Life is dynamic. From birth, even prior to birth, we are bathed in a continuous stream of experience. Through this flow of time, our complex physiology maintains a balancing act, ever changing to maintain some stability, equilibrium, and homeostasis. To survive and flourish, we must sense, process, store, and respond to elements of our dynamic environment. The brain is the primary organ responsible for these tasks. By internalizing and storing elements of the unique sequence and collection of our individual experiences, the brain forces us to become reflections of our personal histories. These histories may be filled with consistent, predictable, nurturing, and enriching experiences or marred by chaotic, threatening, and traumatic experiences. The nature, pattern, and timing of these experiences influence our subsequent functioning. It is from this catalog of life-events that our brain shapes our perceptions and reactions as we move, feel, and think, laugh, love and cry; and remember, create, or hate.

This article discusses the impact of traumatic experiences on child development and function as viewed through the lens of a neurodevelopmental perspective. This focus may provide some insight for those seeking to understand the neuropsychiatric problems resulting from childhood trauma. The recurring theme in a neurodevelopmental view is the remarkable malleability of the developing brain. The brain's exquisite sensitivity to experience in early child-

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

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hoid allows traumatic experiences during infancy and childhood to impact all future emotional, behavioral, cognitive, social, and physiologic functioning.

BACKGROUND: TRAUMA AND CHILDREN

It is the rate the child who escapes childhood without some cruelty, threat, pain, or loss. Far too many children experience severe chronic or traumatic stress. Millions of children each year experience traumatic events: natural disasters, physical assault, sexual assault, and a ton of other traumatic experiences. Millions of other children live in the traumatizing maelstrom of domestic or community violence. These experiences wound and scar the vulnerable, developing child, often resulting in impairments severe and chronic enough to be labeled neuropsychiatric disorders (e.g., reactive attachment disorder, posttraumatic stress disorder). Conservative estimates suggest that more than 8 million children, at any given time, are suffering from a trauma-related neuropsychiatric disorder, and that millions more suffer subclinical but nonetheless problematic. The cost to individuals and society by any measure, economic or human, is high.

NEUROPSYCHIATRIC DISORDERS AND CHILDHOOD TRAUMA

The best-characterized neuropsychiatric problems following childhood trauma are posttraumatic stress disorders.87-94 Terr’s landmark studies87 started the modern era of interest in the psychologic sequelae of childhood trauma. Over the last 20 years, lagging behind a similar rediscovery of adult PTSD, various important aspects of the phenomenology of childhood PTSD have been studied.87,88,89,90-92,95,96 Terr has described two broad categories of PTSD in children: the effects of discrete, encapsulated traumatic events (Type I) versus chronic, pervasive trauma (Type II).87 This distinction is a good start, but better phenomenology is necessary neuropsychiatric syndromes related to childhood trauma29-30,60,61,64,65,66,67.

Children respond neurologically and physiologically in a host of different ways and in ways different from adults. Independent of the direct effects of the trauma, the capacity of traumatic experiences to disrupt and interfere with emotional, behavioral, cognitive, social, and physical development leads to important secondary and tertiary effects on the child. The current Diagnostic Statistical Manual (DSM) has not captured diversity of adaptive and maladaptive syndromes that appear to be related to early life traumatic experiences. Among the comorbid neuropsychiatric diagnoses associated with childhood trauma are major depression, posttraumatic stress disorder (PTSD), adjustment disorder, anxiety disorder, attention deficit hyperactivity disorder (ADHD), dissociative disorder, and, following severe early life neglect and trauma, developmental disorders. Although, this is not the only nor an inevitable outcome of trauma during childhood, posttraumatic hyperarousal or dissociative-like symptoms often coexist with other disorders.29-30,43-47,60,61 Furthermore, severe early trauma appears to be an expresser of underlying constitutional or genetic vulnerability and may be a primary causal factor in the development of disorders later in life.15,48

Initial progress in the area of childhood PTSD was made by comparison with findings or conceptual views derived from adult populations, especially male combat veterans.60-64 These comparisons, once useful, are now very limi-
system reorganizes its basal patterns. An event is traumatic if it overpowers the organism dramatically and negatively disrupts homeostasis. In a very real sense, trauma throws the organism off balance and creates a persisting set of compensatory responses that, in turn, create a new but less flexible state of equilibrium. Trauma-induced homeostasis consumes more energy and is maladaptive compared with "normal" homeostasis. By inducing this expensive homeostasis and compromising full functional capacity, trauma robs the organism. It has survived the traumatic experience, but at a cost.

