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Outcomes of child abuse

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Injury is the leading cause of death and disability in childhood [1], and traumatic brain injury (TBI) accounts for approximately 30% of all deaths in the 0- to 19-year age group. Unfortunately, not all injuries children incur are accidental in nature. According to Centers for Disease Control and Prevention [1] statistics, although motor vehicle accidents and sports injuries account for 32% of TBI in children ages birth through 4 years, unclassified falls accounted for 55% of injuries. Clear-cut assault and other causes make up the remaining 13%. Although the rates of intentional injury are not explicitly delineated, these statistics conservatively suggest that at least 20% to 25% of early childhood TBI may be inflicted. Stated differently, after uncomplicated skull fractures are excluded, 64% of TBI in infants less than 1 year old is probably caused by abuse [2]. These numbers are particularly sobering when considered in conjunction with recent statistics on child abuse. Nationally, there are over 3 million children whose caregivers were referred for investigation of alleged abuse in 1996 [3]. Of these cases, approximately one third were legally indicated, whereas only 1% were identified as false reports [3]. In the 10 years since the last National Incidence Study was completed in 1986, the reported incidence of physical abuse has increased by 42% [3]. In 1997, it was estimated that 1197 children died as a result of maltreatment [3]. There has also been a disproportionate increase in the incidence of maltreatment of children under the age of 12 years [4] and approximately 75% of all maltreatment-related fatalities occurred in children under the age of 4 years [5]. Physical abuse is the second most prevalent form of child maltreatment [6] and accounts for approximately 21% of identified cases of maltreatment [3].

Younger children, in general, experience more severe injury than their older peers. Almost one quarter of injured infants experience moderate to severe TBI [7]. This fact may be accounted for by the higher incidence of inflicted head injury in children less than 6 years of age. Most infants who experience head trauma do so as a result of a specific type of physical abuse [8]. Frequently termed *shaken baby syndrome* or *nonaccidental head trauma*, clinical findings generally confirm retinal, subdural, or subarachnoid hemorrhages. Approximately half of infants with nonaccidental injury also incur skull fractures [9]. Mortality and morbidity rates in infants with shaken baby syndrome are high. Approximately 25% of infants with shaken baby syndrome do not survive; of those who do survive, the outcome may be far worse than nonaccidental forms of brain trauma [9]. Sinal and Ball [10] studied infants who presented for initial examination in coma. Of the infants who survived, the range of deficits was wide, from profound mental retardation (e.g., intelligence quotient ≤25 [11]) to subtle neurologic sequelae or seizures. These latter symptoms were noted in infants without central nervous system (CNS) lacerations or infarctions and without increase in intracranial pressure.

Outcome after TBI in younger children

Although recognized as a clinical problem over three decades ago [12], investigations of the

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outcome of inflicted TBI are rare. This is somewhat surprising because injury incurred to a developing brain is now considered to be a risk factor for poorer outcome [13]. Although researchers are beginning to appreciate the importance of examining the immediate and long-term outcome of TBI in infants and young children, few studies delineate mechanism of injury in a way that allows for the comparison of outcome after inflicted versus noninflicted TBI. In fact, many studies of both younger and older children specifically exclude children with suspected child abuse from the sample [7,14]. One important exception is the study by Anderson et al [15]. In that study of children ranging in age from 2 to 5 years who had sustained TBI, mechanism of injury included falls or blows in 36% and other causes in 6%. This raises the possibility that inflicted abuse may have occurred in well over a third of this group of toddlers and children. Children were administered a battery of standardized neuropsychologic instruments to assess outcome at 1 year. In that controlled study, severity of injury, whether inflicted or not, was related to poorer intellectual development, impaired language abilities, and memory deficits.

