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MEDICINE AND PSYCHOTHERAPY

Neurodevelopment and the Neurophysiology of Trauma I: Conceptual Considerations for Clinical Work with Maltreated Children

—by Bruce D. Perry

Editor's Note: This is the first of two articles about emerging medical research into the neurological effects of trauma. The aim of the series is to inform practitioners from all disciplines about this new body of research and about the possible behavioral effects of neurophysiological changes. This article presents what is known about the neurological effects of trauma. The second article, addressing clinical implications of a neurodevelopmental conceptualization of childhood trauma, will appear in the next issue of The Advisor *(V.6, n.2).*

Introduction

A terrified three-year-old child huddles, sobbing, in a dark corner of his room after being beaten by a drunk parent for spilling milk; a colicky infant cries for eight hours, left alone, soiled and hungry, by an immature, impaired mother; a four-year-old boy watches his father beat his mother—only the most recent of many terrorizing assaults this child has witnessed in his chaotic, violent household.

Many studies, wise parents and experienced clinicians tell us that these experiences will influence dramatically how these children grow up—but how? How do experiences change development? What is going on in these children's heads, literally; what are they sensing, perceiving, thinking and feeling? What are the neurobiological correlates of the perceptions, thoughts, feelings and actions of a child's response to a traumatic event? How does the neurochemical milieu associated with fear influence the developing brain? How does repeated exposure to a traumatizing experience alter development? What are the mechanisms by which experience, any experience, influences development?

The impact of traumatic life experiences on children and formulations regarding how traumatic experiences affect development have been discussed from many perspectives, primarily using descriptive, clinical, or psychological formulations (e.g., Terr, 1983; 1991; Finkelhor, 1984; ; Conte, 1985; Eth and Pynoos, 1985; Browne and Finkelhor, 1986; Alter-Reid et al., 1985; McLeer, 1988). In contrast, the purpose of this paper is to discuss the impact of traumatic life experiences on the development of the brain and, specifically, on those portions of the brain involved in mediating the stress response. Knowledge of the core neurobiology of the stress response can lead to important insights regarding the etiology and treatment of the adverse physiological, emotional, behavioral and cognitive sequelae of childhood trauma.

Traumatized children: The scope of the problem

The significance of understanding the neurodevelopmental effects of traumatic stress cannot be overstated. Each year in the United States at least four million children are traumatized by physical abuse, sexual abuse, domestic violence, community violence, natural disasters or man-made disasters. The potential devastation from traumatic stress can be illustrated by examining the effects of combat on adult populations.

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NEWS

New Board Elected; By-Laws Amended; Annual Meeting a Success

—Teresa Reid

APSAC's membership elected 10 outstanding new Board members in the Fall, 1992, election. New Board members are:

Catherine Ayoub, RN, EdD	Dana Gassaway
Jan Bays, MD	Eliana Gil, PhD
John Briere, PhD	Carolyn Levitt, MD
Jon Conte, PhD	Patricia Toth, JD
David Corwin, MD	Bill Walsh

Two other Board members were appointed by the Board during the year to replace elected members who resigned. When Antonia Dobrec, MSW, resigned, the Board invited Holly Echo Hawk Middleton, MS, Regional Vice President of the Children's Home Society in Vancouver, Washington, to join the Board for the remainder of Toni's term (through 1994). Veronica Abney, MSW, agreed to serve the remainder of the term of Richard Krugman, MD, who resigned near the end of 1992. Veronica, the coordinator of the Suspected Child Abuse and Neglect Team at UCLA's Neuropsychiatric Hospital, will serve on the Board through 1993. Also appointed at the end of 1992 were five new members of APSAC's Advisory Board. They are Ann Burgess, DNSc; Gail Goodman, PhD; Ken Lanning, MS; John E.B. Myers, JD; and Joyce Thomas, RN, MPH.