Homeostasis and Memory

In this regard, the human organism is no different from others. Although exquisitely complex, the core framework of the human brain is designed to sense and respond to the changing environment to promote survival. At the heart of our survival neurobiology is the capacity to make and store internal representations of the external world—memory. The brain creates memories owing to the capacity of neurons and neural systems to change from one homeostatic state to another. In response to a set of stimuli-induced alterations in activity (e.g., sensations), neurons undergo molecular changes that reflect this activity.6, 7 In a very real sense, unless the homeostatic dynamic of a neural system is altered by use, it will not change and will not make internal representations of the experience—it will not make memories. Neurons and neural systems change in a use-dependent fashion. Therefore, when neural systems are influenced by new or extreme patterns corresponding to new or extreme environmental situations, they will change their molecular neurophysiology, creating memories.

This process has important implications for understanding how we create memories of traumatic experiences. For adults, most experiences have only a small component that is new or unique. Typically, the majority of places, faces, words, sounds, smells, and tastes in any given moment are familiar; the brain has sensed, processed, and stored these patterns before. In these situations only some portions of the brain are activated and process outside of their homeostatic range. In the classroom, for example, a lecture may result in cortical activation but will cause little new emotional, motor, or arousal activity. The result, hopefully, is new cognitive memories that store the information from the lecture. Similarly, practicing piano may result in new cerebellar-basal ganglia-motor cortex activity and create motor memories, but it will have little effect on emotional or state-regulation systems.

Trauma, however, induces a total brain response. All parts of the brain will be involved in trying to survive the threat. A traumatic event, by altering activity (and altering the homeostasis) in all parts of the brain—the cortex, limbic, midbrain, and brainstem portions—can create different types of memory. Altering cortical homeostatic states creates cognitive memory; the limbic emotional memory; the midbrain motor memory; and the brainstem, physiologic state memories.6-8 These memories, reflections of the altered equilibrium resulting from a traumatic event, are the heart of trauma-related neurophysiologic signs and symptoms.

Children are more vulnerable to trauma than adults. Traumatic events modify an adult's original state of organization or homeostasis but may be the original organizing experience for the child, thereby determining the foundational organization and homeostasis of key neural systems. Experience in adults alters the organized brain, but in infants and children it organizes the developing brain.9 This difference has profound implications for understanding the differences between trauma in children and adults.

The Dynamic Environments of Development

The neurobiologic capabilities to sense, store, and respond to our environment evolved over millions of years as our pre-hominid and hominid ancestors adapted to the changing demands of their environments. These environments had many components: climate, weather, habitat, predators, and prey, and, crucial to understanding human societies, social structures. Hominus lived for thousands of years in small hunter-forager bands. Our complex social and communication capabilities allowed small, naked, slow, and weak individual humans to survive by creating larger, stronger, and more flexible biologic systems in the harsh natural world inhabited by larger, stronger, and naturally armed animals.5

Central elements of our human development—then and now—are the dynamic changes in the major environmental challenges associated with the life cycle. The world to which the brain must adapt is dynamic and involves shifts in the major sensory stimuli occurring at key transitional life stages. Our stream of experience is comprised of shifting environments: new demands, expectations, tasks, and capabilities.

It is important to emphasize environments. During the life cycle, the primary internal and external environments are changing. In utero, the sensory, the memory, and the homeostatic systems develop in the environment—without the presence of any stimuli. The major sensory cues (i.e., environment) are the mother's movements, rhythmic rocking, and soft humming that are familiar and soothing to the newborn. But most sensory and perceptual inputs are new and challenge the rudimentary homeostatic patterns created during intrauterine life. In contrast, the newborn's environment expands, enriches, and becomes more complex. More sights, sounds, tastes, and touch push the developing neural systems of the infant, child, and adolescent out from a previous set of homeostatic states to new and more functionally well-organized equilibria. This development can proceed in an optimal fashion when the presentant of new stimuli is safe, nurturing, predictable, repetitive, gradual, and attuned to the infant's or child's developmental stage. When new stimuli are extreme, surprising, or mismatched to developmental stage, development is disrupted.

What may be a dramatic, rapid, or unpredictable threat in an environment for the newborn (e.g., a diaper change, which does induce a stress response) may be a familiar, comforting pattern for the 1-year-old and a distressing, humiliating experience for the incontinent 6-year-old or 60-year-old. It is not surprising then, that the neurobiologic systems and solutions to responding to stress change with the unfolding demands and tasks of various life stages.6, 7 These memories, reflections of the altered equilibrium resulting from traumatic events, are the heart of trauma-related neurophysiologic signs and symptoms.

