is growing in (Perry et al., 1995) . Children reflect the world in which they are raised.

These various brain areas develop, organize and become fully functional at different stages during childhood. At birth, for example, the brainstem areas responsible for regulating cardiovascular and respiratory function must be intact while the cortical areas responsible for abstract cognition have years before they are required to be fully functional. Any brain area or system, once organized, is less sensitive to experience – less likely to change in response to experience – less plastic. It is of critical importance, then, that by age three, the vast majority of the brain has been organized. The brain of the three year old infant is ninety percent of adult size, while her body is only fifteen percent of adult size. The vast majority of brain development and organization takes place in these first few years of life.

The degree of brain plasticity is related to two main factors – the stage of development and the area or system of the brain. Once an area of the brain is organized, it is much less responsive to the environment – it is less plastic. For some brain areas such as the cortex, however, significant plasticity remains throughout life, such that experiences can continue to alter, easily, neurophysiological organization and functioning. A critical concept related to memory and brain plasticity is the differential plasticity of various brain systems. Not all parts of the brain are as plastic as others. Once the brain has organized (i.e., after age three), experience-dependent modifications of the regulatory system are much less likely than experience-dependent modifications of cortically-mediated functions such as language development (Figure 2).

(FIGURE 2, about here)

### **The Brain's Response to Threat**

The prime 'directive' of the human brain is to promote survival and procreation. Therefore, the brain is 'over-determined' to sense, process, store, perceive and mobilize in response to threatening information from the external and internal environments (see Goldstein, 1995). All areas of the brain and body are recruited and orchestrated for optimal survival tasks during the threat. This total neurobiological participation in the threat response is important in understanding how a traumatic experience can impact and alter functioning in such a pervasive fashion. Cognitive, emotional, social, behavioral and physiological residue of a trauma may impact an individual for years – even a lifetime.

In order for any experience, traumatic or not, to become part of memory, it must be 'sensed' -- it must be experienced by the individual. The first step in experiencing is sensation. The five senses of the human body have the amazing capacity to transform forms of energy from the external world (e.g., light, sound, pressure) into patterned activity of sensory neurons. The first 'stop' of this sensory input from the outside environment (e.g., light, sound, taste, touch, smell) and from inside the body (e.g., glucose levels, temperature) is the lower, more 'regulatory' parts of the brain -- brainstem and midbrain.

As the sensory input comes into the brain stem and midbrain, it is matched against a previously stored patterns of activation and if unknown, or if associated with previous threat, an initial alarm response begins (e.g., Aston-Jones, Ennis, Pieribone, Nickel, & Shipley. 1986). The alarm response begins a wave of neuronal activation in key brainstem and midbrain nuclei which contain neurons utilizing a variety of neurotransmitters (e.g., norepinephrine, dopamine, serotonin), neuromodulators and neuropeptides such as ACTH, corticotrophin releasing factor, vasopressin. At this point, the complex pattern of sensory neuronal activity associated with a specific visual image -- or in different areas of the midbrain, with a specific smell or sound -- make connections with neuronal networks in these levels of the brain.

A cascade of patterned neuronal activity is initiated in these primitive areas of the brain which moves up to more complex parts of the brain. In addition to sending these signals to higher parts of the brain, this cascade of activity also initiates a set of brainstem and midbrain 'responses' to the new information from the environment, allowing the individual to react in a near-reflexive fashion. In many instances, the brain's response to incoming sensory information will take place well before the signals can get to the higher, cortical parts of the brain where they are 'interpreted'.

Activation of these key systems results in patterns of neuronal activation which move from brain stem through mid brain, to thalamic, limbic and cortical areas. At the level of the brain stem and midbrain, there is very little subjective perception. It is at the level of the thalamus and the limbic areas that the actual sensation of anxiety arises. It is only after communication with cortical areas that the individual is able to make more complex, cognitive associations which allow interpretation of that internal state of anxiety (Singer, 1995).

Simply stated, then, the fear response will involve a tremendous mobilization and activation of systems distributed throughout the brain: terror involves cortical, limbic, midbrain and brainstem-based neurophysiology (see Gorman, Liebowitz, Fyer, & Stein, 1989). Because the neuronal systems alter themselves in a 'use-dependent' way in response to patterned, repetitive neuronal activation, a state of terror will result in patterned, repetitive neuronal activation in this distributed and diverse set of brain systems -- resulting in a set of 'memories.' In each of these areas -- mediating cognitive, motor, emotional and state-regulation -- elements of the traumatic event will be 'stored.' Memories of trauma have been created (see Terr, 1983; Pynoos and Nader, 1989; Schwarz and Kowalski, 1991; Schwarz and Perry; 1994).

This overview describes the sensing, storing and perceiving elements of the response to threat. At each level of the brain, as the incoming input is 'interpreted' and matched against previous similar patterns of activation, a response is initiated. The brain responds to the potential threat. This immediate response capability is very important for rapid response to potentially-threatening sensory signals – the classic example of this is the immediate motor action of withdrawal of the finger after being burned – or the jump that takes place following an unexpected loud sound (startle response). Clearly, in order for the brain to react in this immediate, 'un-interpreted' fashion, the more primitive portions of the brain (i.e., the brainstem and the midbrain) must 'store' previous patterns of sensory neuronal input which are associated with threat – there must be 'state' memories – memories of previous patterns of sensory input which were connected with a bad experience – the combat veteran from Vietnam will have an automatic response to the sound of a helicopter (e.g., Figure 5).

The classic 'response' to the threatening cues involves activation of the autonomic nervous system. Originally described by Cannon (Cannon. 1929; Cannon. 1914) this 'fight or flight' reaction involves the physiological manifestations of alarm, arousal and the emotion of anxiety (e.g., profuse sweating, tachycardia, rapid respiration). These physical symptoms are manifestations of activation of the autonomic nervous system and the hypothalamic-pituitary axis (HPA, see Giller, Perry, Southwick and Mason, 1990), again, an adaptive response to the impending threat.

The physiological hyper-reactivity of post-traumatic stress disorder is a cue-evoked 'state' memory (see Figures 3, 4 and 5). The brain has taken a pattern of neuronal activation previously associated with fear and now, will 'act' in response to this false signal. The 'recall' of traumatic state memories underlies many of the abnormally persistent characteristics of the once-adaptive response to threat (see Perry, in press; Perry 1993; Perry, 1994). This persistence of the 'fear' state and the ability of now non-threatening cues to become paired to a full blown threat response is related to the remarkable capacity of the human brain to make associations.

(INSERT FIGURE 3 about here)

### **The Brain's Ways of Categorizing Information: Association and Generalization**

Neuronal systems are remarkably capable of making strong associations between paired cues (e.g., the growl of a tiger and threat). Associations between patterns of neuronal activity and specific sensory stimuli take place in all brain areas, yet for complex associations involving the integration of multiple sensory modalities more complex brain areas (e.g., amygdala) are required, with the most complex associations taking place in cortical areas. Under ideal conditions, this capacity for association allows the brain to rapidly identify threat-associated sensory information in the environment, allowing the organism to act rapidly to promote long term survival (see Phillips and LeDoux, 1992). Yet the remarkable capacity of the brain to take a specific event and generalize, particularly with regard to threatening stimuli, makes humans vulnerable to the development of 'false' associations and false generalizations from a specific

traumatic event to other non-threatening situations. These processes are crucial to understanding memory and trauma.

Associations between neuronal patterns of activity derived from specific sensory cues are matched against a 'catalogue' of previous experiences. For example, in an individual with a history of traumatic experience, a simple rise in heart rate induced by a non-threatening experience (e.g., exercise; see Case 5) can trigger a brainstem-mediated alarm response if in that individual's past, the neuronal patterns of activation that occur with increased heart rate matched those associated with severe threat. The brain has stored this state memory-- and has generalized this neurophysiological pattern of activity to indicate threat.

Generalization is an adaptive process. It was far preferable for the vulnerable human to be too cautious, too hypervigilant and to over-read non-verbal cues of threat. Learning the association between the growl of the sabertooth and danger should only take one experience. Not many individuals who required more than one trial to learn this had a chance to pass on their genes. Indeed, it is likely that certain sensory cues are genetically-coded to induce an alarm state – as witness the pervasive nature of phobias to snakes or the stereotyped fashion in which infants will exhibit distress at loud, sudden auditory cues.