The Board elected its officers at the annual meeting. Those elected were:

- Patricia Toth, JD, First Vice President (President Elect and Chair of the Membership Committee)
- Linda Meyer Williams, PhD, Second Vice President (Chair of the Program Committee)
- Paul Stern, JD, Treasurer (Chair of the Finance Committee)
- Kathleen Coulborn Faller, MSW, PhD, Secretary (Chair of the Nominating Committee).

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-Bruce D. Perry

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The neuropsychiatric sequelae of combat have been best characterized in veterans of the war in Vietnam. In the 12-year period of the Vietnam Era, three million Americans served in the Vietnam theater. Over the next twenty years, fully thirty percent of these young adults developed Post-Traumatic Stress Disorder (PTSD, DSM III-R) following combat-related traumatic experiences (Kulka et al., 1990). The debilitating symptoms of PTSD fall into three clusters; 1) recurring intrusive recollection of the traumatic event such as dreams, flashbacks and intrusive thoughts, 2) persistent avoidance of stimuli associated with the trauma or numbing of general responsiveness, and 3) persistent symptoms of increased arousal characterized by hypervigilance, increased startle response, sleep difficulties, irritability, anxiety, and physiological hyperreactivity, reflective of a hyper-reactive autonomic nervous system (see DaCosta, 1871; Bury, 1918; Dobbs and Wilson, 1960; Horowitz et al., 1980).

Knowledge of the core neurobiology of the stress response can lead to important insights regarding the etiology and treatment of the adverse physiological, emotional, behavioral and cognitive sequelae of childhood trauma.

How much more pervasive and disruptive traumatic stress may be on young children exposed to chronic violence or abuse during the most vulnerable years of their lives, during the time in which they are developing physically, cognitively, emotionally and socially. If a similar percentage of children exposed to the 'battles of childhood' develop stress-related neuropsychiatric problems, over one million American children each year will join the ranks of other childhood 'veterans' in need of special mental health, educational, and medical services. Furthermore, these childhood problems persist; the great majority of traumatized children carry their scars into adolescence and adulthood.

Despite the scope of this serious public health problem, relatively little research has been dedicated to neurodevelopmental trauma or childhood PTSD. In contrast, studies on the neurobiology of the stress response (e.g., Stone, 1975; Sapolsky et al., 1986; Murberg et al., 1990), 'sensitizing' pharmacological (Kleven et al., 1990; Farfel et al., 1992) or stress paradigms (e.g., Kalivas and Duffy, 1989; Post, 1992), and the basic neurochemical and neurophysiology of PTSD in adults (e.g., Perry, 1988; Krystal et al, 1989; Giller et al., 1990; Perry et al., 1990a) have led to important clinical formulations and interventions. Similar progress in understanding disorders related to childhood trauma will depend upon research advances in neurodevelopment and the impact of traumatic stress upon this process.

The development of a human being from a single cell is an amazingly complex miracle of biology. By adulthood, a single set of genetic material has been differentially expressed in a billion different ways—each resulting in a different cell with unique structural and chemical composition and, therefore, unique functional capabilities. The most complex of all organs, the human brain contains 100 billion neurons and 10 times as many glial cells, each of them unique. The neurons and glial cells of the human brain connect and organize into functional units with specific roles to sense, perceive, process and act on information from outside and inside the individual in a fashion that promotes, first and foremost, survival, and then other actions and transactions of being human.

Understanding the traumatized child requires recognition of a key principle of developmental neurobiology: *the brain develops and organizes as a reflection of developmental experience,* organizing in response to the pattern, intensity and nature of sensory and perceptual experience. The experience of the traumatized child is fear, threat, unpredictability, frustration, chaos, hunger, and pain. Therefore, the traumatized child's template for brain organization is the stress response.

The Neurobiology of Survival

When an infant, child or adult is threatened there is a set of critical, ingrained responses which the body uses to perceive, process and act to defend itself from the threat. In 1914 Walter B. Cannon first coined the phrase "fight or flight" reaction (Cannon, 1914). This well-characterized set of adaptive physiological responses to real or perceived danger involves a series of complex, interactive neurophysiological reactions in the brain, the autonomic nervous system, the hypothalamic-pituitary adrenocortical (HPA) axis, and the immune system (see Loewy and Spyer, 1990).