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functions, stress response neurobiology and functioning are experience- or use-dependent. The individual stress-response style and capacities of any child, then, are related to the process of cataloging experience during development.

Cataloging Experience

Throughout life, the brain is sensing, processing, and storing patterns of neural activation (i.e., making memories) that correspond to various sights, sounds, smells, tastes, textures, and other sensations. All touch-related sensory and limbic areas, which become established as neural, emotional, perceptual, or motoric, the brain stores these patterns, making associations among multiple sensory stimuli and cataloging sequences of experience against which all future experience is matched.19

In this regard, the brain is a conservative organ. It does not like to be surprised. All unidentified sensory and limbic cues are judged to be threatening until proven otherwise. Novel stimuli focus attention, increase arousal, and induce an alarm response until they can be proven neutral or safe. New patients and cues that do not match the stored memories of previous experience prime the stress-response systems in the brain.23,24,25,26 Once categorized as neutral, safe, or go, neural networks are added to the catalog of patterns, cues, and associations against which subsequent environmental cues are matched.

What is safe and comfortable becomes so through experience. In contrast, when the environment, internal or external, matches stored neuronal patterns associated with a previous threatening experience, the brain's stress-response systems are activated. Key signs and symptoms of trauma-related neuropsychiatric disorders result from these memories of fear. Later events can recall stimulus elements of the traumatic experience by triggering complex, multisystem responses (i.e., cognitive, emotional, motor, state) that were associated with original trauma, but now have generalized to similar cues. Memories of fear are created at multiple levels in the brain's hierarchical systems.

Processing Environmental Cues of Threat

Sensory information from the external environment (visual, auditory, tactile, olfactory, gustatory) and the internal environment (e.g., blood glucose, arterial pressure, CO2 levels) enters the central nervous system at the level of the brain stem and midbrain.27 As this primary sensory input comes into the brain stem and midbrain, it is matched against previously stored patterns of activation; if the input is unknown, or it is associated with previous threat, an initial alarm response begins.4 The alarm response initiates a wave of neural 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 adrenocorticotropic (ACTH), corticotropin-releasing factor, and vasopressin.4,29 Activation of these key systems results in patterns of neural activity that spread from the brainstem through the midbrain, to thalamus, and then to prefrontal cortex. At all levels of the brainstem and midbrain, there is little subjective perception. It is at these levels of the thalamus and limbic areas that specific patterns of neural activity result in emotional sensations associated with threat—fear, anxiety, and anger.8,40 At the subcortical and cortical level, more complex cognitive associations are made, allowing interpretation of the experience. The events can be categorized, contextualized, and understood within a larger perceptual or cognitive framework.29 Sensing and perceiving threat must be paired with response to threat if the organism is to survive. At each level of the central nervous system, just as the alll
cerr input is interpreted and matched against previous similar patterns of activation an effector arm is initiated. Each level and area of the brain has some role in the effector response to threat. The brainstem regulates the autonomic and hypothalamic output, alters arousal, and tunes out distracting sensory information; the midbrain regulates elements of motor activity (e.g., startle response); the limbic system modulates emotional reaction and signalling (e.g., facial expression); and the cortex interprets the threat and develops a complex plan. Under ideal circumstances, these multiple removers, using various modes of memory (e.g., auditory, cognitive, emotional, motoric) the brain, stores these patterns, making associations among multiple sensory stimuli and cataloging sequences of experience against which all future experience is matched.9

The specific response patterns for any individual or situation depend on many factors, including the nature, duration, severity, and history of exposure to similar threat. Age, of course, is a primary factor; in different age groups, stimuli will be perceived as threats by one individual and will not respond to children, the higher, more complex parts of the central nervous system will have yet to be organized or fully functioned. The infant can still have a fear-evoked startle, emotional distress, and age-appropriate reactivity in response to a traumatic experience but be unable to make a plan. Nor can the prefrontal threatening experiences be used to describe his or her behavior. Adults often misunderstand the silence of maltreated or traumatized children. As children sometimes so badly that they cannot respond to trauma. It is a very common adult misperception that children are better at coping with change or stress than adults. Despite the apparent ease with which many young children survive trauma, they are much more vulnerable to trauma than adults. Indeed, it is increasingly clear that the sensitivity and organization of stress-response neurochemical systems are related to developmental experiences with stress.