Because paired associations have been created in these regulatory and more 'primitive' parts of the brain, a pattern of incoming sensory information may be interpreted as 'danger' and acted upon in the brainstem, midbrain and thalamus milliseconds before the information gets to the cortex to be interpreted as 'harmless.' For a combat soldier from Vietnam, the sound of a firecracker will still elicit a 'fear' response (e.g., increased heart rate, startle response) even though he knows it is a firecracker. This man's brainstem has interpreted and acted on the information before it has had a chance to get to the cortex to be interpreted in a more complex fashion. Brainstem, midbrain and limbic associative capabilities are at the heart of these automatic trauma-related "flashback" responses – emotional, motor and state memories.

At each level of increasing complexity, the local associations become more complex. The associations in the brainstem are simple and categorical. Associations in the amygdala are more complex and allow interpretation of emotional signal and cues, including facial expressions -- and the intentionality they convey (threat, affiliation). Associations in the cortex are the most complex and may involve a variety of abstract elements -- associations between previously unpaired cues and various levels of 'meaning' can be made -- allowing abstract cognition. (INSERT FIGURE 4 about here)

In post-traumatic stress disorders, associations between specific complex cues (e.g., helicopters) may become linked to the limbically-mediated emotion (anxiety). Limbic activation may result from cortically-mediated images (e.g., interpretation of a specific event as potentially threatening, or imagining a specific traumatic event). Once these limbic areas are activated, there may (or may not be) activation of lower midbrain and brainstem areas involved in the response to threat -- the efferent wing of the alarm

response may or may not be activated. The degree of activation of the rest of the threat-response neurobiology residing in the midbrain and brainstem depends, to some degree, upon the 'sensitivity' of these systems. Indeed, it is likely that PTSD involves a sensitization of these systems to threat-related cues, internal or external (see below).

A sensitizing pattern of previous activation from a traumatic experience can dramatically change the sensitivity of the brain's alarm system (e.g., Kalivas et al., 1990). The result is a state of anxiety, even in the presence of what were originally non-threatening cues. A sensitized stress response apparatus then is likely a common etiology of trauma-related symptoms in children (see Perry et al., 1995). This is certainly the case for traumatized children where it has been demonstrated that exposure to chronic and repeated stressors literally alters a variety of brain stem related functions, including emotional and behavioral functioning (Perry, 1994; Perry, Southwick, & Giller, 1990).

### **Use-dependent Storage of Experience: Types of 'Memory'**

The brain changes in response to patterned neuronal activity (see Perry, 1988; Courschene, Chisum and Townsend, 1994; Perry et al., 1995). All parts of the brain are capable of changing in response to changes in neuronal activity -- hence, all parts of the brain 'store' information and have, in some sense, memory. While the majority of research on memory has been in the cognitive memory, and more recently, implicit and procedural memory, other brain areas, responsible for other functions, change in response to activation and, thereby, make memories. Categorization of these 'memories' constitutes a review of major brain-mediated functions. Cognitive memories arise from 'use-dependent' changes in neuronal patterns of activity present during cognition (e.g., learning names, phone numbers, language). Motor vestibular memories arise from 'use-dependent' activation of motor vestibular parts of the brain (e.g., riding a bicycle, typing, dancing, playing the piano). Emotional or affect memories result from 'use-dependent' changes in neuronal patterns of activity present during specific emotional experiences (e.g., grief, fear, mirth). Emotional memories may (see LeDoux et al., 1990) manifest as first impression or transference. State memories develop when a pattern of activation in state-regulating parts of the brain occurs that is sensitizing, chronic or prolonged (e.g., chronic domestic violence, traumatic stress). The specific nature of the 'memory' or storage and the 'kind' of information that is stored and recalled in each of these major areas (cognitive, emotional, motor, state) is different depending upon the specific function of that given area or system (see Selden et al., 1991; Shors et al., 1990).

During familiar routines the patterns of neuronal activity in the brain (especially in the regulatory systems in the brain) are familiar and similar to previously stored 'templates' of activation -- that is, in equilibrium. Traumatic events disrupt these patterns, causing disequilibrium. Patterns of neuronal activity present during the traumatic experience are unlike those present during routine daily activities and therefore will influence and alter functioning from the cortex (cognition) to the brainstem (core physiological state regulation).

Over time, a thought – recalling the trauma – may activate limbic, basal ganglia and brainstem areas – resulting in emotional, motor and arousal/state changes which are the functional residuals associated with the stored patterns of neuronal activation present in the original event (Greenwald, Draine and Abrams, 1996). Conversely, a state – arousal – may lead to activation of paired neuronal activity in the amygdala – resulting in an emotional change (see LeDoux et al., 1988) which may or may not be sufficient to activate associated cognitive memories. Indeed in many cases, the individual is completely unaware of ‘why’ they feel so fearful or depressed. The external or internal ‘triggers’ may not be something the person is aware of. It is the nature of the human brain to store experience. All experience. To generalize from the specific to the general. In children exposed to chronic abuse or neglect early in childhood, it is the rule that they will have little cognitive understanding (insight) of how the anxiety, impulsivity, social and emotional distress they suffer are related to the brain’s creation of ‘memories’ during previous traumatic experience.

Cognitive and affective (emotional) memory: The word memory, for most lay persons and for most mental health professionals, has come to signify some aspect of cognitive memory. In the recent past, the concept of emotional memory has received considerable attention, in large part due to the excellent research on the function of the amygdala conducted by investigators such as LeDoux (1988; 1989; 1990) and Davis (1992). The neurobiology and psychology of cognitive and affective memory has been reviewed extensively elsewhere (see Mesulam, 1990; Davis, 1992; LeDoux, Romanski, Xagoraris, 1989; Schacter, 1992; Squire, 1992; Siegel, 1996). The focus of the following discussion will be motor and state memory in relation to these other forms of memory (for more traditional discussion of motor and state memory see Knowlton, Maungels and Squire, 1996; Greenwald, Draine and Abrams, 1996).

Motor/vestibular and state memories: In the same way that patterned neuronal activity (use) builds in cognitive memories, so does certain motor activity -- playing piano, riding a bicycle. The motor vestibular movements of a roller coaster may elicit an internal state associated with the playful experiences of a small child being tossed in the air by a parent. The fetal position clearly elicits a sense of soothing and calm. During the calmest, safest, warmest, least threatening time in the history of the ‘brainstem’ the neuronal patterns of proprioception associated with the fetal position were associated with the neuronal patterns of this calm, warm, safe – most soothed state. Therefore, as a motor memory, when the child or adult rolls into the fetal position, the neuronal patterns this evokes can evoke some elements of that original soothed state. Very few people, feeling overwhelmed or sick, will lie on their backs in the ‘spread eagle’ position.

Similarly, a major organizing sensory pattern of the developing brainstem is the somatosensory pattern associated with maternal heart rate. During the crucial final trimester, when the neuronal apparatus of the brainstem is undergoing crucial



processes related to building the organizational capacity to regulate heart rate, blood pressure, body temperature and respiration by birth, the primary 'environmental' sensation is the repetitive, relentless and rhythmic sound and feel of mother's heart beating. The fetus' senses (vibratory and hearing) translate these maternal patterns into patterns of neuronal activation in the developing brain. And, as the brain is organizing in utero, these maternal patterns play a role as organizing templates for the brain. Is it any surprise then, that cross cultural studies demonstrate that mothers, in all cultures, rock children with the same frequency (see Hatfield and Rapson, 1993). This frequency is between 70 and 80 beats/min – the same as the resting mother's heart rate. This 'frequency' of soothing may be related to the use of similar patterns of sound and movement in a host of healing or soothing rituals - again, practices observed through history and across cultures.

One of the most powerful examples of the connections between a motor 'memory' and an 'emotional' and 'state' memory relate to oropharangeal motor activity – eating. For individuals fortunate enough to have an attentive, nurturing caregiver, eating as an infant (the time when the patterns of oropharangeal motor patterns related to eating are being built into the brain) becomes associated with eye contact, social intimacy, safety, calm, touch, cooing (e.g., Hatfield and Rapson, 1993). This wonderful, soothing and interactive somatosensory bath that the nurturing caregiver provides literally organizes and 'grows' the brain areas associated with attachment and emotional regulation (see Perry et al., 1995; Perry, 1997). Disruptions of this 'bath' by neglect, depression, trauma, or other chaotic, inconsistent experiences can result in abnormal development of the neurobiological systems and patterns of activity which are required for normal eating or relationship formation. Rumination and failure to thrive are classic clinical examples of the results of a severely disrupted maternal-infant somatosensory 'dance.' Another example of a disturbed motor memory related to eating is described in Case 3.

