Goals of Trauma Treatment: A Biopsychosocial Perspective


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Elements of Traumatic Experience

“What is Trauma?”

1 a: an injury (as a wound) to living tissue caused by an extrinsic agent  
   b: a disordered psychic or behavioral state resulting from severe mental or emotional stress or physical injury  
   c: an emotional upset <the personal trauma of an executive who is not living up to his/her own expectations>

Merriam-Webster Dictionary

http://www.merriam-webster.com/dictionary/trauma
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Merriam-Webster Dictionary

http://www.merriam-webster.com/dictionary/trauma
## Epidemiology of Traumatic Experience

### Traumatic Experience - Lifetime Prevalence

<table>
<thead>
<tr>
<th>Study</th>
<th>Prevalence (M)</th>
<th>Prevalence (F)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kulka et al (1990)</td>
<td>44.5%</td>
<td>37.2%</td>
</tr>
<tr>
<td>Breslau et al (1991)</td>
<td>43.0%</td>
<td>36.7%</td>
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<tr>
<td>Norris (1992)</td>
<td>73.6%</td>
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<tr>
<td>Resnick et al (1993)</td>
<td>----</td>
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<tr>
<td>Kessler et al (1995)</td>
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<tr>
<td>Breslau et al (1997)</td>
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<td>40%</td>
</tr>
<tr>
<td>Stein et al (1997)</td>
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<td>74.2%</td>
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</table>

### Sources of Traumatic Experience

#### Types of Traumatic Experience – Lifetime Prevalence

<table>
<thead>
<tr>
<th>Event</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rape</td>
<td>0.7% (M)</td>
</tr>
<tr>
<td>Sexual Assault</td>
<td>2.8% (M)</td>
</tr>
<tr>
<td>Witnessing Violence</td>
<td>35.6% (M)</td>
</tr>
<tr>
<td>Accidents</td>
<td>25% (M)</td>
</tr>
<tr>
<td>Car Accidents</td>
<td>32.8% (M)</td>
</tr>
<tr>
<td>Threatened w/ weapon</td>
<td>19% (M)</td>
</tr>
<tr>
<td>Physical Attack</td>
<td>11.1% (M)</td>
</tr>
<tr>
<td>Badly beaten up</td>
<td>13.1% (M)</td>
</tr>
<tr>
<td>Mugged</td>
<td>34% (M)</td>
</tr>
<tr>
<td>Shot/stabbed</td>
<td>8.2% (M)</td>
</tr>
<tr>
<td>Natural Disaster</td>
<td>18.9% (M)</td>
</tr>
<tr>
<td>Learning about trauma</td>
<td>63.1% (M)</td>
</tr>
<tr>
<td>Unexpected death</td>
<td>61.1% (M)</td>
</tr>
</tbody>
</table>

National Co-morbidity Survey and Detroit Area Survey Data (rate/100 people)

Traumatic Experience and Psychiatric Disturbance - PTSD

Types of Traumatic Experience – Lifetime Prevalence

- Assaultive violence: 20.9% (3.4%)
- Raped: 49% (12.2%)
- Shot or stabbed: 15.4% (13.7%)
- Other sexual assault: 23.7% (10.8%)
- Mugged, held up, threatened w/ weapon: 8% (3.7%)
- Badly beaten up: 31.9% (8.6%)
- Serious car or vehicle accident: 2.3% (1.3%)
- Other serious accident or injury: 16.8% (6.2%)
- Fire, flood, earthquake, natural disaster: 3.8% (3%)
- Life-threatening diagnosis: 1.1% (.9%)
- Unexpectedly discovering dead body: 2.3% (.2%)
- Learning of trauma to others: 2.2% (.7%)
- Unexpected death of friend or relative: 14.3% (2.6%)
- Any Trauma: 9.2% (1.0%)

Detroit Area Survey of Trauma Data (rate/100 people)

Trauma-Related Diagnoses in the DSM-IV TR

- **Diagnostic criteria for Adjustment Disorders**
  A. The development of emotional or behavioral symptoms in response to an identifiable stressor(s) occurring within 3 months of the onset of the stressor(s).
  B. These symptoms or behaviors are clinically significant as evidenced by either of the following:
  (1) marked distress that is in excess of what would be expected from exposure to the stressor
  (2) significant impairment in social or occupational (academic) functioning
  C. The stress-related disturbance does not meet the criteria for another specific Axis I disorder and is not merely an exacerbation of a preexisting Axis I or Axis II disorder.
  D. The symptoms do not represent Bereavement.
  E. Once the stressor (or its consequences) has terminated, the symptoms do not persist for more than an additional 6 months.

- Specify if:
  - **Acute**: if the disturbance lasts less than 6 months
  - **Chronic**: if the disturbance lasts for 6 months or longer

- Adjustment Disorders are coded based on the subtype, which is selected according to the predominant symptoms.
  - The specific stressor(s) can be specified on Axis IV.

- **309.0 With Depressed Mood**
- **309.24 With Anxiety**
- **309.28 With Mixed Anxiety and Depressed Mood**
- **309.3 With Disturbance of Conduct**
- **309.4 With Mixed Disturbance of Emotions and Conduct**
- **309.9 Unspecified**

DSM-IV TR, American Psychiatric Association 2004; retrieved from Behavenet.com
Trauma-Related Diagnoses in the DSM-IV TR

**Diagnostic criteria for 308.3 Acute Stress Disorder**

A. The person has been exposed to a traumatic event in which both of the following were present:

1. the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others
2. the person's response involved intense fear, helplessness, or horror

B. Either while experiencing or after experiencing the distressing event, the individual has three (or more) of the following dissociative symptoms:

1. a subjective sense of numbing, detachment, or absence of emotional responsiveness
2. a reduction in awareness of his or her surroundings (e.g., "being in a daze")
3. derealization
4. depersonalization
5. dissociative amnesia (i.e., inability to recall an important aspect of the trauma)

C. The traumatic event is persistently reexperienced in at least one of the following ways: recurrent images, thoughts, dreams, illusions, flashback episodes, or a sense of reliving the experience; or distress on exposure to reminders of the traumatic event.

D. Marked avoidance of stimuli that arouse recollections of the trauma (e.g., thoughts, feelings, conversations, activities, places, people).

E. Marked symptoms of anxiety or increased arousal (e.g., difficulty sleeping, irritability, poor concentration, hypervigilance, exaggerated startle response, motor restlessness).