Studies of inflicted versus accidental injury

Children, and particularly infants [16], who sustain inflicted head injuries may incur a worse outcome than those who experience accidental injury [17]. In the acute period, infants who experience bilateral diffuse hypodensity on CT scans are those with the poorest prognosis, with variable recovery noted in infants whose injury required prophylactic treatment with anticonvulsants. A large retrospective study [18] examined differences in functional outcome at hospital discharge among children incurring either accidental or nonaccidental injuries between the ages of birth to age 4 years. Although this study was not specific to TBI, the rates of head injury were high in the inflicted injury sample. More than two thirds of the abused children incurred intracranial injury as compared with approximately 25% of the unintentional injury group. In addition, injuries in the inflicted group were more severe, and outcomes generally reflect this fact. In this study, children were classified according to number of functional limitations and neurologic sequelae. Although findings were limited because many of the subjects in the abuse group were too young to assess functional status accurately, significant differences were identified between the two groups, with the inflicted group showing more serious difficulties completing activities of daily living and poorer neurologic ratings. These findings suggest that a more comprehensive evaluation of the cognitive status of these two groups might identify important differences during both the shorter and longer term after injury.

One recent controlled longitudinal study [17] did focus on the cognitive and functional outcome of infants, toddlers, and children approximately 6 weeks after inflicted TBI. Children in the study group were compared with a sample of children having accidental injury but who were similar in neonatal history and injury severity as assessed by the Glasgow Coma Scale. Although none of the children in either group had a documented history of previous brain injury, 45% of the children in the inflicted injury group, but none in the accidental group, exhibited at least one sign of preexisting brain trauma. This study applied a measure of global neurobiologic outcome (e.g., Glasgow Outcome Scale) and standardized and age-appropriate measures of cognitive and motor development. Although findings are complicated by the fact that children in the inflicted injury group were younger and required different instruments to assess cognitive status than the accidental group, significantly more children in the inflicted injury group had test scores in the mentally deficient range. Motor scores did not demonstrate the same effect.

Over the longer term, anecdotal studies indicate that children with inflicted TBI may have a poorer outcome than those children with nonaccidental injury. Based on a retrospective review of records of 33 children under the age of 24 months who had incurred subdural hemorrhage 1 year earlier [19], infants and toddlers who incurred inflicted TBI continued to exhibit poorer outcome than their peers with accidental injury. Child abuse was confirmed or strongly suggested in 63.3% and 18.2% of the cases, respectively. Although nine infants were reported to be functioning normally, an equal number had died and 15 infants demonstrated profound disability (e.g., motor impairment, speech delay, or seizure disorder). In this study, normalcy was assessed by clinical presentation rather than by standardized neuropsychologic or developmental testing.

Two studies demonstrate the importance of evaluation of the outcome past the 1-year anniversary of injury in infants who experience shaken baby syndrome. In a study by Bonnier et al [20], 13 infants, 12 of whom were less than or equal to 6

months of age, all met criteria for shaken baby syndrome, demonstrating retinal hemorrhages and intracranial lesions with the absence of any other traumatic or nontraumatic mechanism of injury. The group was divided into those who experienced immediate signs and symptoms of TBI (N = 7) and those who were free of such signs (N = 6) at a 2-month follow-up examination. Perhaps the most compelling argument for the careful assessment of extended outcome after inflicted head injury comes from the finding that all but one of the normal children became disabled after a delay ranging from 6 months to 5 years. By the fifth year of follow-up, five of the six children met criteria for mental retardation. A second study [21] located 22% of an infant sample that had incurred shaken baby syndrome 10 years earlier. The outcome of this group was evaluated using age-appropriate modifications of the Glasgow Outcome Scale. Over half of the children studied had extremely poor outcome, ranging from death caused by respiratory complications to severe disability (e.g., mental retardation and blindness). Taken together, these findings must be interpreted cautiously because they are compromised by the fact that the neurologists and psychologists examining the patients were not blind as to clinical status and there was no control group. It seems, however, that long-term outcome should be investigated more carefully in infants who experience inflicted head injury.