(INSERT FIGURE 5 about here)

### **Use-dependent Learning: State Dependent Storage and Recall**

As described above, the brain changes in a use-dependent fashion. All parts of the brain can modify their functioning in response to specific, repetitive patterns of activation. These use-dependent changes in the brain result in changes in cognition (this, of course, is the basis for cognitive learning), emotional functioning (social learning), motor-vestibular functioning (e.g., the ability to write, type, ride a bike) and state-regulation capacity (e.g., resting heart rate). No part of the brain can change easily without being activated -- you can't teach someone French while they are asleep or teach a child to ride a bike by talking with them.

Mismatch between modality of teaching and the 'receptive' portions of a specific child's brain occur frequently. This is particularly true when considering the learning experiences of the traumatized child -- sitting in a classroom in a persisting state of arousal and anxiety -- or dissociated. In either case, the child is essentially unavailable to process efficiently the complex cognitive information being conveyed by the teacher.

This principle, of course, extends to other kinds of 'learning' -- social and emotional. The traumatized child frequently has significant impairment in social and emotional functioning. Social and emotional capabilities are learned; they develop in response to experience -- experiences which these children often lack -- or fail at. Indeed, hypervigilant children frequently develop remarkable non-verbal skills in proportion to their verbal skills (street smarts). It is common that they over-read (misinterpret) non-verbal cues -- eye contact means threat, a friendly touch is interpreted as an antecedent to seduction and rape -- accurate in the world they came from but now, hopefully, out of context. During development, these children spent so much time in a low-level state of fear (mediated by brainstem and midbrain areas) that they were focusing consistently on non-verbal cues. In our clinic population, children raised in chronically traumatic environments demonstrate a prominent Verbal-Performance split on IQ testing (n = 108; WISC Verbal = 8.2; WISC Performance = 10.4, Perry, in preparation).

This is consistent with the clinical observations of teachers that these children are 'bright' but can't learn easily. Often these children are labeled as learning disabled. These difficulties with cognitive organization contribute to a more primitive, less mature style of problem-solving -- with aggression often being employed as a "tool".

This principle is critically important in understanding why a traumatized child -- in a persisting state of arousal -- can sit in a classroom and not learn (see Figures 3 and 4). At rest, the brain of this child has different areas activated -- different parts of the brain 'controlling' his functioning. The capacity to internalize new verbal cognitive information depends upon having portions of the frontal and related cortical areas being activated -- which, in turn, requires a state of attentive calm (see Castro-Alamancos and Comori, 1996). A state the traumatized child rarely achieves (Perry et al., 1995).

Children in a state of fear store and retrieve information from the world differently than children who feel calm (see Eich, 1995; Kim and Fanslow, 1992; McNally et al., 1990). We all are familiar with 'test' anxiety. Imagine what life would be like if all experiences evoked the persisting emotion of anxiety. If a child has information stored in cortical areas but in the specific moment is very fearful, this information is inaccessible. In this regard, cognitively-stored information does little good in the life-threatening moment. Simple didactic conflict-resolution models are doomed to fail unless they involve elements of role-playing. Imagine how much you would trust an Army that went through combat training by sitting in classroom -- or the E.R. physician about to run her first code after only learning how to do that by reading a book. In the midst of most threatening experiences -- situations where violence often takes place -- the 'problem-solving' information in the cortex is not easily accessed. It is of interest to note that information learned in song, rhyme or rap is more easily recalled when in a state of high arousal. This is due, of course, to the fact that this information is stored in a different fashion than traditional verbal cognitive information.

## **Traumatic Memories: Case Examples**

Case 1: State and affect memories elicited in a non-conscious state.

*D. is a 9 yo boy. He was victim of chronic and pervasive physical threat and abuse from this biological father. From the age of 2 until 6 he was physically and sexually abused by his father. At age 6 he was removed from the family. His mother acknowledged pervasive abuse.*

*At age 8, he was seriously injured in a fall. He suffered from serious brain injury such that he was in a coma for 8 months following the injury. He continues to be difficult to arouse, is non-verbal, no form of meaningful communication is noted. In the presence of his biological father, he began to scream, moan, his heart rate increased dramatically. Audiotapes of his biological father elicit a similar response.*

Clearly the sensory information (sounds, smells) which was associated with father was reaching this child's brain in a way that elicited a 'state' memory. This child's brain did not have the capacity to have conscious perception of the presence of his father. However, the auditory and olfactory sensory neuronal patterns which entered this boy's brain were associated, even at the level of his brainstem and midbrain, with past states of fear. Therefore, with exposure to this unique pattern, the boy's brain sensed, processed and 'acted' on the information despite the fact that higher areas of his brain were damaged and incapable of full function. Exposure to his father elicited no cognitive, narrative memory; his agitation and increased heart rate were manifestations of affective and 'state' memories which were the products of many years of traumatic terror which had become associated with his father – and all of his father's attributes.

Case 2: State and affect memory with no 'associated' cognitive memory:

*F. is a 15 yo male. From birth to age 8, he was continually exposed to severe physical abuse from his biological father. He witnessed many episodes of his mother being severely beaten by this father. As he grew older, he attempted to intervene and was seriously injured on several occasions.*

*At age 8, his mother left his father. From that time forward, he was noted to be quiet, withdrawn and, in school, he 'tuned out' and was noted to be daydreaming. At age 10, he began having syncopal episodes of unknown origin. He received multiple evaluations by neurologists and cardiologists. We were to 'rule out' psychogenic causes of his fainting. On initial evaluation, the mother related the long history of domestic violence, abuse and a series of medical work-ups for a host of non-specific symptoms including headaches, fainting, 'seizures', chest pains and chronic emotional and behavioral problems.*

*His resting heart rate was 82. When asked about his biological father, his heart rate fell to 62 and he became very withdrawn. On a walk in the hall, we asked about his abuse and his heart rate fell below 60 and he fainted. He was placed on Trexan, an opioid*

*receptor antagonist, with a marked decrease in his syncope. He has persisting problems.*

This child exhibited a clear sensitization of the neurobiological systems involved in the dissociative response to threat (see Figure 3). Sensitization of the brainstem catecholamine systems are involved in the hyperarousal, hypervigilance, startle response and sympathetic nervous system hyper-reactivity in post-traumatic stress disorder (PTSD: Perry, Southwick and Giller, 1990). Similar sensitization of the opioid systems involved in dissociation is likely to account for the symptoms of cue-specific bradycardia, syncope, 'daydreaming,' and a host of other dissociation-related signs and symptoms (Perry et al., submitted).

Case 3: Motor memories evoking state and affect memories.

*I first saw him in the basement cafeteria-style 'lunchroom at the Residential Treatment Facility. He -- small, wiry, herky-jerky, nose-running, dirty shirt sleeves, always out of chair, run on speech, T. -- sat with six other young boys at a round table covered in an institutional plastic, red-checkerboard tablecloth. It was lunch-time and all of the children were eating hot dogs, beans and potato chips. All except him.*

*I was a new consultant to this Center. Sixty children, the majority 'in the system' after being removed from their abusive families and failing in an escalating series of 'least restrictive' placements -- foster family to another foster family to a psychiatric hospital to a therapeutic foster home back to the hospital to a residential treatment center for six months to a different foster family to this Residential Treatment Facility. Failed placements, failing system.*

*T. stood out because he was loudly demanding that someone 'cut my hot dog -- cut my hot dog -- cut my hot dog'. A chant, a pressured, almost psychotic chant. A staff member came up to him and chided him for being 'a baby' about not eating the hot dog without it being cut. The staff member, with some good intentions, felt that this was the time to take a stand and make T. 'grow up'.*

*"T. its time you act your age. See all the other kids are eating without me cutting this up. I won't cut it up." T. escalated, shouting louder, frantic. The staff member stood his ground. T. rose from his chair, the staff member commanded him to stay at the table. The confrontation ended with T. a sobbing, hysterical, out of control, child being physically restrained by two staff. He was led off to a quiet place -- to re-group, re-organize and, in some sense, to re-develop, emerging from his primitive, terrified*

*disorganized state through various levels of psychological, cognitive and emotional organization back to his most mature level of functioning.*

*“You can’t indulge this kind of demanding behavior.” The staff member said to me as they carried T. from the cafeteria. The other children seemed familiar with these confrontations -- and with the resulting physical restraint. They kept eating. One of the children at his table looked at me and said, “ This always happens when we have hot dogs.”*