F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or impairs the individual's ability to pursue some necessary task, such as obtaining necessary assistance or mobilizing personal resources by telling family members about the traumatic experience.

G. The disturbance lasts for a minimum of 2 days and a maximum of 4 weeks and occurs within 4 weeks of the traumatic event.

H. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition, is not better accounted for by Brief Psychotic Disorder, and is not merely an exacerbation of a preexisting Axis I or Axis II disorder.

DSM-IV TR, American Psychiatric Association 2004; retrieved from Behavenet.com
Trauma-Related Diagnoses in the DSM-IV TR

- **Diagnostic criteria for 309.81 Posttraumatic Stress Disorder**
  A. The person has been exposed to a traumatic event in which both of the following were present:
  - (1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others
  - (2) the person's response involved intense fear, helplessness, or horror. **Note:** In children, this may be expressed instead by disorganized or agitated behavior
  B. The traumatic event is persistently reexperienced in one (or more) of the following ways:
  - (1) recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. **Note:** In young children, repetitive play may occur in which themes or aspects of the trauma are expressed.
  - (2) recurrent distressing dreams of the event. **Note:** In children, there may be frightening dreams without recognizable content.
  - (3) acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, **illusions**, **hallucinations**, and dissociative **flashback** episodes, including those that occur on awakening or when **intoxicated**). **Note:** In young children, trauma-specific reenactment may occur.
  - (4) intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
  - (5) physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
  C. Persistent avoidance of stimuli associated with the trauma and **numbing** of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:
    - (1) efforts to avoid thoughts, feelings, or conversations associated with the trauma
    - (2) efforts to avoid activities, places, or people that arouse recollections of the trauma
    - (3) inability to recall an important aspect of the trauma
    - (4) markedly diminished interest or participation in significant activities
    - (5) feeling of detachment or estrangement from others
    - (6) restricted range of **affect** (e.g., unable to have loving feelings)
    - (7) sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span)
  D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:
    - (1) difficulty falling or staying asleep
    - (2) **irritability** or outbursts of anger
    - (3) difficulty concentrating
    - (4) **hypervigilance**
    - (5) exaggerated **startle response**
  E. Duration of the disturbance (symptoms in Criteria B, C, and D) is more than 1 month.
  F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
  **Specify if:**
  - **Acute:** if duration of symptoms is less than 3 months
  - **Chronic:** if duration of symptoms is 3 months or more
  - **Specify if:**
  - **With Delayed Onset:** if onset of symptoms is at least 6 months after the stressor

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**DSM-IV TR, American Psychiatric Association 2004**

retrieved from Behavenet.com
Long-Term Treatment and Prevention of Posttraumatic Stress Disorder

Jonathan R. T. Davidson, M.D.

Posttraumatic stress disorder (PTSD) is a disabling condition almost universally associated with psychiatric co-morbidity, reduced quality of life, and a chronic, often lifelong, course. Although acute treatment with selective serotonin reuptake inhibitors (SSRIs) has been shown to be effective, successful strategies for preventing PTSD have not been established. In addition, studies of the long-term treatment of chronic PTSD are just beginning to emerge. This review considers available evidence for the secondary prevention of PTSD in the acute aftermath of trauma and the long-term treatment of established PTSD. Unanswered questions pertaining to duration of treatment, candidates for long-term treatment, and potentially harmful treatments will also be considered.

(J Clin Psychiatry 2004;65[suppl 1]:44-48)

Posttraumatic stress disorder (PTSD), which has long been known to be a negative consequence of wartime exposure, is becoming increasingly recognized as a psychiatric illness of significance in civilian populations around the world. This is particularly relevant for countries in which civilians of the general community are exposed to ongoing violence and unrest. Not surprisingly, the worldwide prevalence of PTSD in civilian populations varies widely, from 37.4% in Algeria to 1.3% in Germany. As predicted by the World Health Organization's Global Burden of Disease study, exposure to traumatic events such as combat and natural disasters significantly increases the risk of PTSD. Interestingly, the risk of PTSD is also increased among individuals who have previously experienced psychological trauma, such as sexual abuse. In fact, untreated PTSD had symptoms for a mean duration of 5 years. Unfortunately, PTSD rarely exists as a pure disorder, and psychiatric comorbidities, such as depression, alcohol abuse, substance abuse, other anxiety disorders, and suicidality, add to the burden of this disorder. The net effect of PTSD and its associated comorbidities is significantly impaired quality of life, missed educational and occupational opportunities, and excessive hospitalization and use of health care resources. The social and economic burden of PTSD is heavy for patients, their friends and families, and society in general.2 Early diagnosis and treatment of PTSD is crucial in order to prevent the chronic and lifelong course of this disorder.
How can Traumatic Experience be an Etiological Factor for Psychiatric Illness?

- Trauma = *Psychiatric Disturbance*???
  - Traumatic Experiences Produce Excessive Stress


**Stage 1. Alarm Reaction/Compensation:** Any physical or mental trauma will trigger an immediate set of reactions that combat the stress. Because the immune system is initially depressed, normal levels of resistance are lowered, making us more susceptible to infection and disease. If the stress is not severe or long-lasting, we bounce back and recover rapidly.

**Stage 2: Resistance:** Eventually, sometimes rather quickly, we adapt to stress, and there's actually a tendency to become more resistant to illness and disease. Our immune system works overtime for us during this period, trying to keep up with the demands placed upon it. We become complacent about our situation and assume that we can resist the effects of stress indefinitely. Therein lies the danger. Believing that we are immune from the effects of stress, we typically fail to do anything about it.

**Stage 3: Exhaustion/De-compensation:** Because our body is not able to maintain homeostasis and the long-term resistance needed to combat stress, we invariably develop a sudden drop in our resistance level. No one experiences exactly the same resistance and tolerance to stress, but everyone's immunity at some point collapses following prolonged stress reactions. Life sustaining mechanisms slow down and sputter, organ systems begin to break down, and stress-fighting reserves finally succumb to what is called "diseases of adaptation."
Clinical "Shock"
Loss of Body Weight +N
Gastro-Intestinal Ulcers
Temporary Rise in Plasma K
Temporary Fall in Plasma Cl

Pathway Unknown
But Not Involving
Hypophyseal and/or
Adrenal Hyperactivity

NONSPECIFIC DAMAGE

HYPOPHYSIS
(Increased Production of
Corticotrophic Hormones)

Decreased Production of
Gonadotropic Hormone

SOMATIC GROWTH
(Inhibition)

MAMMARY GLANDS
(Cessation of Lactation)

TESTIS
(Involution of Male Accessory Sex Organs)

OVARY
(Anomalies of Sexual Cycle)

ADRENAL CORTEX
(Enlargement and Increased
Product of Corticoid Hormones)

Corticotropic Hormones

CARBOHYDRATE METABOLISM

THYMUS
and other

LYMPHATIC ORGANS
(Involution)

??