Development of Stress Response Neurobiology

At birth, despite having all of its 100 billion neurons in place, the human brain is not completely or organized. Brain-mediated functions depend on the process of synaptogenesis and synaptic linking by specialized connections called synapses. During the first months of life the number of synaptic connections rise dramatically. In months of age, the synaptic number is much higher than they ever will be.7,20 The development and organization of functionally important neuronal networks (systems) is use-dependent. Activation of these connections that are used are maintained and strengthened, whereas those that are not are pruned out and lost. In a very concrete sense, the experiences of early childhood create patterns of neuronal activity that are fundamental to the template neural networks and patterns (biomechanisms) against which all future experience will be sensed, processed, and internalized.

The brain is comprised of many different systems and areas, each mediating some components of brain function. Not all of these changes can be observed at the same time. At birth, simple regulatory functions (e.g., respiration, temperature-
distinct response patterns during any given traumatic event. The predominant response patterns appear to shift from dissociative to hyperarousal during development. Although incompletely characterized in children, these two major response patterns illustrate key principles of the neurodevelopmental perspective on trauma.

Hyperarousal: Fight or Flight Responses

The initial phase of the hyperarousal continuum is an storm reaction that begins to activate the sympathetic nervous system. This alarm reaction is mediated by the locus coeruleus (LC). The LC is a bilateral nucleus of noradrenergic-containing neurons located on the floor of the pons. It sends diverse axonal projections to virtually all major brain regions, enabling it to function as the primary regulator of norepinephrine tone and activity. The LC is important in the process of attending to novel stimuli and may orchestrate "importance" of simultaneous sensory information, and modulating attention and arousal states. In this context, role of one mediator of the stress response is the LC, a key mediator of the stress response. Other important brainstem nuclei, including the ventral tegmental nucleus (VTN), play a part in regulating the sympathetic nervous system.

The activity in the LC mirror the degree of arousal (i.e., sleep, calm-alert, stress-vigilant, fight-flight) related to stress or distress in the environment. Fear increases LC and VTN activity, increasing the release of norepinephrine in all of the LC and VTN terminal fields throughout the brain. Fear refers to the potential for physical harm, whereas alarm refers to the potential for psychological harm.

The LC tunes out noncritical information and mediates hypervigilance. This nucleus regulates the complex interactive process, which includes activation of centrally controlled autonomic nervous system, and the hypothalamic-pituitary-adrenal (HPA) axis with resulting release in adreno-corticotropin and cortisol. The sympathetic nervous system is further activated, increasing heart rate, blood pressure, and respiratory rate, mobilizing glucose, and increasing muscle tone. All of these actions prepare the body for defense—fight or flight or run away from the potential threat. As a full right-flight response is activated. This response was first described by Cannon and has been the most studied, best-characterized stress-response pattern in humans.

If a child faced with threat responds with hyperarousal, there will be a dramatic increase in LC and VTN activity. The hypothalamic-pituitary-adrenal (HPA) axis is crucial to orchestration of the peripheral response to stress. Corticotropin-releasing hormone (CRH) selectively stimulates and regulates ACTH secretion in rats. ACTH, once released, in turn stimulates adrenal secretion of glucocorticoids or cortisol, which causes a myriad of peripheral adaptive responses, including glucose mobilization, altered cellular metabolism.

As with central neurobiologic systems, stress, distress, and trauma alter HPA regulation (i.e., a new homeostasis has been induced by the stress). Allostatic maladaptions of the HPA axis have been noted in adults with PTSD. Chronic
activation of the HPA system in response to stress has negative consequences. The homeostatic state associated with hyperarousal wears the body out.18,19 Hippocampal damage, impaired glucose utilization, and vulnerability to metabolic insults10 may result. Preliminary studies in a sample of abused children suggest similar hippocampal and limbic abnormalities.20

Following an acute fear response, the brain creates a set of memories from the event. These memories are reactivated when the child is exposed to a specific reminder of the traumatic event (e.g., gunshots, the perpetrator). Furthermore, these memories can be reactivated when the child simply thinks about or talks about the event. Unfortunately, one of the amazing strengths of the human brain, its capacity to make associations from the specific to the general, begins to betray the traumatized child. Traumatic events generalize (e.g., gunshots to loud noises, a specific perpetrator to any strange male). In other words, despite being away from threat and the original scene, these key parts of the child’s brain are activated again and again. The memories of fear are seared into the child’s neurobiology. 