*Over the years I worked there, I came to see that T. would cut his bananas, he would take popsicles off of the stick and eat them with a spoon. He had a number of other ‘unusual’ or bizarre eating habits. He had a host of swallowing ‘difficulties’. He needed to eat soft food -- rarely eating foods that were solid. He chewed forever and frequently gagged. While T. could tell anyone what these habits were, he had no idea why he had to do things that way.*

*“I just have to”.*

*“ And if you don’t ?”*

*“I just get angry”*

*“ Angry ?”*

*“Well, I guess. Maybe scared. Mixed up .....I don’t know .”*

*T was an 8 yo boy. He had been forced to fellate his father from birth. And later, other men. Many other men. He was very young when this happened -- from birth on -- at age 6 he was finally taken from this life of pervasive, socialized abuse.*

*Normal oropharyngeal ‘patterns’ of stimulation during development (primarily from eating) are associated with the development of normal eating and swallowing capabilities. Furthermore, these patterns of oropharyngeal stimulation take place in association with caretaker’s soothing touch, and gaze, and smell and warmth and the satiey of being fed. This should be one of the most soothing, comforting positive sets of experiences an individual will have -- and it follows us through life. Eating involves ‘trainable’ neuromuscular events -- motor memories, if you will, -- and these motor memories are linked to positive emotional, olfactory, gustatory and cognitive memories.*

*But for T., the development of oropharyngeal stimulation was associated with other things -- fear, pain, gagging, suffocating in the flesh of a pedophile. No satiety, no calm, no comfort. Rather than the soothing*

*warmth of the maternal breast, his brain internalized the confused, inconsistent, painful states associated with his abuse. Solid food in his mouth, his throat, evoked the state memories ingrained during the critical formative stages of his first six years. Eating for T. evokes fear and confusion -- he has to eat to survive-- there is some positive effect of eating but often enough, the evocative nature of the meal can erase these positive effects.*

*With each meal, some small part of T. relives the abuse of his early childhood, some set of deeply burned-in state memories are accessed. These rarely, if ever, come to his awareness as a 'cognitive' memory -- he will likely never be able to have the insight to make the association between his eating habits and his early abuse.*

*Each meal scratches at the slowly healing scars of his childhood.*

*He remains small for his age. (from Perry, in press)*

Case 4: Cognitive narrative memory evokes affect, motor and state memory.

*C, a 5 year old child, witnessed her father shoot and kill her mother and then himself. She started attending our clinic within days after the event. She came to the clinic on a regular basis. Five weeks later, during a one hour free-play therapy session, a semi-structured PTSD interview was conducted (see Figure 6). When asked about the worst thing that ever happened to her, she had a marked alteration in her facial expression, stopped playing, moved her face and head away from the interviewer and stared into space. After a long moment, she stated, "I wanted to stay up late last weekend and have pizza, but I had to go to bed." The rest of the interview was characterized by single word responses which minimized her distress or the traumatic nature of her parent's death. (FIGURE 6 about here)*

This child exhibited a classic dissociative response when evocative cognitive cues of the murder/suicide were brought up. This dissociative state was protective and was evoked – a state memory – by merely thinking about the events. Her mood, motor movements and state of physiological regulation all were altered by this narrative memory. Narrative recall 'memory' was strongly linking to affect, motor and state memories in this child, a common finding in individuals following trauma (see Burke, Heuer and Reisberg, 1992).

Case 5: State memories evoking cognitive and affect memories

*T. is an 18 yo female. She was brutally raped at age 17. She had no previous history of psychiatric symptoms or treatment. She was seen within a month after her rape for symptoms related to an acute trauma response (e.g., anxiety, sleep problems, guilt, dysphoria). A prominent memory of the event was "I felt my heart was going to burst it was beating so hard. I felt it pounding against my chest." Prior to the event, she had exercised on a regular basis, deriving significant pleasure from the activities. Since the time of the rape, she had been unable to exercise. At multiple points during the treatment, she was free of anxiety, felt energetic and returned to exercising. She would immediately find herself having intrusive ideations of the rape, dysphoria and anxiety. Ultimately the connection between her increased heart rate and the affective and cognitive memories of the rape was identified. Once the recognition was made that increased heart rate would trigger a cascade of emotions and recollections, she was able to guide herself through a form of progressive desensitization. Over time she was able to disconnect increased heart rate from the distressing affect and the cognitive recollections of the rape.*

This example of a 'state' memory eliciting intense affect and cognitive memory illustrates powerful associations that can exist between various neuronal systems and functions that are co-activated during a trauma. In many cases, the state-elicited distress is not associated with a clear cognitive recollection, nor is the manifestation of the symptom proximal in time to the trauma. In these situations, it is often very difficult to make the connections which allow for effective therapeutic interventions. Indeed, it is likely that many 'states' of distress are activated by accessing state or affect memories without any clear cognitive or narrative associations to a specific trauma or experience. This is very common in young children who are pre-verbal or cognitively immature. Often their behaviors or symptoms are never connected to past experiences and traumatized children may get labeled as having Attention Deficit Hyperactivity Disorder or Conduct Disorder based merely upon the current presentation of symptoms. These diagnostic labels, while often accurate, do not give the clinician any clues to etiology, prognosis or treatment approach that would be suggested by knowing the relationships between past trauma and current functioning.

Case 6: State memory in a pre-verbal child:

*X is a 3 year old boy referred to our clinic following the murder of his 18 month old sister. X witnessed the murder of his 18 month old sister by a caretaker. Over a series of non-intrusive clinical contacts, X developed a sense of familiarity and comfort at our clinic. Approximately two months after the event, a semi-structured interview was conducted. During the non-intrusive part of the interview, X was spontaneous, interactive, smiling and age-appropriate in his play. When the direct questioning began (see Figure 7), his heart rate increased but his behaviors remained constant. Within 5 seconds of being asked about his sister, his heart rate dramatically increased and his play stopped. He broke eye-contact, physically slowed and became essentially non-responsive. This dissociative response was accompanied by a decrease in his heart*

*rate to the previous baseline level of the free play portion of the interview. Similar alterations in heart rate and induction of a 'protective' dissociative response could be elicited by exposure to cues associated with the murder.*

In this situation, this child, upon direct questioning, gave no verbal or narrative information about the event. This 'lack' of a narrative memory, however, did not mean that the child had not 'stored' the experience. Clearly, he demonstrated clear and unambiguous evidence of emotional and state memories when verbal or non-verbal cues were used to evoke the event (Figure 7).

(INSERT FIGURE 7 about here)

### **Traumatic Memory and the Law: Vulnerable Children**

Specific problems are posed by the child narrative when it is part of a forensic proceeding. The focal conflict is that the law is a primarily verbal domain while communication in children – particularly when communicating about a traumatic event – is primarily non-verbal. In the law words are considered the only essential element of a narrative. In the traumatized child, the narrated words are mere shadows of what is being communicated as they recall the event. The child's recall of a traumatic event involves not just the narrative shards as recalled using cognitive memory but also the intense fear of the emotional memory, the motor agitation of the motor memories and the physiological arousal (or dissociative response) of the state memory. Yet the syntax, semantics and grammar of these non-cognitive narrations do not yet have the standing in court that the syntax, semantics and grammar of verbal language does.

Learning the language of trauma and translating the verbal and non-verbal elements of this language will require many more years of investigation. Yet, as this investigation is underway, it is the task of all of us working with maltreated children to educate our peers and the rest of society that this language exists (e.g., Briere and Conte, 1993; Ceci and Bruck, 1993). To educate our society that traumatic events, like other experience, change the brain. Further, that the brain stores elements of the traumatic events as cognitive memory, motor memory, emotional memory and state memory, altering the functional capacity of the traumatized individual. And, in the end, by robbing the individual potential of millions of children each year, childhood trauma and neglect robs the potential of our families, our communities and our societies.

### **Trauma, Memory and History: Transgenerational Memory of Culture and Society**



Traumatic events impact millions of children and adults across the world each year. War, rape, floods, earthquakes, physical abuse, neglect --- all create memories for individuals, for families, for communities and for societies. The memory of trauma is carried not only through the life of the individual by their neurobiology but it is carried in the life of a family through family myths, childrearing practices and belief systems. Major traumatic events in the history of a people or culture become memorialized, as well, and carried forward across generations in our literature, our laws and our very social structures.