Some of the systems involved in the stress response are termed "adrenergic" or "noradrenergic." These terms refer to systems using adrenaline (also known as ephinephrine) and noradrenaline (also known as norephinephrine), as their chemical messengers. Most people are familiar with adrenaline as the hormone responsible for causing some of the well-known features of the stress response: dilation of the pupils, rapid heartbeat, high blood pressure, and sweating.

The neurophysiology of the alarm reaction has been studied extensively in human and animal models (see Selye, 1936; Stone, 1975; Stone, 1988; Murberg et al., 1990). Acute stress is associated with a variety of physiological responses including the activation of the HPA axis with a concomitant peripheral release of hormones, including ACTH, epinephrine (adrenaline) and cortisol; a significant increase in centrally-controlled peripheral sympathetic nervous system tone; and the activation of a variety of neurochemical systems in the central nervous systems (CNS). The major method of communication in this process is neurochemical transmission. A chemical neurotransmitter is released from one neuron and interacts with specific neurotransmitter receptors on other neurons, communicating some form of chemical message to these

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neurons and, thereby, altering their activity and functioning.

One of the most critical neurotransmitter systems involved in the stress response is the noradrenergic nucleus called the locus coeruleus (LC: Korf, 1976). This bilateral grouping of norepinephrine-containing neurons originates in the pons, a more primitive, regulatory part of the brain, and sends branching projections (axons) throughout brain, connecting directly or indirectly with virtually all major brain regions (Moore and Bloom, 1975; Fillenz, 1990). This diverse set of connections facilitates the orchestrating role of the LC, which acts as a general regulator and monitor of many important brain activities, controlling noradrenergic tone and activity throughout brainstem, midbrain, limbic and cortical areas (Foote et al., 1983). The LC plays a critical role in arousal, vigilance, regulation of affect, behavioral irritability, locomotion, attention, the response to stress, sleep regulation and the startle response (Korf, 1976; Redmond and Huang, 1979; Foote et al., 1983; Aston-Jones and Bloom, 1981; Svensson, 1987; Waterhouse et al., 1988; Fillenz, 1990). Another key adrenergic/noradrenergic system in the brain is the ventral tegmental nucleus (VTN) which is involved in regulation of the sympathetic nuclei in the pons/medulla (Moore and Bloom, 1975). Acute stress results in an increase in LC and VTN activity. This increases the release of norepinephrine from these neurons and influences various functions throughout the brain and the rest of the body.

Understanding the traumatized child requires recognition of a key principle of developmental neurobiology: the brain develops and organizes as a reflection of developmental experience, organizing in response to the pattern, intensity and nature of sensory and perceptual experience. . . . The traumatized child's template for brain organization is the stress response.

The neurophysiological activation seen during acute stress is usually rapid and reversible. When the stressful event is of a sufficient duration, intensity, or frequency, however, the brain is altered. Stress-induced sensitization may occur—the neurochemical systems mediating the stress response (e.g., LC noradrenergic systems) change, becoming more sensitive to future stressors related to the original experience. The molecular mechanisms underlying this phenomenon are not well understood but are related to the same cascade of molecular processes involved in learning and memory.

The stressful experience, via a cascade of neurochemical events, alters the microenvironmental milieu of the central nervous system (CNS), resulting in altered gene expression. The portion of the genome that is expressed in a given neuron is dependent upon the local microenvironment in the nucleus of the neuron. This microenvironment, in turn, is a direct reflection of a biochemical cascade which begins with experience. Experience activates the neurosensory apparatus and alters the pattern and quantity of neurotransmitter release throughout the neuronal networks responsible for sensation, perception, and processing of information. This change in neurotransmitter activity influences, initially, the extracellular milieu of the neurons in the system and then, by neurotransmitter receptor/effector activation, changes important intracellular chemical constituents (i.e., second and third messengers) in all of the neurons synaptically connected to these systems. It is the changes in these second (e.g., cAMP, phosphatidyl inositol) and third messengers which alter the microenvironmental milieu of the nucleus, resulting in changes in gene transcription. These new gene products may then result in permanent or structural changes which are associated with sensitization, learning, memory and, in the developing brain, differentiation (see Kandel and Schwartz, 1982; Goelet and Kandel, 1986).