In summary, the outcome of children with inflicted head trauma may be different from that associated with accidental injury. Although most of the literature reviewed here stresses the importance of risk prevention and accurate diagnosis, the effects of this particular type of neurotrauma indicate that there is an important role for outcome assessment over the longer term. Although anecdotal reports of attention deficit, learning disabilities, motor deficits, and developmental delays are mentioned in the literature, empirical studies have yet to investigate these varied outcomes in a rigorous way. In addition, research suggests that inflicted head injury does not occur in isolation and that it is likely linked inextricably to ongoing child abuse. For example, in the Jayawant et al study [22], 12% of the sample had a history of previous abuse, and the abuse of another child in the family was identified in 6 of the 17 children with siblings. This gives rationale to assessing children with a history of early child abuse for posttraumatic stress disorder (PTSD), a psychiatric illness whose likelihood increases in children who have experienced a history of abuse and neglect. As with head trauma, researchers are just beginning to understand the impact of PTSD on the developing brain.

Developmental impact of inflicted childhood head injury

The occurrence of inflicted TBI seems to be strongly associated with ongoing abuse within the family [22]. Although the actual incidence of child abuse and maltreatment is difficult to determine, reports of suspected abuse have risen by almost 50% over the last 10 years. According to a 1995 report, of the 1.5 million cases of reported and substantiated child abuse, 25% involved physical abuse [9]. A recent epidemiologic study of hospital admissions for child abuse and neglect in children from birth to 9 years documented head trauma in 22% and shaken baby syndrome, categorized separately, in 6% of the group [38].

As Fulton [2] suggests, "…psychological recovery [from shaken baby syndrome] may never be complete." The authors' recent work in the field of abuse and neglect indicates that inflicted childhood head trauma may indeed have a far-reaching impact, with deterioration in the CNS occurring well beyond the acute injury period. Although one might characterize this impact as psychiatric in nature, work in developmental traumatology is beginning to characterize the neurobiology of trauma associated with child abuse and neglect that is not necessarily overtly physical in nature [37].

Developmental traumatology is a relatively new area of child psychiatry that investigates the psychiatric and psychobiologic impact of maltreatment on the developing child. Concepts from developmental psychopathology, developmental neuroscience, and stress and trauma research are integrated to investigate the consequences of child maltreatment on the development and regulation of major biologic stress response systems and their subsequent impact on brain development and function [24].

In this work the PTSD model is applied to study childhood abuse and neglect. PTSD is now widely recognized in the pediatric population and the diagnostic criteria are similar to those that are applied to adults [23,24]. One exception is that in children younger than 4 years, objective evidence based on observable behaviors rather than subjective experiences is warranted [25]. Important to the current discussion is the fact that children and adolescents with PTSD experience a high rate

of physical abuse. In clinically referred samples incidence rates range as high as 50% [26,27]. Nonclinically referred samples show a somewhat smaller but still significant incidence of PTSD related to maltreatment. For example, Famularo et al [28] reported a 39% incidence rate in their sample.

The neurobiology of trauma

Recent research indicates that child abuse and maltreatment have a traumatic impact on biologic and psychologic development and might be viewed as an environmentally complex developmental disorder [24]. In the authors' laboratory, studies have been completed that explore brain changes associated with abuse and neglect from both neuroimaging and neuropsychologic perspectives.