It is the unique property of living systems to carry forward elements of past experience – indeed, for all living systems, the present is contingent upon and a reflection of that past experience. In a very true sense, a body collective – a group – is a living, dynamic system. And, as the individual carries its own history forward using the apparatus of neurobiological mechanisms related to memory, each living group carries its memories forward in time. Yet living groups – families, clans, societies – carry this information forward using different mechanisms of recording and storage.

Over the history of humankind, the methods for recording and storing the experiences of the group have evolved. In our distant past, humans living groups passed experience from generation to generation using oral tradition – and sociocultural practices – language, arts, belief systems, rules, law – all were reflections of the past – and with each generation, modification, amendment, and alteration of the past ‘memory’ was modified by present experience. With the development of written language, information could be passed across generations more efficiently. Sociocultural advance occurred at an increased rate, made possible by more efficient ‘remembering’ of the lessons (good and bad) from the past. The ‘brain’ of humankind – the libraries of the world -- kept ‘civilization’ alive through its darkest moments – and even if generation after generation during a given period in history did not take advantage of this ‘memory’ – the information was not lost to humankind.

Later in history, again, with the introduction of the printing press, the past was more efficiently stored and passed on. Books became available for everyone. More people became literate. Information of all sorts – arts, science, social studies-- was stored in books. Again, a tremendous advancement in human sociocultural evolution can be traced to this process – to literacy and widespread education. Information from the past – primarily cognitive information – enriched the present. The rate of creativity was accelerated; invention and innovation – new ideas, machines, products, processes – were facilitated by the more efficient sociocultural ‘memory’ allowed by books and literacy. Now, in the span of a lifetime, the accumulated and distilled experience of thousands of generations could be absorbed – and acted upon to create sociocultural advances.

And now, we are in the first generations of a new era of recording, storing and transmitting information – electronic media – tapes, photographs, videos, films – all immortalize the experiences of humankind. The electronic media allow a unique and

different form for the memory of an individual, family, community and society to pass from generation to generation.

There is great hope for humankind in these advances. In the past, the inefficient methods of recording, storing and passing on the horror of war, rape, neglect, abuse, starvation, misogyny, slavery – allowed these lessons of living to be edited, modified, distorted and, with tragic consequences, forgotten. Only elements of the experience of war were passed across generations – the heroism of an individual, the success of the nation -- and the emotional ‘memory’ of war – the hate, rage, death, loss – has been transformed, altered and, all too often, forgotten.

Creative artists have always played the role of ‘emotional’ memory for a culture. In ways that standard recording of simple facts and figures cannot convey, a painting, poem, novel, or film can capture the emotional ‘memory’ of an experience. But in a society where access to and ‘artistic’ literacy is low, the emotional lessons of the past are easily lost. And when the last veteran of each distant war died, an element of the emotional ‘memory’ of that horror died as well. Unable to carry the emotional memory of war to the next generation – history could much more easily repeat itself – or more honestly, we could much more easily repeat history. But with documentary and creative film and video, which can convey both the fact and the emotion, maybe it will be harder for us to forget the past – and we, therefore, will be not so doomed to repeat it.

Yet the ever present danger of recording, storing and passing on false images, false stories, false history can be equally destructive. The responsible use of film, video, electronic storage may allow us to use these advances to promote and pass on those qualities which create, sustain and grow our humanity and, over many generations, to leave behind those qualities which rob our humanity (racism, misogyny, factionalism).

Can we change our world to create fewer traumatic memories to carry into the next generations – fewer traumatic events to shape our children who will create our future social structures ?

How can we heal the scars of individual and group trauma that haunt us today ? Can we ever make racism, misogyny, maltreatment of children – distant memories ? There are solutions. These conditions are not the inevitable legacy of our past. When an individual becomes self-aware, there is the potential for insight. With insight comes the potential for altered behavior. With altered behavior comes the potential to diminish the transgenerational passage of dysfunctional or destructive ideas and practices.

And so it must be for groups. As a society, we cannot develop true insight without self-awareness. Enduring socio-cultural changes in racism, misogyny and maltreatment of children cannot occur without institutional and cultural insight and the resulting altered institutional and cultural behavior. The challenge for our generation is to understand the dynamics and realities of our human living groups in a way that can result in group insight – which, inevitably, will lead to the understanding that we must change our institutionalized ignorance and maltreatment of children.

## Acknowledgments

This work was supported, in part, by grants from CIVITAS Initiative, the Hogg Foundation, the Children's Crisis Care Center of Harris County, Maconda O'Connor and Anonymous X.

## References

Aston-Jones, G., Ennis, M., Pieribone, J., Nickel, W.T., & Shipley, M.T. 1986. The brain nucleus locus coeruleus: restricted afferent control over a broad efferent network. *Science, Vol. 234, 734-737.*

Bennett, E. L., Diamond, M. L., Krech, D., & Rosenzweig, M. R. (1964). Chemical and anatomical plasticity of the brain. *Science, 146, 610-619.*

Briere, J. & Conte, J. Self-reported amnesia for abuse in adults molested as children. *Journal of Traumatic Stress, 6, 21-31, 1993.*

Brown, J. W. (1994). Morphogenesis and mental process. *Development and Psychopathology, 6, 551-563.*

Burke, A., Heuer, F & Reisberg, D. Remembering emotional events. *Memory & Cognition, 20, 277-290, 1992.*

Cannon, W.B. (1914). The emergency function of the adrenal medulla in pain and the major emotions. *American Journal of Physiology, 33, 356-372.*

Cannon, W.B. (1929). *Bodily changes in pain, hunger, fear and rage..* New York: Appleton.

Castro-Alamancos, M.A., & Connors, B.W. 1996. Short-Term Plasticity of a Thalamocortical Pathway Dynamically Modulated by Behavioral State. *Science, Vol. 272, 274-276.*

Ceci, S.J. & Bruck, M. Suggestibility of the child witness: a historical review and synthesis. *Psychological Bulletin, 113, 403-439, 1993.*

Clugnet, M.C., & LeDoux, J.E. (1990). Synaptic plasticity in fear conditioning circuits: induction of LTP in the lateral nucleus of the amygdala by stimulation of the medial geniculate body. *J Neurosci, Vol. 10, 2818-2824.*

Courchesne, E., Chisum, H., & Townsend, J. (1994). Neural activity-dependent brain changes in development: Implications for psychopathology. *Development and Psychopathology*, 6(4), 697-722.

Davis, M. The role of the amygdala in fear and anxiety. *Annual Review of Neuroscience*, 15, 353-375, 1992.

Eich, E Searching for mood dependent memory. *Psychological Science*, 6, 67-75, 1995.

Frankel, M: Adult reconstruction of childhood events in the multiple personality literature. *American Journal of Psychiatry*, 1993. 150: p. 954-958.

Giller, E.L., Perry, B.D., Southwick, S., & Mason, J.W. (1990). Psychoneuroendocrinology of post-traumatic stress disorder. In M.E. Wolf & A.D. Mosnaim (Eds.), *Post-traumatic Stress Disorder: Etiology, Phenomenology and Treatment*. (pp. 158-170). Washington, D.C. American Psychiatric Press, Inc.

Goldstein, D.S. (1995). *Stress, Catecholamines and Cardiovascular Disease*. New York: Oxford University Press.

Gorman, J.M., Liebowitz, M.R., Fyer, A.J., & Stein, J. (1989). A neuroanatomical hypothesis for panic disorder. *American Journal of Psychiatry*, Vol. 146, 148-162.

Greenwald, A.G., Draine, S.C., Abrams, R.L. Three cognitive markers of unconscious semantic activation. *Science*, 273, 1699-1702, 1996.

Gunnar, M.R. (1986). Human developmental psychoneuroendocrinology: A review of research on neuroendocrine responses to challenge and threat in infancy and childhood. In M.E. Lamb, L.A. Brown, & B. Rogoff (Eds.), *Advances in developmental psychology*. (pp. 51-103). Hillsdale, NJ: Lawrence Erlbaum.

Hatfield, E and Rapson, R. (1993) Love and attachment processes. In M. Lewis and J.M. Haviland (Eds.), *Handbook of Emotions*. (pp. 595-605) , New York, The Guilford Press.

Kandel, E. R. (1989). Genes, nerve cells and remembrance of things past. *Journal of Neuropsychiatry and Clinical Neurosciences*, 1, 103-125.

Kim, J and Fanslow M: Modality specific retrograde amnesia of fear. *Science*, 1992. 256: p. 675-677.