The use-dependent activation of the brain leads to sensitization. Sensitization of catecholamine (LC/VTN-amphidalar) systems leads to a cascade of associated changes in brain-related functions.21-26 Sensitization of the brain stem and midbrain neurotransmitter systems mediated by the hyperarousal response also means that the other critical physiologic, cognitive, emotional, and behavioral brain systems will become sensitized. Because the LC/VTN and its target regions (amphidalar nuclei) also mediate a variety of other functions, sensitization of these systems by repetitive reexposure of the trauma leads to dysregulation of these functions. A traumatized child may, therefore, exhibit motor hyperactivity, anxiety, behavioral impulsivity, sleep problems, and hyperactivity.27-30

This means, of course, that the stress response itself becomes sensitized. Everyday stresses that previously may not have elicited any response are now able to elicit an exaggerated reactivity in children who are hyperreactive and overly sensitive. Simply stated, the child is in a persistent fear state. Furthermore, this means that the child’s new base homeostatic or "standard" emotional state is a state of anxiety. This child will be more easily threatened or terrorized. Over time, these children exhibit a set of maladaptive emotional, behavioral, cognitive, and physiological responses noted in the original adaptive response to a traumatic event.

The few research studies examining catecholamine systems in children following trauma suggest a dysregulated, sensitized stress-response neurobiology. In a pilot study, sexually abused girls exhibited greater total catecholamine synthesis as measured by plasma excretion of epinephrine, norepinephrine, and dopamine when compared with matched controls.31-32 In a group of 60 children with PTSD, altered cardiovascular regulation (e.g., increased resting heart rate) has been demonstrated. These alterations have been associated with an increased sympathoadrenal response and vasodilation.33-38 In other studies, clonidine, an α

The Dissociative Continuum

Infants, of course, are not capable of fight or flight. Their threat response patterns are unique and, in the initial stages of distress, are characterized by a precursor form of a hyperarousal response. In these pre-alarm and alarm stages, the infant will use his or her limited behavioral repertoire to attract the attention of a caregiver. These behaviors include changes in facial expression, body movements, and, most important, vocalization (i.e., crying). This is a successful adaptive strategy if the caretaker comes to feed, warm, soothe, fight for, or flee with the infant.

Unfortunately, for many infants and children these strategies are not effective. Indeed, millions of children, if they are fussy, difficult, or weepy, are maltreated by the very adults who should be protecting them. In the absence of an appropriate caregiver reaction to the initial alarm outcry, the child will abandon this behavior. Furthermore, if the infant or child has few if any positive responses and negative responses, he or she will abandon this set of adaptations. The converse of use-dependent development occurs, disuse-related behavior extinction. This defeat response is well-characterized in animal models of stress reaction and learned helplessness. This defeat reaction in a common element of the presenting emotional and behavioral phenomenology of many neglected and abused children.39-44 Indeed, adults, professional or not, often puzzle over the emotional nonreactivity, passivity, and compliance of many abused children. All too often this defeat response is mistaken for resilience: "Can you believe how easy it is for her to talk about all those horrible things they did to her, and she is so easy to have around, so easy-going? What a tough little girl! But I guess kids are just resilient, right?" Wrong. Children are maladaptive. Children become resilient if they incorporate a stress-response neurobiology that mirrors their experiences of predictable and nurturing caregiving. In the face of threat, the infant or young child will be forced to activate other responses to adapt. If the child is old enough, this may involve moving further along the hyperarousal continuum (the child’s version of fight or flight) for infants, however, this will involve activation of dissociative adaptations. Dissociation is a broad descriptive term that includes a variety of mental mechanisms involved in disengaging from the external world and attending to stimuli in the internal world. It can involve distraction, avoidance, numbing, daydreaming, fantasy, derealization, depersonalization, and, in extreme, freezing or catalepsy. In our experiences with young children and infants, the predominant adaptive responses during the trauma are consistent with dissociative mechanisms. Children report going to a "different place," assuming persona of heroes or animals, a sense of "watching a movie that I was in" or "just floating"—classic depersonalization and denial responses. Observers report these children as numb, robotic, nonreactive, daydreaming, "acting like he was not there," and starting off in a glazed look. The younger the child the more likely they will be primary dissociative adaptations. Immobilization, unavailability, or pain will increase the dissociative components of the stress response patterns at any age.