Stressful experiences or certain drugs, then, when they result in a certain pattern of catecholamine (norepinephrine, dopamine, epinephrine) activity trigger a neurochemical cascade which results in altered expression of proteins (including receptors) involved in catecholamine responsivity (Kalivas and Duffy, 1989; Kleven et al., 1990; Farfel et al., 1992). This altered catecholamine responsivity (sensitization) likely underlies the hypervigilance, increased startle, affective lability, anxiety, dysphoria, and increased autonomic nervous system hyper-reactivity seen in adult PTSD (see Krystal et al., 1989; Perry et al., 1990a). In the adult, mature brain, increases in, or unusual patterns of, catecholamine activity may result in sensitization. In the developing brain, however, neurotransmitters, in addition to their roles in cellular communication, play an important role in the basic neurodevelopmental process (Lauder, 1988). Trauma related alterations in catecholamine activity during childhood, therefore, may alter brain development, resulting in altered functional capabilities of the traumatized brain (Perry, in press).

The Developing Brain

Brain development requires that a small number of cells with similar properties divide, migrate and differentiate to result in billions of cells with different physical and chemical properties. Each of these cells has the same genetic material (genotype) but they have different portions of this genetic material being actively utilized, resulting in the different expressed properties (phenotype) of each individual neuron. The expressed properties of a neuron—the size, shape, protein makeup, chemical constituents—confer the functional properties of the neuron. By differentially expressing portions of the genome, the remarkable structural and functional diversity of neuronal components of the human brain is possible.

Differentiation is the process by which cells become specialized, expressing those components of the genome which confer special properties associated with the functions of the neuron in the mature brain. This process takes place throughout development. While the majority of neurons have been born

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(neurogenesis) by birth (i.e., the final number of cells in the newborn brain is roughly the same as in the mature brain), the majority of individual cell growth and specialization has not taken place. Over the three years following birth, the important processes of neuronal migration, axo-dendritic projection, myelinization, synaptogenesis, and neurochemical differentiation continue to take place. As the brain develops, neurons divide, migrate, and differentiate in response to chemical, microenvironmental cues (morphogens) which confer information to, and direct specific differentiation of, the cell. Each neuron's unique structural, biochemical and functional character, then, is a function of its unique environmental history—the specific pattern, timing and quantity of these microenvironmental cues.

Some of the most important of these microenvironmental cues are receptor-mediated signals from neurotransmitters and hormones. Hormones, neurotransmitters and direct cell to cell contacts act as morphogens. The quantity, pattern of exposure and timing of morphogenic cues orchestrate and guide neuronal development. Indeed, catecholamine cues during development are important in determining critical functional properties of mature neurons, including the density of neurotransmitter receptors (e.g., Miller and Friedhoff, 1988; Perry et al., 1990b). Alterations in the pattern, timing, and quantity of catecholamine (or any critical neurotransmitter system) activity during development might be expected to result in altered development of catecholamine receptor/effector systems and the functions mediated, in part, by these systems.

Despite the scope of this serious public health problem, relatively little research has been dedicated to neurodevelopmental trauma or childhood PTSD. In contrast, studies on the neurobiology of the stress response, 'sensitizing' pharmacological or stress paradigms, and the basic neurochemical and neurophysiology of PTSD in adults have led to important clinical formulations and interventions.

A trauma-induced prolonged stress response will result in an abnormal pattern, timing and intensity of catecholamine activity in the developing brain. The time during development that this prolonged or abnormal catecholamine activity is present determines, to some degree, the nature and severity of the disrupted development. In general, the earlier and the more pervasive the trauma, the more neurodevelopment will be disrupted. The intrauterine environment is not necessarily protective. There is some evidence to suggest that prenatal or maternal traumatic stress has significant impact on neurodevelopment—battering the pregnant mother may also be battering the developing fetus (Amaro et al., 1980). The majority of child abuse or neglect takes place after birth, however. The development of the human brain continues beyond birth and its development remains vulnerable to the abnormal patterns of neurotransmitter and hormone activity associated with traumatic stress. Young children victimized by trauma are at risk for developing permanent vulnerabilities—changes in neuronal differentiation and organization—alterations in brain development which persist into adolescence and adulthood, with potential impact on all aspects of emotional, cognitive and behavioral functioning.