Knowlton, B.J., Mangels, J.A., Squire, L.R. A neostriatal habit learning system in humans. *Science*, 273, 1399-1401, 1996

Lauder, J. M. (1988). Neurotransmitters as morphogens. *Progress in Brain Research*, 73, 365-388.

Kalivas, P.W., Duffy, P., Abhold, R., & et.al. (1990). Sensitization of mesolimbic dopamine neurons by neuropeptides and stress. In P.W. Kalivas & C.D. Barnes (Eds.), (pp. 119-124). Caldwell, N.J. Telford Press.

Kandel, E.R. (1989). Genes, nerve cells and remembrance of things past. *Journal of Neuropsychiatry and Clinical Neurosciences*, 1, 103-125.

Kandel, E.R., & Schwartz, J.H. (1982). Molecular biology of an elementary form of learning: modulation of transmitter release by cyclic AMP. *Science*, 218, 433-443.

LeDoux, J.E., Cicchetti, P., Xagoraris, A., & Romanski, L.R. (1990). The lateral amygdaloid nucleus: Sensory interface of the amygdala in fear conditioning. *Journal of Neuroscience*, Vol. 10, 1062-1069.

LeDoux, J.E., Iwata, P., Cicchetti, D., & Reis, D.J. (1988). "Different projections of the central amygdaloid nucleus mediate autonomic and behavioral correlates of conditioned fear.". *Journal of Neuroscience*, Vol. 8, 2517-2529.

LeDoux, J.E., Romanski, L., & Xagoraris, A. (1989). Indelibility of subcortical emotional memories. *Journal of Cognitive Neuroscience*, 1, 238-243.

Loewy, A and Spyer K, Central Regulation of Autonomic Functions. 1990, New York: Oxford University Press.

Madison, D.V., Malenka, R.C., & Nicoll, R.A. (1991). Mechanisms underlying long-term potentiation of synaptic transmission. *Annu Rev Neurosci*, Vol. 14, 379-397.

Maunsell, J.H.R. (1995). The brain's visual world: representation of visual targets in cerebral cortex. *Science*, 270, 764-769.

McNally, R.J., Kaspi, S.PI, Riemann, B.C. & Zeitlin, S.B. Selective processing of threat cues in posttraumatic stress disorder. *Journal of Abnormal Psychology*, 99, 398-402, 1990.

Mesulam, M: Large scale neurocognitive networks and distributed processing for attention, language and memory. *Annals of Neurology*, 1990. 28: p. 597-613.

Perry, B.D. (1988). Placental and blood element neurotransmitter receptor regulation in humans: potential models for studying neurochemical mechanisms underlying behavioral teratology. *Progress in Brain Research*, 73, 189-206.

Perry, B: Neurodevelopment and the neurophysiology of trauma: (I) conceptual considerations for clinical work with maltreated children. *The APSAC Advisor*, 1993. 6: 1-12.

Perry, B. D. (1994). Neurobiological sequelae of childhood trauma: post-traumatic stress disorders in children. In M. Murberg (Ed.), *Catecholamines in Post-traumatic Stress Disorder: Emerging Concepts*. (pp. 253-276). Washington, D.C. American Psychiatric Press.

Perry, B.D. Neurodevelopmental Aspects of Childhood Anxiety Disorders: Neurobiological Responses to Threat In: *Textbook of Pediatric Neuropsychiatry* (CE Coffey & RA Brumback, Eds) American Psychiatric Press, Inc, Washington, DC, in press

Perry, BD Incubated in Terror: Neurodevelopmental Factors in the 'Cycle of Violence' In: *Children, Youth and Violence: The Search for Solutions* (J Osofsky, Ed), Guilford Press, New York, 1997

Perry, B. D. (in press). *Maltreated Children: Experience, Brain Development and the Next Generation*. New York and London: W.W. Norton.

Perry, B., Arvinte, A., Marcellus, J. and Pollard, R. (submitted) Syncope, bradycardia, cataplexy and paralysis: Sensitization of an opioid-mediated dissociative response following childhood trauma.

Perry, B. D., Pollard, R. A., Baker, W. L., Sturges, C., Vigilante, D., & Blakley, T. L. (1995). Continuous heartrate monitoring in maltreated children [Abstract]. Annual Meeting of the American Academy of Child and Adolescent Psychiatry, New Research,

Perry, BD, Pollard, R, Blakely, T, Baker, W, Vigilante, D Childhood trauma, the neurobiology of adaptation and 'use-dependent' development of the brain: how "states" become "traits". *Infant Mental Health Journal* 16 (4): 271-291, 1995

Perry, B, Southwick S, and Giller E: Adrenergic receptor regulation in post-traumatic stress disorders, in *Advances in psychiatry: biological assessment and treatment of post-traumatic stress disorder*, E. Giller, Editor. 1990, American Psychiatric Press: Washington, D.C. , pp .

Phillips, R.G., & LeDoux, J.E. (1992). Differential contribution of amygdala and hippocampus to cued and contextual fear conditioning. *Behav Neurosci*, Vol. 106, 274-285.

Pynoos, R and Nader K: Children's memory and proximity to violence. *Journal of the American Academy of Child and Adolescent Psychiatry*, 1989. 28: p. 236-241.

Schacter. D. L. Understanding implicit memory: a cognitive neuroscience approach. *American Psychologist*. 1992, 47, 559-569.

Schwarz, E and Kowalski J: Malignant memories: Posttraumatic stress disorder in children and adults following a school shooting. *Journal of the American Academy of Child and Adolescent Psychiatry*, 1991. 30: p. 937-944.

Schwarz, E, Kowalski J, and McNally R: Malignant memories: Posttraumatic changes in memory in adults after a school shooting. *Journal of Traumatic Stress*, 1993b. 6: p. 95-103.

Schwarz, E.D., & Perry, B.D. (1994). The post-traumatic response in children and adolescents. *Psychiatric Clinics of North America*, 17 (2): 311-326.

Selden, N.R.W., Everitt, B.J., Jarrard, L.E., & Robbins, T.W. (1991). Complementary roles for the amygdala and hippocampus in aversive conditioning to explicit and contextual cues. *Neuroscience*, Vol. 42, 335-350.

Shors, T.J., Foy, M.R., Levine, S., & Thompson, R.F. (1990). Unpredictable and uncontrollable stress impairs neuronal plasticity in the rat hippocampus. *Brain Res Bull*, Vol. 24, 663-667.

Siegel, D.J. Cognition, memory and dissociation. *Child and Adolescent Psychiatric Clinics of North America*, 5, 509-536, 1996.

Singer, W. (1995). Development and plasticity of cortical processing architectures. *Science*, 270, 758-764.

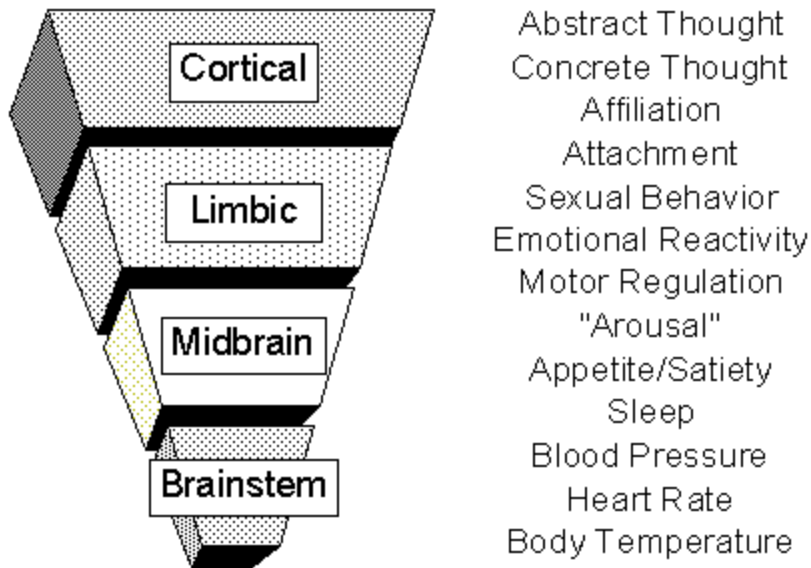
Squire, L. Declarative and non-declarative memory: multiple brain systems supporting learning and memory. *Journal of Cognitive Neuroscience*. 1992, 4: 232-243.

Terr, L: Chowchilla revisited: the effects of psychic trauma four years after a school-bus kidnapping. *American Journal of Psychiatry*, 1983b. 140: p. 1543-1550.

Terr, L: Childhood traumas: An outline and overview. *American Journal of Psychiatry*, 1991. 148: p. 10-20.