Imaging studies

Previous investigations suggest that maltreated children with PTSD may experience permanent alterations of biologic stress systems that adversely affect brain development [29]. The authors recently reported findings of a cross-sectional study of 44 maltreated children and adolescents with PTSD and 61 matched healthy comparison subjects, including anatomic MRI scans to measure various brain structures. MRI was performed using a General Electric 1.5-T unit. Coronal sections were obtained perpendicular to the anterior-commissure–posterior-commissure line to provide a more reproducible guide for image orientation. Axial proton density and T2-weighted images were obtained to enable a neuroradiologist to exclude clinically significant structural abnormalities. The interested reader is referred to De Bellis et al [29] for detailed information regarding image analysis and statistical methods. Compared with controls, maltreated children and adolescents had smaller intracranial and cerebral volumes than nonabused comparison subjects. After adjustment for intracranial volume, the total midsagittal area of the corpus callosum and middle and posterior regions were smaller. Conversely, the right, left, and lateral ventricles were proportionally larger in the maltreated group as compared with controls. Brain volume showed a significant positive correlation with age of onset of PTSD and a significant negative correlation with duration of abuse. Various hallmark psychiatric symptoms of PTSD (e.g., intrusive thoughts, avoidance, and hyperarousal) correlated positively with ventricular volume and negatively with brain volume and total corpus callosum and regional measurements. These data suggest that maltreatment in childhood, whether or not it includes documented TBI, may be associated with adverse influences on brain development.

In a second study [30] the authors demonstrated functional differences in the brain metabolism between maltreated children and adolescents with PTSD and matched controls. MR spectroscopy (MRS) provides a novel approach to measure in vivo neurobiologic alterations in children's brains. MRS can be applied to measure the *N*-acetyl (NA) resonance signal in the proton (^{1}H) spectrum. *N*-acetylaspartate (NAA) is the principal source of this NA signal, and is a metabolite thought to be present only in neurons. Measures of NAA are markers of neural integrity. Decreased NAA is consistently associated with neuronal loss [31]. The prefrontal cortex is actively developing during childhood and adolescence. Recent positron emission tomography studies provide evidence of anterior cingulate involvement in adult women without PTSD who had experienced sexual abuse as children [32,33] and in Vietnam veterans with PTSD [34]. The authors' own MRI study [29], discussed previously, found a trend for a decrease in the genu, an area of the corpus callosum that subserves the anterior cingulate of the prefrontal cortex. The anterior cingulate region is part of an executive attention system because it is activated during decision making and novel or dangerous situations [35]. Because intrusive thoughts of trauma (i.e., danger) and poor concentration are core symptoms of PTSD, it was hypothesized that cingulate integrity may be affected in PTSD.

To test this hypothesis, the authors compared measures of anterior cingulate NAA in case-matched pairs of PTSD and control subjects. The authors predicted reduced NAA in abused subjects with PTSD compared with healthy nonabused controls. The two groups were matched with respect to age, sex, socioeconomic status, and intelligence quotient. In vivo localized ^{1}H MRS was conducted on a voxel placed in the cingulate using the 1.5-T MRI scanner at the University of Pittsburgh Medical Center Magnetic Resonance Research Center. The position of the voxel ($2\times 1.5\times 1$ cm, volume = 3 mL) was determined from sagittal and coronal MR images at the anterior-most point of the genu of the corpus callosum. A stimulated echo acquisition mode pulse sequence with a TE of 25 milliseconds, and TR of 1.5 seconds and 300 acquisitions was used. The MRI spectrum was obtained with maximal receiver gain, 2500-Hz sweep width, and 2048-point spectral resolution. Spectral postprocessing and quantification were

performed for NAA and the other metabolites (choline and creatine) using estimations from an external standard (i.e., from spectra of phantoms containing known concentrations of these compounds using SAGE software). NAA concentration was quantified in relationship to H_2O concentration and compared using a two-tailed Wilcoxon signed rank test for paired samples. For further details of ^1H MRS methods see De Bellis et al [30]. The ratio of NAA to creatine was significantly lower in the maltreated children and adolescents with PTSD than in the comparison subjects. This study must be considered preliminary because of several limitations (e.g., small sample size, use of a single voxel for MRS studies, tissue heterogeneity within the voxel, and lack of absolute quantification of metabolite measures). To the authors' knowledge, however, it is the first to report lower levels of NAA in the cingulate region in children and adolescents with PTSD. These findings suggest neuronal loss that is consistent with the possible alteration of neuronal metabolism in maltreated children with PTSD.