The relationships between the age of the traumatized child, vulnerability, and subsequent adverse sequelae are predicted by another key principle of neurodevelopment—critical and sensitive periods.

Critical and sensitive periods

As important in neurodevelopment as the pattern, quantity, and quality of the neurochemical signals which neurons receive is the timing of signals. There are times in development during which a set of signals must be present for the neurons to differentiate normally. These are called critical periods. In addition, there are times when an undifferentiated neuron is specially receptive or sensitive to a set of signals. At these times, termed sensitive periods, the neuron will use this set of signals to facilitate further specialization as part of a larger functional subsystem in the brain. As neurons develop, they organize into larger functional units, co-developing to specialize in a given set of brain functions. Neurons that transduce light, for example, connect with neurons that perceive light, neurons that localize the perception of light in space, neurons that process this information, others that allow responding to this information and so forth. During this process of co-development, the strength of the connections in this network is dependent, first, upon the presence of the signal (e.g., the light), and then upon the pattern and intensity of this signal (see Jacobson, 1991). The times in development during which these connections are being made and these patterns of activation are taking place are critical to the development of normal functional capabilities in the mature brain (see Meaney et al., 1988). Without certain patterns of activation and certain microenvironmental signals which determine differentiation and facilitate the co-development of these networks, there will be disorganized development and diminished functional capabilities in the mature system.

There are many examples of disrupted neurodevelopment and function in animals following deprivation of sensory cues, primarily visual, tactile, and auditory (Jacobson, 1991). For humans, some extreme illustrations of these principles have been provided by cruel experiments of nature. Children raised with little or no exposure to verbal language never develop the neural apparatus needed for optimal speech or language development (Mason, 1942; Freedman, 1981); children raised in sensory-deprived settings have major deficits in developing integrated neurosensory processing (e.g., Davis, 1940; Freedman and Brown, 1968); children with various visual deficits (e.g., strabismus) develop abnormal visual perceptual and association capabilities (e.g., Lipton, 1970; Bishop, 1987; Freedman, 1992). The length of

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critical and sensitive periods in animals has been documented for a variety of situations. In humans, however, there is very little information regarding these windows of vulnerability; the majority of the irreversible sensory processing deficits have resulted from deprivations during the first three years of life.

The development of networks of neurons mediating a given set of important brain functions is, therefore, dependent upon the quantity, quality, and pattern of activation during key time periods during development. Understanding the principles of this use-dependent development is critically important for understanding the neurodevelopmental effects of childhood trauma. There are, of course, critical and sensitive periods for the development of important brain systems and functions other than neurosensory processing. There is overwhelming evidence suggesting sensitive, if not critical, periods for brain functions associated with mental health, including attachment, affect modulation, anxiety regulation, and behavioral impulsivity (Spitz, 1945; Spitz and Wolf, 1946; Patton and Gardner, 1963; Provence, 1983), all of which utilize to varying degrees the same neurobiological subsystems which mediate the stress response. The best examples of this in humans, again, is from cruel experiments of nature. The orphans described by Spitz (1945) and the more recent Rumanian orphans illustrate the potential neurodevelopmental devastation resulting from affective, tactile, and emotional undernourishment.

The brains of traumatized children develop as if the entire world is chaotic, unpredictable, violent, frightening and devoid of nurturance—and unfortunately, the systems that our society has developed to help these children often continue to fill their lives with neglect, unpredictabilily, fear, chaos and, most disturbing, more violence.