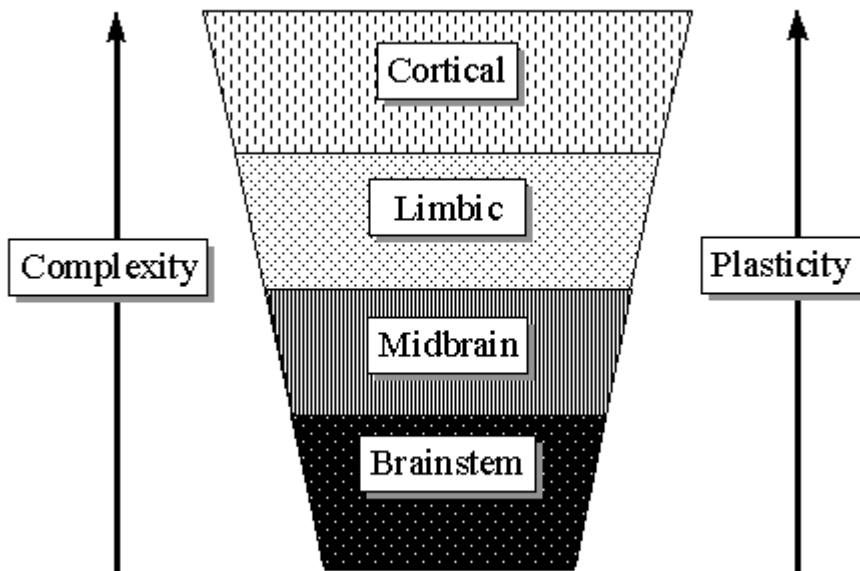
Thoenen, H. (1995). Neurotrophins and neuronal plasticity. *Science*, 270, 593-598.

## Figures

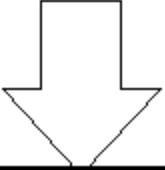


**Figure 1. Hierarchy of brain function.** The human brain is organized from the most simple (e.g., fewest cells: brainstem) to most complex (e.g., most cells and most synapses: frontal cortex). The various functions of the brain, from most simple and reflexive (e.g., regulation of body temperature) to most complex (e.g., abstract thought) are mediated in parallel with these levels. These areas organize during development and change in the mature brain in a 'use-dependent' fashion. The more a certain neural system is activated, the more 'built in' this state becomes-- creating an internal representation of the experience corresponding to this neural activation. This use-dependent capacity to make internal representations of the external or internal world is the basis for learning and memory.





**Figure 2. Plasticity and brain organization:** Once the human brain has organized in childhood, it remains capable of changing in response to experience. Yet not all parts of the brain are equally 'changeable.' With relatively brief cognitive experiences we can learn a new phone number but it is much more difficult to make a new 'motor' memory. To make a new 'state' memory - to modify the brainstem, however - is even more difficult. Prolonged or repeated activation of the neurophysiology of stress or alarm must occur to make state memories (see text). This, of course, is the reason that therapeutic activities require prolonged and repetitive experience. The parts of the brain in which the dysfunctional symptoms arise are often midbrain and brainstem, which are relatively more difficult to modify than the cortex.



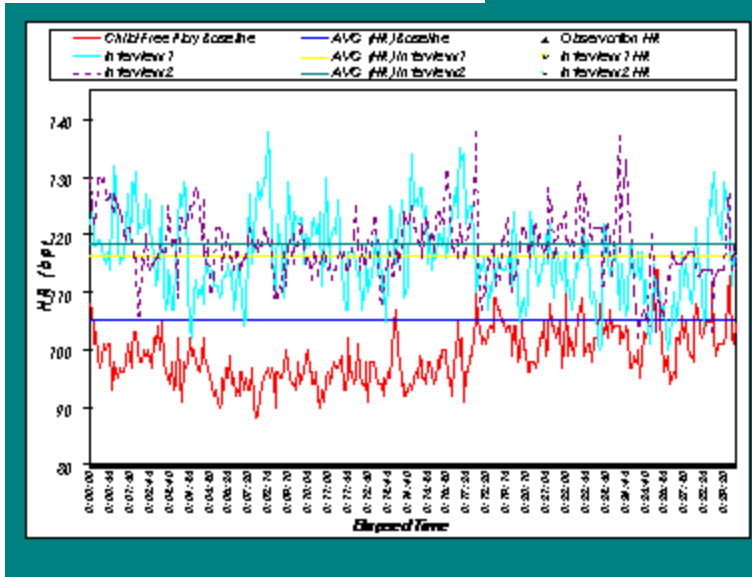
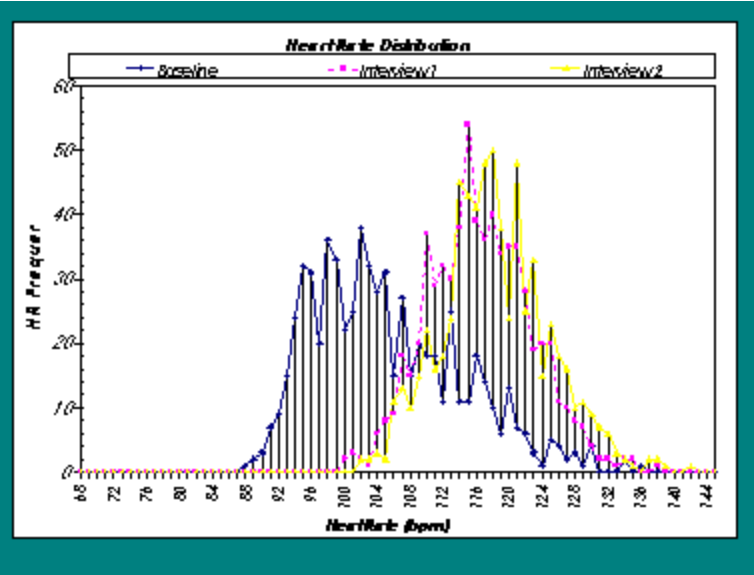
<b>Adaptive Response</b>	Rest <i>(Adult Male)</i>	Vigilance	Freeze	Flight	Fight
<b>Hyperarousal Continuum</b>	Rest <i>(Male Child)</i>	Vigilance	Resistance	Defiance	Aggression
<b>Dissociative Continuum</b>	Rest <i>(Female Child)</i>	Avoidance	Compliance	Dissociation	Fainting
<b>Mental State</b>	CALM	AROUSAL	ALARM	FEAR	TERROR

**Figure 3. State-dependent adaptations to threat.** Different children have different styles of adaptation to threat. Some children use a primary hyperarousal response some a primary dissociative response. Most use some combination of these two adaptive styles. In the fearful child, a defiant stance is often seen. This is typically interpreted as a willful and controlling child. Rather than understanding the behavior as related to fear, adults often respond to the 'oppositional' behavior by becoming more angry, more demanding. The child, over-reading the non-verbal cues of the frustrated and angry adult, feels more threatened and moves from alarm to fear to terror. These children may end up in a very primitive "mini-psychotic" regression or in a very combative state. The behavior of the child reflects their attempts to adapt and respond to a perceived (or misperceived) threat.

Sense of Time	Extended Future	Days Hours	Hours Minutes	Minutes Seconds	Loss of Sense of Time
<b>Primary secondary Brain Areas</b>	<b>NEOCORTEX</b> <i>Subcortex</i>	<b>SUBCORTEX</b> <i>Limbic</i>	<b>LIMBIC</b> <i>Midbrain</i>	<b>MIDBRAIN</b> <i>Brainstem</i>	<b>BRAINSTEM</b> <i>Autonomic</i>
<b>Cognition</b>	Abstract	Concrete	"Emotional"	Reactive	Reflexive
<b>Mental State</b>	CALM	AROUSAL	ALARM	FEAR	TERROR

**Figure 4. State-dependent cognition and the response to threat.** One of the most important elements of understanding the child living in the Vortex of Violence, is that all humans process, store, retrieve and respond to the world in a state-dependent fashion. When a child is in a persisting state of arousal due to persisting exposure to threat, the primary areas of the brain which are processing information are different from those in a child who can be calm. The calm child may sit in the same classroom next to the child in an alarm state, both hearing the same lecture by the teacher. Even if they have identical IQs, the child that is calm can focus on the words of the teacher and, using neocortex, engage in abstract cognition. The child in an alarm state will be less efficient at processing and storing the verbal information the teacher is providing. This child's cognition will be dominated by sub-cortical and limbic areas, focusing on non-verbal information -- the teachers facial expressions, hand gestures, when she seems distracted. And, because the brain internalizes (i.e., learns) in a 'use-dependent' fashion, this child will have more selective development of non-verbal cognitive capacities. The children raised in the vortex of violence have learned that non-verbal information is more important than verbal -- "when daddy smells like beer and walks funny, I know he will hurt mommy."