In animals, the defeat response is mediated by different neurobiologic mechanisms than the fight or flight response. What little is known about the neurobiology and phenomenology of dissociation appears to most approximate the defeat reaction described in animals.45,46 As with the hyperarousal response, there is brainstem-mediated CNS activation that results in increases in circulating epinephrine and associated stress steroids.47,48 A major difference in the CNS, however, is that vagal tone increases dramatically, decreasing blood pressure and heart rate (occasionally resulting in fainting) despite increases in circulating epinephrine.

Dopaminergic systems, primarily mesolimbic and mesocortical, play an important role in defeat response mechanisms in animals.49,50,51 These dopamine...
typically, modulation affects you opioid enous of sense. The to regulating and freezing, a human's response, including post-traumatic stress disorder, can be exacerbated by stress. The child's ability to cope with trauma can be hindered by exposure to multiple stressful events, which can lead to dysregulation and freezing responses. Children who have experienced trauma may exhibit freezing behaviors as a defense mechanism, often as a response to sensory stimuli or physical threats. The freezing response is characterized by a rigid, immobile posture, cessation of voluntary movement, and a withdrawal from the environment. This response is thought to be an adaptive mechanism for survival, allowing the child to remain still and avoid potential harm. However, chronic freezing can have negative effects on a child's development and social interactions. It is important to recognize and address freezing behaviors in children who have experienced trauma to facilitate their emotional regulation and social engagement.
and noncompliance. Typically these inattentive boys are diagnosed with ADHD. Young girls who have been similarly traumatized are not brought to the clinician by the parents (thus, perhaps, the 3:1 male-to-female ratio). The maltreated, dissociating girl daydreaming in the classroom is less bothersome to caregivers and teachers than the hyperactive, impulsive, and noncompliant boy. Girls are maltreated as much, if not more, than boys. Girls' brains process trauma with the same principles of neurophysiology as boys. Girls are damaged by traumas as much as boys, yet they are much less likely to get our help.

SUMMARY AND FUTURE DIRECTIONS: THERAPEUTIC AND POLICY IMPLICATIONS

All experiences change the brain, but not all experiences have an equal impact on the brain. Because the brain is developing and organizing at such an explosive rate in the first years of life, experiences during this period have the most potential to influence the brain in positive and negative ways. Traumatic experiences and therapeutic experiences affect the same brain and are limited by the same principles of neurophysiology. Traumatic events disrupt homeostasis in the multiple areas of the brain that are recruited to respond to the threat. Use-dependent internalization of the developments of the experience creates neurological influence future functioning. To heal (i.e., alter or modify memories of trauma), therapeutic interventions must activate key structural changes of the brain that have been altered by the trauma. Understanding the persistence of fear-related emotional, behavioral, cognitive, and physiologic patterns can lead to focused therapeutic experiences that modify these parts of the brain affected by trauma.

A neurodevelopmental view of childhood traumas provides novel directions for assessment, intervention, and policy. Primary among these is the clear neurobiologic rationale for early identification and aggressive, proactive interventions that will improve our ability to protect, heal, educate, and enrich traumatized and neglected children. Future clinical and research efforts in these areas must begin to define and use child-specific and developmentally informed models to guide assessment, intervention, education, therapeutic, and policy.

References

6. Adler A: Neuropsychiatric complications in victims of Boston's Coconut Grove disas-
ter. JAMA 123:1098–1103, 1943
300
8. American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disor-
15. Bloomer JM, Hornbuckle R, Johnson D, et al: Childhood physical abuse and com-
239, 1993
endochrom and behavioral activating effects following infusion into the locus cer-
22. Carlson V, Cicchetti D, Bamet D, et al: Disorganized/disoriented attachment relation-
23. Castro-Alamand M, Conners BW: Short-term plasmid of a thalamocortical path-
ADAPTATION STRESS, and STRESS, of Schwartz & Kandel. 53.


Clayton GB: Stress and brain miniaturization: A review. Neuropsycho Behav Rev 22:3...


78. Perry BD: Neurodevelopmental aspects of childhood anxiety disorders: Neurobiological

79. Perry BD: Placeental and blood element neurotransmitter receptor regulation in hu-
man Brain: Practical note for studying neurochemical mechanisms underlying behav-
ioral teratology. Prog Brain Res 73:189-206, 1988


91. Sapolsky RM, Uno H, Robert CS, et al: Hippocampal damage associated with pro-

92. Scheftins MS, Zanakah CH, Dridi MI, et al: Two approaches to the diagnosis of post-


103. Terz LC: Chowderilla revisited: The effects of psychotherapy four years after a school


106. Vanenti C, Perry BD, Giurini AD, et al: Brain ependyoma systems: Detailed comparis-