Neuropsychologic outcome

The authors' neuroimaging studies of children with maltreatment-related PTSD indicate that the disorder is associated with diffuse CNS effects (i.e., smaller cerebral volumes and corpus callosum areas) but no anatomic changes in limbic structures [29]. Functional imaging procedures described previously indicate that medial prefrontal cortical dysfunction may be associated with both adult and pediatric PTSD [30]. The authors hypothesized that PTSD subjects would perform more poorly on cognitive measures, particularly the domains mediated by the prefrontal cortex. A review of the literature, however, confirms that cognitive function as indexed by performance on standardized neuropsychologic instruments has not been extensively evaluated in children with PTSD. It is particularly important to characterize the neuropsychologic deficits associated with childhood PTSD because they are likely to have broad developmental ramifications, affecting both responses to therapy and school performance.

Because the neuropsychologic consequences of these brain alterations have not been studied extensively, the authors examined cognitive functioning in maltreated child and adolescent subjects with PTSD and nonmaltreated comparison subjects [36]. Children completed a comprehensive neuropsychologic assessment, including measures of language, attention, abstract reasoning and executive function, learning and memory, visual-spatial processing, and psychomotor functioning. The sample was comprised of 14 medication-naive children and adolescents with PTSD secondary to maltreatment and 15 healthy nonmaltreated comparison subjects similar to the PTSD subjects in age, sex, race, socioeconomic status, and intelligence quotient. Diagnosis of PTSD and maltreatment history was confirmed for all subjects by a psychiatric interview by a board-certified child psychiatrist. Comparison subjects had no lifetime history of any axis I diagnosis according to the *Diagnostic and Statistical Manual-IV* [11].

Inclusion criteria for the PTSD group were reported and indicated child maltreatment experiences by Child Protective Services before this investigation. The availability of one nonabusing caregiver who could cooperate with this protocol and a stable home environment (i.e., child in no danger from perpetrators for at least the prior 3 months) were also study requirements. Children with complicated medical (e.g., birth complications or known head injury with loss of consciousness), psychological (e.g., intelligence quotient <80, pervasive developmental disorder, or learning disability), or psychiatric (e.g., schizophrenia, treatment with psychotropic medications, substance abuse, or dependence) histories were excluded from the sample. Children completed a comprehensive neuropsychologic battery that included tests of language, attention, abstract reasoning and executive function, learning and memory, visual-spatial skills, and psychomotor function. Children with PTSD performed more poorly on measures within four of the six cognitive domains. After a Bonferroni correction for multiple comparisons was applied within each of the cognitive domains, results within the domains of attention (i.e., Stroop Color/Word, Digit Vigilance omissions) and executive function (i.e., Wisconsin Card Sorting Test number of categories, Animal Naming) remained significant.

Children with maltreatment-related PTSD demonstrated significant deficits within the domains of attention and executive function when compared with nonmaltreated sociodemographically similar children. PTSD children were more susceptible to distraction and demonstrated increased impulsivity, making more errors on a task of sustained attention. PTSD children also demonstrated deficits on the Wisconsin Card Sorting Test, an instrument requiring hypothesis testing and problem solving, and on a measure of semantic organization, two tests designed to measure frontal lobe

function [19]. These findings are consistent with the authors' neuroimaging studies showing CNS changes in the frontal cortex. In contrast, after corrections to protect from experiment-wise error, PTSD children did not perform differently than comparison children on measures of language, memory and learning, visual-spatial abilities, or psychomotor skills. Findings were limited, however, by the small sample size and work continues with a larger sample.

Although this study must be considered preliminary because of the lack of a maltreated comparison group without PTSD, maltreatment-related PTSD was associated with attention and executive skill deficits. Although this study was not designed to assess outcome after inflicted child abuse, these findings suggest that there may be complicated and probably synergistic interaction between sequelae of inflicted TBI and physiologic changes in the brain associated with the chronic stress. Further studies that include groups with and without PTSD related to head trauma are necessary to assess the complex relationship between these two disorders.