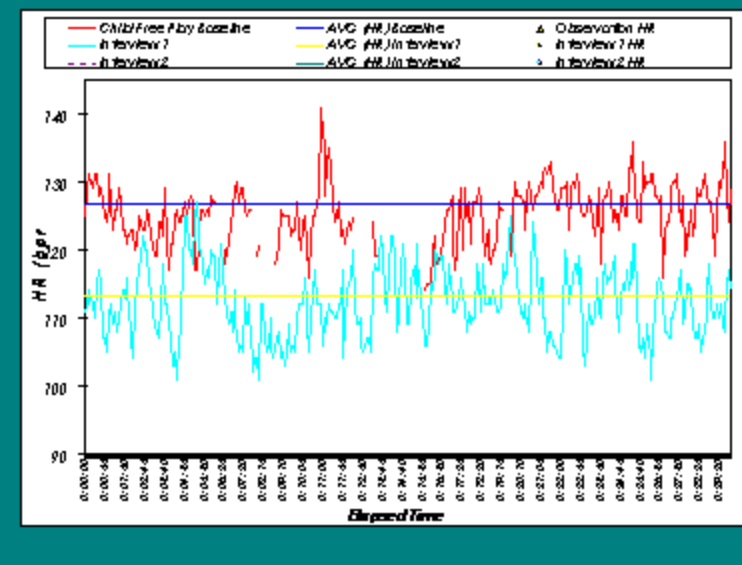
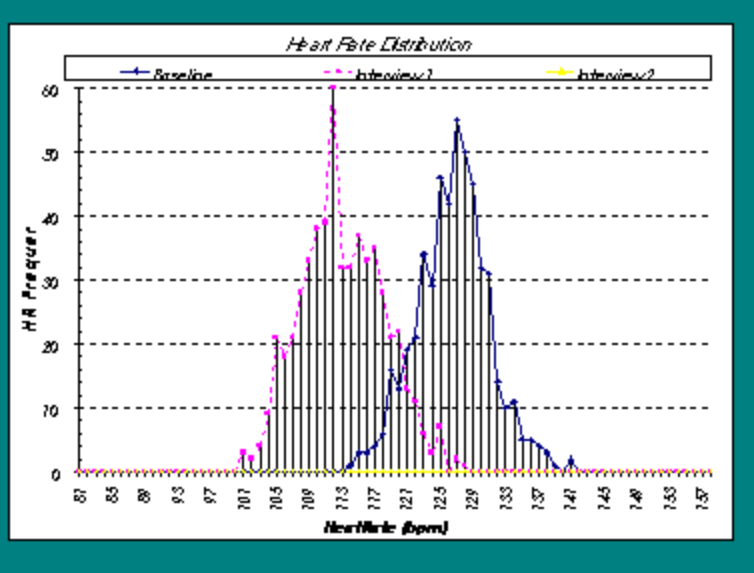
As a child moves along the continuum of arousal, the part of the brain which is 'orchestrating' functioning shifts. This process reflects ontogeny, such that the more distressed one is, the more primitive are the brain areas responsible. An important reflection of this is how the sense of time is altered in alarm states. Sense of future is foreshortened. The critical time period for the individual shrinks. The threatened child is not thinking (nor should she think) about months from now. This has profound implications for understanding the cognition of the traumatized child. Immediate reward is most reinforcing. Delayed gratification is impossible. Consequences of behavior become almost inconceivable to the threatened child. Reflection on behavior -- including violent behavior -- is impossible for the child in an alarm state. Cut adrift from internal regulating capabilities of the cortex, the brainstem acts reflexively, impulsively, aggressively -- to any perceived threat. Eye-contact for too long becomes a life-threatening signal. Wearing the wrong colors.-- a hand gesture -- cues that to the calm adult reading about another 'senseless' murder in the paper are insignificant but to the hypervigilant, armed adolescent born and raised in the vortex of violence, are enough to trigger a 'kill or be killed' response.



**Figure 5. Physiological state memory in a child with PTSD.**

(Left) Continuous tracing of a free play session, (with the same adult therapist as during the structured interview: mean HR = 105) and two separate structured PTSD interviews (mean HR = 119) with a young male (age = 9). This boy met DSM IV criterion for PTSD and exhibited prominent symptoms of physiological hyper

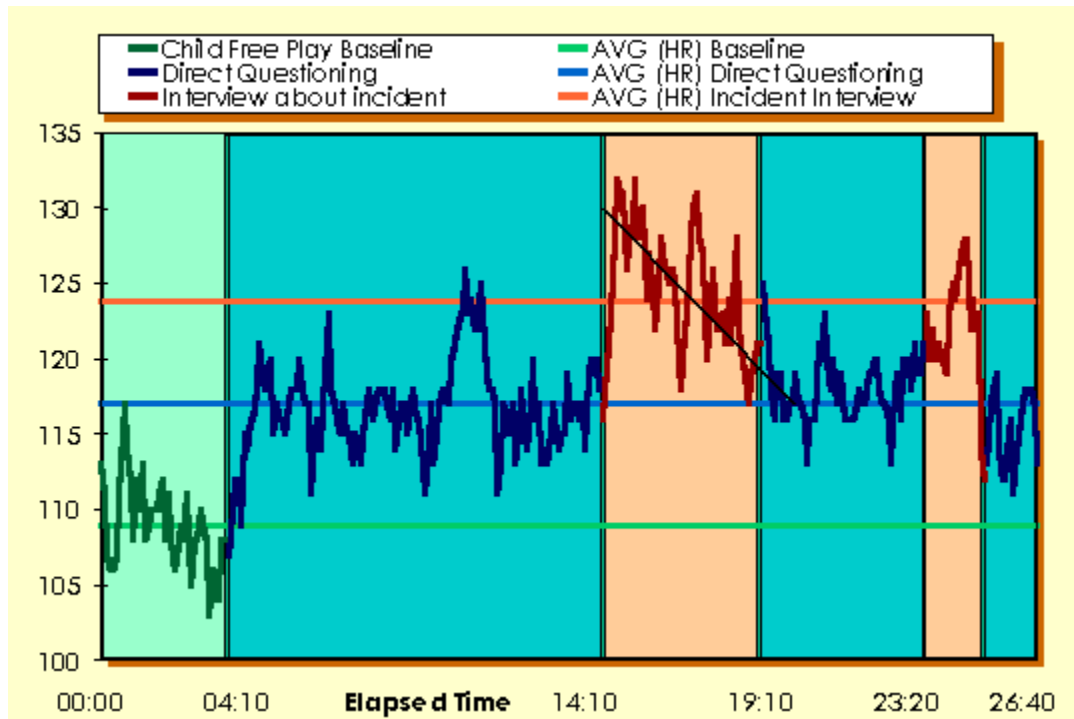
reactivity (e.g., motor hyperactivity, anxiety, impulsivity, hypervigilance, sleep disturbance). ( Right) HR distribution under free play and interview conditions. Note 1) the reproducibility of the increased baseline heart rate during interviews and the separation of the free play (baseline) distribution for the interviews.



**Figure 6. Physiological state memory in a child with primary dissociative symptoms.** (Left)

Continuous tracing of a similar free play session as described above. This tracing is from a young girl (age = 8) who met criterion for PTSD. She exhibited primarily dissociative and avoidant symptoms. During the structured interview, she exhibited profound symptoms of dissociation, gave 'invalid' and inaccurate responses to the

questions and had a marked decrease in her HR (mean = 112 compared to the free play mean of 128). (Right) HR distribution demonstrating almost complete separation of the two conditions—both with the same therapist/interviewer during the same session.



**Figure 7. State memory of a witnessed murder in a three year old child.** A three year old child witnessed the murder of his 18 month old sister. During a therapeutic session, a free play 'baseline' heart rate was established. When direct, but non-threatening, questioning began, his heart rate increased. Within 5 seconds of being asked about his sister, a dramatic increase in heart rate was seen with an immediate behavior inhibition and dissociative response. His heart rate fell and non-intrusive questioning resumed. When the issue was briefly mentioned again, an initial increase in heart rate, change in behavior and dissociation-related decrease in heart rate was observed.