The sensitive periods for the stress response apparatus in the brain—developmental phases during which an individual is most vulnerable to traumatic stressors—occur when the stress-mediating catecholamine systems are undergoing neurogenesis, migration, synaptogenesis and neurochemical differentiation. The functional capabilities of the CNS systems mediating stress in the adult are determined by the nature of the stress experiences during the development of these systems, i.e., in utero, during infancy, and during childhood (Perry, 1988: in press; Perry et al., 1990). A number of fascinating studies in animals demonstrate the exquisite sensitivity of the developing CNS to stress (see Suoumi, 1986).

In rats exposed to perinatal-handling stress, major alterations in the ability of the rat to learn and to mobilize a stress response are seen later in life (Weinstock et al., 1988). The most interesting aspect of these studies is that exposure to unpredictable stress resulted in deficits while exposure to consistent, daily stress resulted in improved or superior behavior—these animals were resilient. The pattern of stress and the predictability are important in determining how traumatic a stressor is. Elements of predictability and some elements of control make the stress much less destructive. One can speculate on equivalent controlled or daily stress and uncontrollable, non-scheduled stressors in the development of a human. An infant who is allowed to have an optimal degree of frustration, one who can control, during rapprochement, his own optimal degree of tension and anxiety (i.e., stress) and return to mother for comfort, is one whose developing CNS is establishing an appropriate neurochemical milieu for the development of a flexible, maximally-adaptive physiological apparatus for responding to future stressors. A child who is reared in an unpredictable, abusive or neglectful environment (see Spitz and Wolfe, 1946) will likely have evoked in his developing CNS a milieu which will result in a poorly organized, dysregulated CNS catecholamine system. One would hypothesize that such an individual would be susceptible to the development of more severe signs and symptoms when exposed to psychosocial stressors through the course of his or her life.

Studies in humans suggest that this is the case. Increased psychiatric symptoms and disorders are observed in adults who have severe, unpredictable early life stressors (Brown and Harris, 1977; Lloyd, 1980; Rutter, 1984). A provocative study by Breier and co-workers (1988) reported the effects of parental loss during childhood on the development of psychopathology in adulthood. They examined a number of adults who had suffered a parental loss during childhood and found that the subjects with psychiatric disorders and symptoms had significant biological and immunological changes related to early parental loss relative to control groups. The authors concluded that early parental loss (a traumatic event) accompanied by the lack of a supportive relationship subsequent to the loss (an external stress-reducing factor) is related to the development of adult psychopathology.

Other studies have documented relationships between developmental trauma and borderline personality disorders (Ogata et al., 1988; Herman et al., 1989), depressive disorders (Kaufman, 1991), dissociative disorders (Putnam, 1991; Peterson, 1991) and a variety of other medical and psychiatric conditions (Coddington, 1972a: 1972b; Garmezy, 1978; Beautris et al., 1982; Boyce, 1990; Greenwood et al., 1990; Davidson et al., 1991). Clearly, these studies provide correlative data indicating that developmental stress is a major expressor of any underlying constitutional or genetic vulnerability and, in some cases, may be the primary etiological factor in the development of certain neuropsychiatric disorders.

The abnormal pattern of stress-mediating neurotransmitter and hormone activations during development alters the brains of traumatized children. The specific nature of these functional alterations is seen in all of the brain functions which are directly or tangentially related to CNS catecholamine systems. Unfortunately, the CNS catecholamines (and likely other important neurotransmitter systems altered by these experiences) are involved in almost all core regulatory activities of the brain. The brainstem and midbrain catecholamines are involved in regulation of affect,

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anxiety, arousal/concentration, impulse control, sleep, startle, autonomic nervous system regulation, memory, and cognition. Clearly the physical signs and symptoms seen in traumatized children include dysfunction and dysregulation in these domains. Indeed, the core symptoms seen in severely traumatized children may be traced back to dysregulation of these root neurophysiological regulatory functions.