CIRCULATING LYMPHOCYTES
(Lymphopenia increased
g-Globulin and Antibodies in Blood)

ELECTROLYTE METABOLISM
(Na Retention)

BLOOD VESSELS
(Periarteritis Nodosa)

HEART
(Myocarditis Infarcts
Aschoff's (?) Nodules)

KIDNEY
(Nephrosclerosis) (Polyarthritis)

JOINTS
(Hypertension)
1. Amygdala detects stress (biological/psychological origin) and stimulates the Hypothalamus
2. Hypothalamus releases Corticotrophin Releasing Factor (CRF)
3. CRF stimulates the Pituitary
4. Pituitary releases Adrenocorticotropic Hormone (ACTH)
5. ACTH stimulates Adrenal gland (located on kidney)
6. Adrenal gland releases **GLUCOCORTICOIDS (CORTISOL); use of body resources (carbs, fat, sugar, etc)**
Cortisol, Your Body and You

- Cortisol release = increased energy and alertness
  - Short-term benefit
- Continual/Chronic Cortisol release =
  - Impaired cognitive performance
  - Suppressed thyroid function
  - Blood sugar imbalances (e.g., hyperglycemia)
  - Decreased bone density
  - Decrease in muscle tissue
  - Higher blood pressure
  - Lowered immunity and inflammatory responses
    - Slowed wound healing
  - Increased abdominal fat
    - Increased risk for heart attack, stroke, and higher HDL cholesterol
Cortisol, Your Brain and You

- **Cortisol Release and Brain Systems**
  - Cortisol increases excitatory neurotransmission
  - Sympathetic Nervous System
    - Contributes to arousal
    - Exaggerated startle response
  - Limbic System
    - Hippocampus (strongest site of binding)
      - Involved in memory formation, retrieval
    - Anterior Cingulate
      - Initial emotional regulation
  - Frontal Lobe
    - Prefrontal Cortex & Orbitofrontal Cortex
      - Involved in short-term memory (organization)
      - Problem-solving
      - Executive functioning, decision-making
      - Automatic and controlled emotional-regulation
  - Probably no real solitary site of action – these are integrated brain systems
Review

Stress and hippocampal abnormalities in psychiatric disorders

M. Sala\textsuperscript{a}, J. Perez\textsuperscript{b}, P. Soloff\textsuperscript{c}, S. Ucelli di Nemi\textsuperscript{a}, E. Caverzasi\textsuperscript{a}, J.C. Soares\textsuperscript{d,e}, P. Brambilla\textsuperscript{b,}\textsuperscript{*}

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\textsuperscript{b}Biological Psychiatry Unit, IRCCS S. Giovanni di Dio, Fornengostrati, via Pilestroni 4, 25125 Brescia, Italy
\textsuperscript{c}Department of Psychiatry, Western Psychiatric Institute and Clinic, University of Pittsburgh School of Medicine, Pittsburgh, PA, USA
\textsuperscript{d}Division of Mood and Anxiety Disorders, Department of Psychiatry, University of Texas Health Science Center at San Antonio, San Antonio, TX, USA
\textsuperscript{e}South Texas Veterans Health Care System, Audie L. Murphy Division, San Antonio, TX, USA

Received 7 July 2003; received in revised form 16 December 2003; accepted 18 December 2003

Abstract

The hippocampus plays a main role in regulating stress response in humans, but is itself highly sensitive to neurotoxic effects of repeated stressful episodes. Hippocampal atrophy related to experimental stress has been reported in laboratory studies in animals. Several controlled brain imaging studies have also shown hippocampal abnormalities in psychiatric disorders, including posttraumatic stress disorder (PTSD), major depressive disorder (MDD), and borderline personality disorder (BPD). This paper reviews the physiological role of the hippocampus in stress circuitry and the effects of stress on cognitive functions mediated by the hippocampus. We also review brain imaging studies investigating hippocampus in PTSD, MDD, and BPD. This literature suggests that individuals with PTSD, MDD, and BPD may suffer hippocampal atrophy as a result of stresses associated with these disorders. Prospective, longitudinal studies will be needed in high-risk offspring and first-episode subjects to explore the relationship between stress and hippocampal atrophy in these neuropsychiatric illnesses.

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Keywords: Posttraumatic stress disorder; Borderline personality disorder; Depression; MRI; MRS; PET

1. Introduction

The hippocampus is an important brain region regulating stress responses in humans and a major feedback site for hippocampal atrophy, and resulting cognitive impairments associated with stress. The findings from brain imaging studies investigating the hippocampus in PTSD, MDD, and BPD, identified through a comprehensive Medline search...
Impact of Stress on Brain Structure and Function

- **Stress negatively affects brain physiology over time**
  - Decreased Hippocampal Volume
    - Occurs in chronic stress-related populations
    - MRI, MRS PET data
  - Decreased Anterior Cingulate Volume
    - Decreased N-acetylaspartate (NAA)
    - May pre-date hippocampal atrophy

- **Reduced Cognitive Flexibility and efficiency**
  - Decreased executive functioning, memory, visuospatial reasoning, and abstraction

- **Up-regulates autonomic tone**
  - Exaggerated startle – symptom **and** marker???
Smaller Hippocampal Volume in Dutch Police Officers with Posttraumatic Stress Disorder

Ramón J.L. Lindauer, Erik-Jan Vlieger, Margje Jalink, Miranda Olff, Ingrid V.E. Carlier, Charles B.L.M. Majoie, Gerard J. den Heetin, and Berthold P.R. Gersons

Background: Previous magnetic resonance imaging studies of posttraumatic stress disorder (PTSD) have reported smaller hippocampal volume, especially in war and sexual abuse victims. Our aim was to assess hippocampal volume in traumatized police officers with and without PTSD in the absence of alcohol abuse and moderate to severe major depression.