Summary

The limited research available regarding the outcome of inflicted TBI suggests that this type of injury may be especially deleterious to infants and young children. It is likely that mechanisms of injury, age at injury, and circumstances of injury (i.e., child abuse and maltreatment) all contribute to these findings. Until recently, the investigations of TBI and child abuse and maltreatment have occurred on two separate tracks. The review of these two literatures indicates that the TBI outcome literature is strongly grounded in neuropsychologic methodology, whereas the child abuse and maltreatment literature depends most heavily on less brain-specific measures of general intellectual ability and academic achievement. The evidence reviewed here suggests that it is time for these areas of research to converge. Children with inflicted head injury should be evaluated not only to assess outcome related to TBI but to disconfirm the presence of PTSD, a developmental disorder that may also result in CNS changes.

In this vulnerable population, longitudinal assessment well past the period of initial insult is imperative to assess the rate of development of skills, to identify deficient areas, and to plan appropriate interventions.

References

[1] Division of Injury Control. Center for Environmental Health, Centers for Disease Control: Childhood injuries in the United States. Am J Disabled Child 1990;144:627–46.
[2] Fulton DR. Shaken baby syndrome. Crit Care Nursing Quat 2000;23:43–50.
[3] US Department of Health and Human Services. Child maltreatment 1996: Reports from the states to the national child abuse and neglect data system. Washington, DC: US Government Printing Office; 1998.
[4] Sedlak AJ, Broadhurst DD. Third national incidence study of child abuse and neglect. Washington, DC: US Department of Health and Human Services; 1996.
[5] US Department of Health and Human Services. Child maltreatment 1997: Reports from the states to the national child abuse and neglect data system. Washington, DC: US Government Printing Office; 1999.
[6] National Research Council. Understanding abuse and neglect. Washington, DC: National Academy Press; 1993.
[7] Ewing-Cobbs L, Fletcher JM, Levin HS, et al. Longitudinal neuropsychological outcome in infants and preschoolers with traumatic brain injury. J Int Neuro psychol Soc 1997;3:581–91.
[8] Committee on Child Abuse and Neglect. Shaken baby syndrome: inflicted cerebral trauma. Am Acad Pediatr 1993;92:872–5.
[9] Conway Jr EE. Nonaccidental head injury in infants: the shaken baby syndrome revisited. Pediatr Ann 1998;27:677–90.
[10] Sinal SH, Ball MR. Head trauma due to child abuse: serial computerized tomography in diagnosis and management. South Med J 1987;80:1505–12.
[11] American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th edition. Washington, DC: American Psychiatric Association; 1994.
[12] Caffey J. On the theory and practice of shaking infants: its potential residual effects of permanent brain damage and mental retardation. Am J Disabled Child 1972;124:161–9.
[13] Taylor HG, Alden J. Age-related differences in outcomes following childhood brain insults: an introduction and overview. J Int Neuropsychol Soc 1997; 3:555–67.
[14] Levin HS, Song J, Scheibel RS, et al. Concept formation and problem-solving following closed head injury in children. J Int Neuropsychol Soc 1997;3: 598–607.
[15] Anderson VA, Morse SA, Klug G, et al. Predicting recovery from head injury in young children: a prospective analysis. J Int Neuropsychol Soc 1997;3: 568–80.
[16] Duhaime AC, Christian CW, Rorke LB, et al. Current concepts: nonaccidental head injury in infants—