Clinical implications

The human brain and all of the functions that this amazing organ mediate develop as a reflection of developmental experiences. This mirroring quality of the developing human brain has evolved as an extension of the primary mandate of the brain to perceive, process, and act on information from the environment in order to maximize survival potential. If the child is raised in an unpredictable, chaotic, violent environment, it is highly adaptive to have a hypervigilant, hyper-reactive arousal system; if primary relationships are characterized by violence, neglect and unreliability, intimacy becomes maladaptive; if a young child is frequently assaulted, it becomes adaptive to overinterpret non-verbal cues, to quickly act on impulses, and to strike out before being struck. The symptoms of hypervigilance, cognitive distortion, physiological and behavioral hyperreactivity, intimacy avoidance and dissociation commonly seen in traumatized children were all, at some time in the lives of these children, necessary, adaptive and appropriate responses to traumatic stress.

The same remarkable qualities of the developing brain which allow the growing child to internalize and rapidly learn about the world ultimately betray the traumatized child. Their brains develop as if the entire world is chaotic, unpredictable, violent, frightening and devoid of nurturance—and unfortunately, the systems that our society has developed to help these children (the juvenile justice, foster care and mental health systems) often continue to fill their lives with neglect, unpredictabililty, fear, chaos and, most disturbing, more violence. Neurodevelopmental principles and the basic neurophysiology of the stress response would predict that the primary, baseline neurophysiological state of the traumatized child is a persisting state of alarm, most similar to a state of fear. Much more research in the basic neurobiology of development and the neurophysiology of traumatized children is required. Only then can the relationships between neurodevelopment and trauma-related neuropsychiatric problems be understood well enough to guide innovative therapeutic approaches and initiate social policy changes to bring an end to the war on children.

Due to space limitations, complete references for this series will be published with the second article in the next issue of The Advisor, *V.6, n 2, 1993. To receive a copy of references for this article immediately, write or call APSAC, 332 S. Michigan Ave., Suite 1600, Chicago, IL 60604. Phone: 312-554-0166.*

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NEW RESOURCE

Development of a Database for Child Protection Teams

--by Marcia Herman-Giddens

The Child Protection Team (CPT) at Duke University Medical Center is a multidisciplinary group that evaluates children from Durham and surrounding areas of North Carolina referred for concerns of abuse or neglect. Since its establishment in 1978, the Team has evaluated almost 5,000 children. The Team is also responsible for teaching physicians, nurses, pediatric residents, physician assistants, and medical students about child abuse and neglect as well as conducting training sessions around the state for other professionals. Research has always been a component of the Team's work.

As our program grew, we began to seek a database that would suit our needs for reporting and research. After talking with colleagues we were not able to identify an existing database that met our purposes. Since we did not have the funds to have the software written by a firm, we submitted our proposal to a software development competition and were fortunate to be chosen.

The system that was developed met our requirements. It is a user-friendly database that records patient demographics, including the protection of confidentiality, the ability to link children with their mothers when the last names were different, and a safeguard against entering duplicate history numbers. It tracks the referral source, with easy access to numbers and sources of referrals along with their addresses. The outcome of each evaluation is recorded, including the diagnosis, final determination of the case, hospital admissions and mortality. We also record necessary follow-ups, and referrals to social service or police agencies. A record of whether or not physical findings were present is also important to us given our ultimate goal of developing a more detailed database of physical and sexual abuse for research and epidemiological purposes. Finally, we are able to transfer data to other databases or to ASCII in order to generate graphics for our reports.

We now use this flexible program for generating the data requirements detailed above as well as for creating weekly listings for meetings and review.

We think other child protection teams might benefit from this database. Droege Computing Services, Inc., has made the database available as Shareware, which gives users a chance to try software before buying it. Systems requirements are as follow:

- IBM compatible (AT, 80286, or later CPU recommended)
- Hard disk
- 640K or more RAM
- MS-DOS 3.1 or later
- LAN compatibility

Readers who are interested in more information may write Droege Computing Services, 1816 Front Street, Suite 130, Durham, NC 27705. For $5.00, Droege will send a floppy diskette and a description of the program for trial.

Marcia Herman-Giddens, PA, MPh, is an Assistant Clinical Professor in the Department of Pediatrics at Duke University Medical Center.