Methods: In a case-matched control study, 14 police officers with current PTSD and 14 traumatized police officers without lifetime PTSD were examined using magnetic resonance imaging. Three temporal lobe areas were manually segmented: hippocampus, amygdala, and parahippocampal gyrus. Volumetric analysis was used to measure gray matter, white matter, and cerebrospinal fluid. Results: After controlling for total brain volume, the hippocampal volume in the PTSD group was significantly smaller in comparison with the traumatized control group (total 10.6%; left 12.6%). Volumes of amygdala, parahippocampal gyrus, gray matter, white matter, and cerebrospinal fluid were not significantly altered. A significant negative correlation was found between reexperiencing symptoms and hippocampal volume in the PTSD group.

Conclusions: We confirmed previous findings of smaller hippocampal volume in PTSD in a new population made up of police officers, excluding comorbidity as a confounder. The finding of smaller hippocampal volume was specific to PTSD.

Key Words: Brain, hippocampus, magnetic resonance imaging, posttraumatic stress disorder, segmentation

Structural brain imaging studies in posttraumatic stress disorder (PTSD) have reported smaller hippocampal volumes in combat veterans (Bremner et al 1995; Gilbertson et al 2002; Gurvits et al 1996), in adults with PTSD secondary to child abuse (Bremner et al 1997), in female adult survivors of childhood sexual abuse (Stein et al 1997; Bremner et al 2003), and in adults with different kinds of trauma, but mostly childhood sexual abuse (Villarreal et al 2002). Reductions in hippocampal some limitations. First, populations were often victims of combat exposure or childhood sexual abuse (Bremner et al 1995, 1997; Fennema-Notestine et al 2002; Gurvits et al 1996; Schuff et al 2001; Stein et al 1997) with mostly chronic, unremitting forms of PTSD (Gilbertson et al 2002). To generalize the finding of smaller hippocampal volume, it is necessary to study also the hippocampal volume in other populations with a shorter duration of PTSD symptomatology. Second, comorbidity in the studies was high and included major depression, additional anxiety disorders, and substance disorders (Bremner et al 1995, 1997; De Bellis et al 1999, 2001, 2002; Schuff et al 2001; Stein et al 1997; Villarreal et
Statistical Analysis

Statistical analysis was performed with SPSS (Chicago, Illinois) 11.0 for Windows. Demographic and clinical variables were compared between PTSD subjects with and without major depression, compared with none of the traumatized control subjects. No significant changes were found for the hippocampal volume between PTSD subjects with and without major depression.

Table 2. Brain Volumes in Posttraumatic Stress Disorder Subjects and Traumatized Control Subjects (n = 28)

<table>
<thead>
<tr>
<th>Variable</th>
<th>PTSD (n = 14)</th>
<th>Control subjects (n = 14)</th>
<th>F(1)</th>
<th>p*</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Hippocampus (cc)</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Total</td>
<td>4.21</td>
<td>.48</td>
<td>4.71</td>
<td>.50</td>
</tr>
<tr>
<td>Right</td>
<td>2.18</td>
<td>.22</td>
<td>2.37</td>
<td>.30</td>
</tr>
<tr>
<td>Left</td>
<td>2.03</td>
<td>.28</td>
<td>2.34</td>
<td>.22</td>
</tr>
<tr>
<td>Amygdala (cc)</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Total</td>
<td>2.30</td>
<td>.47</td>
<td>2.55</td>
<td>.39</td>
</tr>
<tr>
<td>Right</td>
<td>1.13</td>
<td>.23</td>
<td>1.26</td>
<td>.23</td>
</tr>
<tr>
<td>Left</td>
<td>1.18</td>
<td>.27</td>
<td>1.29</td>
<td>.20</td>
</tr>
<tr>
<td>Parahippocampal Gyrus (cc)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>2.10</td>
<td>.22</td>
<td>2.05</td>
<td>.22</td>
</tr>
<tr>
<td>Right</td>
<td>1.04</td>
<td>.13</td>
<td>1.02</td>
<td>.12</td>
</tr>
<tr>
<td>Left</td>
<td>1.06</td>
<td>.17</td>
<td>1.03</td>
<td>.14</td>
</tr>
<tr>
<td>Gray Matter (cc)</td>
<td>598,063</td>
<td>42,350</td>
<td>601,778</td>
<td>41,042</td>
</tr>
<tr>
<td>White Matter (cc)</td>
<td>605,899</td>
<td>70,072</td>
<td>623,210</td>
<td>91,545</td>
</tr>
<tr>
<td>Cerebrospinal Fluid (cc)</td>
<td>186,669</td>
<td>30,618</td>
<td>175,270</td>
<td>21,871</td>
</tr>
<tr>
<td>Total Brain Volume (cc)</td>
<td>1,203,961</td>
<td>103,671</td>
<td>1,224,988</td>
<td>123,809</td>
</tr>
<tr>
<td>Gray Matter/ICV</td>
<td>.43</td>
<td>.023</td>
<td>.43</td>
<td>.022</td>
</tr>
<tr>
<td>White Matter/ICV</td>
<td>.44</td>
<td>.017</td>
<td>.44</td>
<td>.025</td>
</tr>
<tr>
<td>Cerebrospinal Fluid/ICV</td>
<td>.13</td>
<td>.015</td>
<td>.13</td>
<td>.013</td>
</tr>
</tbody>
</table>

SD, standard deviation; PTSD, posttraumatic stress disorder; ICV, intracranial volume.
*Multivariate analysis of covariance (MANCOVA) with total brain volume (TBV) as covariate.
**TBV differences were analyzed with an independent t test.

www.elsevier.com/locate/biopsych
Brief Report

*N*-Acetylaspartate Concentration in the Anterior Cingulate of Maltreated Children and Adolescents With PTSD

Michael D. De Bellis, M.D.
Matcheri S. Keshavan, M.D.
Steven Spencer, B.S.
Julie Hall, B.A.

**Objective:** Anterior cingulate dysfunction has been implicated in the pathophysiology of posttraumatic stress disorder (PTSD). The authors hypothesized that integrity of the anterior cingulate may be affected in childhood PTSD.

**Method:** Single voxel proton magnetic resonance spectroscopy (proton MRS) was used to measure the relative concentration of *N*-acetylaspartate and creatine, a marker of neural integrity, in the anterior cingulate of 11 children and adolescents who met DSM-IV criteria for PTSD secondary to maltreatment and 11 healthy matched comparison subjects.

**Results:** The ratio of *N*-acetylaspartate to creatine was significantly lower in the maltreated subjects with PTSD than in the comparison subjects.

**Conclusions:** The lower *N*-acetylaspartate/creatine ratio in subjects with PTSD suggests that anterior cingulate neuronal metabolism may be altered in childhood PTSD.