The shaken baby syndrome. N Engl J Med 1998;338: 1822–9.
[17] Ewing-Cobbs L, Kramer L, Prasad M, et al. Neuroimaging, physical, and developmental findings after inflicted and noninflicted head traumatic brain injury in young children. Pediatrics 1998;102:300–7.
[18] DiScala C, Sege R, Li G, et al. Child abuse and unintentional injuries: a 10-year retrospective. Arch Pediatr Adolesc Med 2000;154:16–22.
[19] Levin HS, Culhane KA, Hartman J, et al. Developmental changes in performance on tests of purported frontal lobe functioning. Dev Neuropsychol 1991;12:377–95.
[20] Bonnier C, Nassogne M, Evrard P. Outcome and prognosis of whiplash shaken infant syndrome: late consequences after a symptom-free interval. Dev Med Child Neurol 1995;37:943–56.
[21] Duhaime AC, Christian C, Moss E, et al. Long term outcome in infants with the shaking-impact syndrome. Pediatr Neurosurg 1996;24:292–8.
[22] Jayawant S, Rawlinson A, Gibbon F, et al. Subdural haemorrhages in infants: population based study. BMJ 1998;317:1558–61.
[23] Pynoos RS, Eth S, editors. Witnessing acts of personal violence. Washington, DC: American Psychiatric Press; 1985.
[24] De Bellis MD. Developmental traumatology: the psychobiological development of maltreated children and its implications for research, treatment, and policy. Dev Psychopathol 2001;13:537–61.
[25] Scheeringa MS, Zeanah CH, Drell MJ, et al. Two approaches to the diagnosis of posttraumatic stress disorder in infancy and early childhood. J Am Acad Child Adolesc Psychiatry 1995;34:191–200.
[26] Dubner AE, Motta RW. Sexually and physically abused foster care children and posttraumatic stress disorder. J Consult Clin Psychol 1999;67: 367–73.
[27] Green A, editor. Children traumatized by physical abuse. Washington, DC: American Psychiatric Press; 1985.
[28] Famularo R, Fenton T, Kinscherff R. Maternal and child posttraumatic stress disorder in cases of maltreatment. Child Abuse Negl 1994;18:27–36.
[29] De Bellis MD, Keshavan MS, Clark DB, et al. Developmental traumatology Part II: brain development. Biol Psychiatry 1999;45:1271–84.
[30] De Bellis MD, Keshavan MS, Spencer S, et al. N-acetylaspartate concentration in the anterior cingulate of maltreated children and adolescents with PTSD. Am J Psychiatry 2000;157:1175–7.
[31] Prichard JW. MRS of the brain—prospects for clinical application. In: Young IR, Charles HC, editors. MR spectroscopy: clinical applications and techniques. London: Livery House; 1996. p. 1–25.
[32] Shin LM, McNally RJ, Kosslyn SM, et al. Regional cerebral blood flow during script-driven imagery in childhood sexual abuse-related PTSD: a PET investigation. Am J Psychiatry 1999;156:575–84.
[33] Bremmer JD, Narayan M, Staib LH, et al. Neural correlates of memories of childhood sexual abuse in women with and without posttraumatic stress disorder. Am J Psychiatry 1999;156:1787–95.
[34] Bremmer JD, Staib LH, Kaloupek D, et al. Neural correlates of exposure to traumatic pictures and sound in Vietnam combat veterans with and without posttraumatic stress disorder. Biol Psychiatry 1999;45:806–16.
[35] Posner MI, Petersen SE. The attention system of the human brain. Ann Rev Neuroscience 1990;13:25–42.
[36] Beers SR, De Bellis MD. Neuropsychological function in children with maltreatment-related posttraumatic stress disorder. Am J Psychiatry 2002; 159:483–6.
[37] De Bellis MD. Posttraumatic stress disorder and acute stress disorder. In: Ammerman RT, Hersen M, editors. Handbook of prevention and treatment with children and adolescents. New York: John Wiley; 1997. p. 455–94.
[38] Sbalchiero J, Orsay E, Bunney EB, et al. Child abuse and neglect in Illinois, 1996–1998. Ann Emerg Med 2000;36:S48.