Symptoms of posttraumatic stress disorder (PTSD) represent pathological sequelae to traumatic experiences and may be thought of as conditioned fear responses to traumatic stimuli. The anterior cingulate cortex is involved in the extinction of conditioned fear responses and is implicated in the pathophysiology of PTSD (for review, see reference 1). Evidence for anterior cingulate dysfunction in adult PTSD comes from recent positron emission structural alterations that can be observed by using magnetic resonance imaging (6). Few studies have examined the in vivo neurochemistry of such neurobiological alterations. Magnetic resonance spectroscopy (MRS) is a safe means to measure neurobiological alterations in the brains of living children. The *N*-acetyl signal in the proton (1H) spectrum mainly comprises *N*-acetylaspartate and a small proportion of *N*-acetylaspartylglutamate. The total
Figure 1. Anterior cingulate N-acetylaspartate/creatinine ratios in 11 maltreated children and adolescents with PTSD and 11 healthy matched comparison subjects.

Method

Eleven subjects who met DSM-IV criteria for PTSD secondary to maltreatment and 11 healthy comparison subjects were recruited for single-voxel proton MRS studies of the anterior cingulate region. Subjects were case matched within 6 months of age (mean age=10.23 years [SD=2.8] for the PTSD group and mean= 10.15 years [SD=3.0] for the comparison group; age range=4.2-14.8 years) and by sex (six male pairs and five female pairs), race, Tanner stage, and Hollingshead four-factor index of socioeconomic status (8) (mean=38.0 [SD=10.0] for the PTSD group and mean=38.4 [SD=7.5] for the comparison group). Subjects were case matched within 8 points of full-scale IQ (mean=114.9 [SD=

Results
Brief Report

Neuropsychological Function in Children With Maltreatment-Related Posttraumatic Stress Disorder

Sue R. Beers, Ph.D.
Michael D. De Bellis, M.D., M.P.H.

Objective: Studies in adults have reported changes in concentration, learning, and memory in individuals with posttraumatic stress disorder (PTSD). However, there are few studies of cognitive function in children with PTSD. The goal of the current study was to evaluate cognition in children with PTSD.

Method: The cognitive status of 14 pediatric psychiatric outpatients with maltreatment-related PTSD and 15 sociodemographically similar children who were healthy and had not been maltreated was examined. Neuropsychological instruments measured language, attention, abstract reasoning/executive function, learning and memory, visual-spatial processing, and psychomotor function.

Results: The children with PTSD performed more poorly on measures of attention and abstract reasoning/executive function.

Conclusions: Although based on a small number of subjects, these results support cognitive differences between children with and without maltreatment-related PTSD.

(Am J Psychiatry 2002; 159:483–486)

Posttraumatic stress disorder (PTSD) is now widely recognized in children. Although findings are equivocal (1), studies of adults have reported cognitive problems in individuals with PTSD, particularly in the areas of concentration, learning, and memory (2). In contrast, cognitive function indexed by performance on standardized neuropsychological instruments has not been extensively evaluated in children with PTSD. It is particularly important to characterize the neuropsychological deficits associated with PTSD.

Method

We recruited 14 medication-naïve children with PTSD secondary to maltreatment who were psychiatric outpatients and 15 healthy comparison children who had not been maltreated and who were similar to the PTSD patients in age, race, socioeconomic status, and IQ. The mean age of the PTSD patients was 11.38 years (SD=2.60) and that of the comparison children was 12.17 (SD=1.75). Six of the PTSD patients were girls and eight were boys; seven of the comparison subjects were girls and eight were boys. In the PTSD group, 10 patients were white, two were African American, and two were biracial (white and African American); in
<table>
<thead>
<tr>
<th>Cognitive Domain and Measure</th>
<th>Comparison Children</th>
<th>Children With PTSD</th>
<th>Analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td><strong>Language</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clinical Evaluation of Language Fundamentals Concepts</td>
<td>10.85</td>
<td>3.65</td>
<td>10.83</td>
</tr>
<tr>
<td>and Directions</td>
<td>11.20</td>
<td>2.24</td>
<td>10.57</td>
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<tr>
<td>WISC-III Vocabulary</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Attention</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroop Color and Word Test</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Word</td>
<td>48.79</td>
<td>8.52</td>
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<tr>
<td>Color</td>
<td>45.86</td>
<td>8.73</td>
<td>42.10</td>
</tr>
<tr>
<td>Color/word</td>
<td>53.00</td>
<td>9.00</td>
<td>42.00</td>
</tr>
<tr>
<td>Interference</td>
<td>53.36</td>
<td>6.72</td>
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</tr>
<tr>
<td>Digit Vigilance Test</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Total time</td>
<td>490.13</td>
<td>149.82</td>
<td>490.36</td>
</tr>
<tr>
<td>Omission errors</td>
<td>6.87</td>
<td>4.55</td>
<td>17.36</td>
</tr>
<tr>
<td>WISC-III Digit Span</td>
<td>10.38</td>
<td>2.81</td>
<td>9.08</td>
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<tr>
<td><strong>Abstract reasoning/executive function</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
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<td>Wisconsin Card Sorting Test</td>
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<td>Categories</td>
<td>5.45</td>
<td>0.69</td>
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<tr>
<td>Perseverative responses</td>
<td>14.36</td>
<td>7.43</td>
<td>38.33</td>
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<td>Controlled Oral Word Association Test</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Animal Naming</td>
<td>20.00</td>
<td>4.04</td>
<td>14.11</td>
</tr>
<tr>
<td>Total Words</td>
<td>31.79</td>
<td>11.58</td>
<td>23.20</td>
</tr>
<tr>
<td>WISC-III Similarities</td>
<td>12.47</td>
<td>1.46</td>
<td>10.50</td>
</tr>
<tr>
<td>Trail Making B</td>
<td>31.60</td>
<td>18.25</td>
<td>30.40</td>
</tr>
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<td><strong>Learning and memory</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
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<td>California Verbal Learning Test</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>List A: Total Words, trials 1–5</td>
<td>54.40</td>
<td>8.41</td>
<td>49.38</td>
</tr>
<tr>
<td>List B</td>
<td>6.00</td>
<td>1.41</td>
<td>5.31</td>
</tr>
<tr>
<td>Short delay free recall</td>
<td>11.27</td>
<td>2.09</td>
<td>9.46</td>
</tr>
<tr>
<td>Long delay free recall</td>
<td>11.67</td>
<td>2.02</td>
<td>9.92</td>
</tr>
<tr>
<td>Discriminability</td>
<td>97.93</td>
<td>2.22</td>
<td>94.64</td>
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<td>Rey-Osterrieth Complex Figure recall</td>
<td>16.57</td>
<td>5.87</td>
<td>11.69</td>
</tr>
<tr>
<td><strong>Visual-spatial function</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rey-Osterrieth Complex Figure copy</td>
<td>31.20</td>
<td>4.26</td>
<td>22.93</td>
</tr>
<tr>
<td>Money Road Map</td>
<td>24.62</td>
<td>7.07</td>
<td>23.30</td>
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<tr>
<td><strong>WISC-III</strong></td>
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<td></td>
<td></td>
</tr>
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<td>Block Design</td>
<td>11.93</td>
<td>2.74</td>
<td>10.00</td>
</tr>
<tr>
<td>Object Assembly</td>
<td>11.07</td>
<td>2.81</td>
<td>10.50</td>
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<tr>
<td>Judgment of Line Orientation</td>
<td>22.60</td>
<td>5.67</td>
<td>17.71</td>
</tr>
<tr>
<td>Psychomotor speed</td>
<td>13.53</td>
<td>6.52</td>
<td>14.75</td>
</tr>
<tr>
<td>Trail Making A</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grooved Pegboard Test</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dominant hand</td>
<td>69.07</td>
<td>7.12</td>
<td>83.36</td>
</tr>
<tr>
<td>Nondominant hand</td>
<td>77.80</td>
<td>10.84</td>
<td>80.93</td>
</tr>
<tr>
<td>WISC-III Coding</td>
<td>10.31</td>
<td>4.13</td>
<td>9.46</td>
</tr>
</tbody>
</table>

*After Bonferroni correction, p < 0.01.
Exaggerated Acoustic Startle Reflex in Gulf War Veterans With Posttraumatic Stress Disorder

Charles A. Morgan III, M.D., Christian Grillon, Ph.D., Steven M. Southwick, M.D., Michael Davis, Ph.D., and Dennis S. Charney, M.D.

Objective: Exaggerated startle reflex is reputed to be one of the cardinal symptoms of post-traumatic stress disorder (PTSD). The goal of this study was to assess the magnitude of the acoustic startle reflex in Gulf War veterans with PTSD. Method: The eye-blink component of the startle reflex was measured in response to six blocks of pseudorandomized 40-msec white noise bursts of varying intensities (90, 96, 102, 108, and 114 dB) in 10 Gulf War veterans with PTSD, seven Gulf War veterans without PTSD, and 15 civilian subjects without PTSD. Results: The magnitude of the first startle response, as well as the magnitude of startle response averaged across blocks of testing, was significantly greater in Gulf War veterans with PTSD than in veteran and civilian comparison groups. Conclusions: Consistent with some clinical studies investigating the startle response in Vietnam veterans with PTSD, this investigation provides evidence for exaggerated startle response in this disorder. Preclinical studies of shock sensitization of the startle response suggest that the higher levels of startle response seen in the PTSD subjects may reflect a sensitization of the fear/alarm response created by the stress of combat trauma.


Accounts of psychopathology resulting from events such as exaggerated startle response, accompanying a
ACOUSTIC STARTLE REFLEX IN PTSD

FIGURE 1. Amplitude of the Acoustic Startle Reflex in Gulf War Veterans With PTSD, Gulf War Combat Comparison Subjects, and Healthy Civilian Comparison Subjects

- Civilian comparison subjects (N=15)
- Combat comparison subjects (N=7)
- Gulf War veterans with PTSD (N=10)

---

0.003), and significant group main effect (F=4.7, df=1, 23, p<0.04).

Subsequent post hoc tests indicated that the magnitude of the startle response was greater in the PTSD than in the civilian comparison group at 108 dB (F=7.3, df=1, 23, p<0.01) and 114 dB (F=6.9, df=1, 23, p<0.01). The magnitude of the startle response tended to be greater in the PTSD group at the 102-dB level of stimulation (F=3.5, df=1, 23, p<0.07). There were no significant differences at the 90- and 96-dB levels of stimulation. The magnitude of the startle response in the PTSD subjects was significantly greater than that in the combat comparison subjects at 96 dB (F=5.1, df=1, 15, p<0.03), 102 dB (F=9.8, df=1, 15, p<0.02), 108 dB (F=6.4, df=1, 15, p<0.02), and 114 dB (F=7.6, df=1, 15, p<0.01). The magnitude of the startle response tended to be greater in the PTSD group at 90 dB (F=3.6, df=1, 15, p<0.07).

Neither the group-by-block interaction nor the linear group-by-block interaction was significant, which suggests that the rate of habituation did not differ among groups. In addition, the group-by-intensity-by-block interaction was not significant.

DISCUSSION

To our knowledge, this is the first study to examine the startle response of Gulf War veterans with PTSD. Startle amplitude was greater in the PTSD patients...
Assessments - Cognitive

Selecting Assessments
- Should cover a variety of functions
  - Orientation
  - Attention
  - Verbal and Visual functioning
  - Comprehension/Abstract Reasoning/Problem-Solving
  - Motor functioning
  - Memory
  - Concurrent Psychiatric/Mood assessments also desirable

Advantageous if
- Norms/ranges or cutoffs available
  - (examine extent of difficulty/impairment)
- Repeated administration is permitted (e.g., minimal practice effects)
  - Monitor changes (e.g., decline, stability, improvement)
- Some insight into localization
  - Cortical (Frontal, Temporal, Parietal, Occipital)
  - Hemispheric
  - Cortical/Subcortical
Sample Battery

- **Example Battery**
  - Wechsler Test of Adult Reading
    - Premorbid Intellectual functioning
  - Wechsler Abbreviated Scale of Intelligence
    - Intellectual Screen
  - NCSE
    - Domains of deficits (e.g., memory, abstract reasoning, comprehension)
- **SLUMS**
  - Dementia
- **Trail-Making Test (Forms A+B)**
  - Processing speed
  - Complex attention/Simple Executive functioning
- **Verbal Fluency (Semantic and Phonemic)**
  - Language/Executive functioning
- **Complex Ideational Material (Boston Diagnostic Aphasia Examination)**
  - Comprehension/receptive aphasia
  - Suitability for testing
Assessment - Clinical

- **Assessment of DSM-IV TR criteria**
  - Adjustment Disorder
  - Acute Stress Disorder
  - PTSD

- Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I)
- Psychiatric Diagnostic Interview 4th Edition (PDI-IV)
- Diagnostic Interview for the DSM (DIM)
- Composite International Diagnostic Interview (CIDI)
- Schedule for Assessment of Affective Disorders and Schizophrenia (SADS)
  - Also “Kiddie” K-SADS
- Minnesota Multiphasic Personality Inventory, 2nd Edition (MMPI-2)
  - Restructured Form (RF) also available
  - PTSD subscales; anxiety and related subscales

- Also assessment of co-morbidity (e.g, Major Depressive Disorder)
National Center for PTSD

CLINICIAN-ADMINISTERED PTSD SCALE FOR DSM-IV

Name: ___________________________   ID #: ___________________________

Interviewer: ___________________________   Date: ___________________________

Study: ___________________________
<table>
<thead>
<tr>
<th>EVENT #1</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>What happened?</strong> (How old were you? Who else was involved? How many times did this happen? Life threat? Serious injury?)</td>
</tr>
<tr>
<td><strong>How did you respond emotionally?</strong> (Were you very anxious or frightened? Horrified? Helpless? How so? Were you stunned or in shock so that you didn't feel anything at all? What was that like? What did other people notice about your emotional response? What about after the event - how did you respond emotionally?)</td>
</tr>
<tr>
<td><strong>Describe (e.g., event type, victim, perpetrator, age, frequency):</strong></td>
</tr>
<tr>
<td>A. (1) Life threat? NO YES [self ___ other ___]</td>
</tr>
<tr>
<td>Serious injury? NO YES [self ___ other ___]</td>
</tr>
<tr>
<td>Threat to physical integrity? NO YES [self ___ other ___]</td>
</tr>
<tr>
<td>A. (2) Intense fear/help/horror? NO YES [during ___ after ___]</td>
</tr>
<tr>
<td>Criterion A met? NO PROBABLE YES</td>
</tr>
</tbody>
</table>
Criterion B. The traumatic event is persistently reexperienced in one (or more) of the following ways:

1. (B-1) recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions.
   **Note:** In young children, repetitive play may occur in which themes or aspects of the trauma are expressed.

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Intensity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Have you ever had unwanted memories of (EVENT)? What were they like? (What did you remember?) [IF NOT CLEAR:] (Did they ever occur while you were awake, or only in dreams?) [EXCLUDE IF MEMORIES OCCURRED ONLY DURING DREAMS] How often have you had these memories in the past month (week)?</td>
<td>How much distress or discomfort did these memories cause you? Were you able to put them out of your mind and think about something else? (How hard did you have to try?) How much did they interfere with your life?</td>
</tr>
<tr>
<td>0 Never</td>
<td>0 None</td>
</tr>
<tr>
<td>1 Once or twice</td>
<td>1 Mild, minimal distress or disruption of activities</td>
</tr>
<tr>
<td>2 Once or twice a week</td>
<td>2 Moderate, distress clearly present but still manageable, some disruption of activities</td>
</tr>
<tr>
<td>3 Several times a week</td>
<td>3 Severe, considerable distress, difficulty dismissing memories, marked disruption of activities</td>
</tr>
<tr>
<td>4 Daily or almost every day</td>
<td>4 Extreme, incapacitating distress, cannot dismiss memories, unable to continue activities</td>
</tr>
</tbody>
</table>

QV (specify)

<table>
<thead>
<tr>
<th>Past week</th>
<th>Past month</th>
<th>Lifetime</th>
</tr>
</thead>
<tbody>
<tr>
<td>F _____</td>
<td>F _____</td>
<td>F _____</td>
</tr>
<tr>
<td>I _____</td>
<td>I _____</td>
<td>I _____</td>
</tr>
<tr>
<td>Sx: Y N</td>
<td>Sx: Y N</td>
<td>Sx: Y N</td>
</tr>
</tbody>
</table>

2. (B-2) recurrent distressing dreams of the event. **Note:** In children, there may be frightening dreams without recognizable content.

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Intensity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Have you ever had unpleasant dreams about (EVENT)? Describe a typical dream. (What happens in them?) How often have you had these dreams in the past month (week)?</td>
<td>How much distress or discomfort did these dreams cause you? Did they ever wake you up? [IF YES:] (What happened when you woke up? How long did it take you to get back to sleep?) [LISTEN FOR REPORT OF ANXIOUS BEHAVIOR: YELING, ACTING OUT, ETC.]</td>
</tr>
<tr>
<td>0 Never</td>
<td>0 None</td>
</tr>
<tr>
<td>1 Once or twice</td>
<td>1 Mild, minimal distress or disruption of activities</td>
</tr>
<tr>
<td>2 Once or twice a week</td>
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</tr>
<tr>
<td>4 Daily or almost every day</td>
<td>4 Extreme, incapacitating distress, cannot dismiss memories, unable to continue activities</td>
</tr>
</tbody>
</table>
**Comorbid Depression**

- **Assessment of Depression**
  - Common comorbidity in anxiety disorders

- **Depression Screening Instruments**
  - Beck Depression Inventory (BDI)
  - Symptom Checklist-90 (SCL-90)
  - Children’s Depression Inventory (Depression subscale)
  - Zung Self-Assessment Depression Scale
  - Hamilton Rating Scale for Depression (HRDS; HAM-D)
  - General Health Questionnaire
  - Center for Epidemiologic Studies Depression (CES-D) Scale
Center for Epidemiological Studies Depression Scale (CES-D)

Instructions: I am going to read a list of ways you may have felt. Please tell me how often you have felt this way during the past week: rarely or none of the time; some or a little of the time; occasionally or a moderate amount of time; or most or all of the time.

<table>
<thead>
<tr>
<th>During the past week, that would be from ______ through ______ today:</th>
<th>Rarely or none of the time (less than 1 day)</th>
<th>Some or a little of the time (1-2 days)</th>
<th>Occasionally or a Moderate Amount of Time (3-4 days)</th>
<th>Most or all of the time (5-7 days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. You were bothered by things that usually don't bother you.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2. You did not feel like eating; your appetite was poor.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3. You felt that you could not shake off the blues even with help from your family or friends.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4. You felt that you were just as good as other people.</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>5. You had trouble keeping your mind on what you were doing.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6. You felt depressed.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7. You felt that everything you did was an effort.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>8. You felt hopeful about the future.</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>9. You thought your life had been a failure.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>10. You felt fearful.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>11. Your sleep was restless.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>12. You were happy.</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>13. You talked less than usual.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>14. You felt lonely.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>15. People were unfriendly.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>16. You enjoyed life.</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>17. You had crying spells.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>18. You felt sad.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>19. You felt that people disliked you.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>20. You could not get “going.”</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

To total: Add all circled numbers in each column

Total:
Center for Epidemiologic Studies Depression Scale (CES-D)/Monitoring Flow Sheet:

Patient Name_________________________ Phone_________________________ Patient ID_________________________

Currently on antidepressant? Yes___ No___ Antidepressant/Dose_____________________

Prior history of depression? Yes___ No___ Prior Antidepressant use? Yes___ No___

Antidepressant/Dose_____________________

<table>
<thead>
<tr>
<th>Month of Treatment</th>
<th>Date of Test</th>
<th>Your Initials</th>
<th>CES-D Score</th>
<th>Score ≥ 16? Yes/No</th>
<th>Intervention or Antidepressant/Dose</th>
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Center for Epidemiologic Studies Depression Scale (CES-D) Scoring
The CES-D consists of 20 questions. Patients are instructed to circle the number for each statement that best describes how often they felt or behaved this way during the past week. The score is the sum of the weights for the 20 items. The weight for each item corresponds to the number chosen for each (0-3), except for items 4, 8, 12, and 16, which are reversed (3-0). The possible range of scores for the scale is 0-60. The following cut-off scores best approximate the severity stages of depression: 0-9=none or minimal, 10-16=mild, 17-24=moderate, and ≥24=moderate to severe. Scores greater than 16 have been considered to reflect the need for further assessment and evaluation of the patient for depression.

Treatment

- **Treatment of Trauma-Related Disorders (Davidson, 2004)**

- **Pharmacotherapy**
  - Acute: Beta Blockers, corticosteroids, imipramine (TCA) high-potency benzodiazepines
  - Chronic: Selective Serotonin Reuptake Inhibitors (SSRIs); should avoid Norepinephrine Reuptake Inhibitors (NRIs)

- **Psychotherapy**
  - Cognitive-Behavioral Therapies: Stress Inoculation, Prolonged Exposure, Cognitive Therapy
  - Both Acute (< 3 months) and Chronic 3-12 months
  - Avoid Single-session Debriefing (negative outcomes noted)
  - Combination Pharmacotherapy and Psychotherapy Desired

- **Holistic Therapy**
  - Exercise, relaxation and health promotion desirable – Trauma has psychophysiological aspects
Davidson (2004)

Figure 1. Efficacy of Propranolol Treatment Within 6 Hours of Trauma Exposure in the Secondary Prevention of Acute Stress Disorder

![Graph showing efficacy of propranolol treatment](image)

no differences in traumatic memories between treatment groups, the rates of PTSD among patients who received hydrocortisone (11%; 1 of 9 patients) were significantly lower than in the placebo group (64%; 7 of 11 patients; p = .02). Given the limitations of this retrospective design, the finding of lower rates of PTSD in patients whose steroid levels were therapeutically manipulated during the period of trauma warrant further study.

A small pilot study of 25 children and adolescents aged 2 to 19 years with burn injury (mean 45% burn surface area) were randomized to a 7-day course of double-blind imipramine, 1 mg/kg or chloral hydrate. Severity of acute stress disorder was measured at baseline and again at 6-week follow-up using a structured interview. Rates of improvement in symptoms of acute stress were higher in the imipramine group (83%) than in the chloral hydrate group (38%; p < .02), suggesting a possible role for antidepressants in the prophylaxis of PTSD in pediatric patients with burn injury. Future studies would ideally assess the optimal antidepressant dose and consider drug-drug interactions and pharmacokinetic alterations associated with thermal injury.

Although benzodiazepines have not been shown to effectively treat established PTSD, they might have value in the prevention of PTSD in patients with symptoms of acute stress. In one study, a 1-month course of clonazepam or alprazolam was administered within 1 week of trauma exposure to 13 trauma victims. A group of 13...
found in 1 series of randomized, controlled studies to be ineffective 4 months after the trauma and again at 3-year follow-up. In fact, psychological debriefing resulted in markedly worse outcomes for patients who were experiencing intrusive and avoidance symptoms in the immediate posttrauma period than for control patients (Figure 2), and the investigators concluded that psychological debriefing was harmful and should not be used to prevent PTSD.

In contrast to single-session psychological debriefing, the literature supports the efficacy of brief courses of cognitive-behavioral interventions for emergent acute PTSD when administered for 9 months up to more than 1 year. Similarly, CBT has been found to maintain short-term treatment response for up to 12 months in some cases.

Long-Term Treatment With SSRIs

The efficacy of a 1-year course of paroxetine was studied as part of a neuroimaging trial designed to measure the
CONCLUSIONS

Short of avoiding exposure to traumatic events altogether, early and appropriate intervention in the immediate aftermath of trauma is considered a rational method for preventing the development of PTSD. Unfortunately, the secondary prevention of PTSD is not well studied. Although the prophylactic studies reviewed here of early propranolol, hydrocortisone, or imipramine administration are small and often not well controlled, their findings suggest that some sort of pharmacotherapeutic intervention could interrupt the natural course of PTSD. Further studies are clearly warranted. In sharp contrast, the results of 2 benzodiazepine studies in patients with symptoms of acute stress warn against extended use of these agents, at least as monotherapy, in the acute posttrauma period.

Psychotherapy is another component of treatment for symptoms of acute stress in trauma victims. Single-session psychological debriefing remains a controversial intervention that has been shown in well-designed trials to be ineffective and even deleterious relative to the development of chronic PTSD, although it may possibly be of benefit in other ways.28 However, brief courses of CBT shortly after exposure to trauma have been shown to prevent the development of PTSD 6 months later.29-34

Once PTSD is established, patients should be treated to full remission, which also should be the goal of long-
Additional Resources

- Trauma Resources for Clinicians and Patients
- Internet Resources
  - National Institute of Mental Health
  - National Alliance on Mental Illness
  - Dr. Baldwin’s Trauma Pages
- Community Resources
  - Johnson County Mental Health
  - Wyandotte County Mental Health
  - Tri-County Mental Health
  - Swope Health Services
  - Grief Support Network
  - KU Medical Center
  - Truman Medical Center
  - Western Missouri Mental Health Center
Questions and Discussion