TRAUMATIC STRESS AND POSTTRAUMATIC STRESS DISORDERS

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Part I — Lecture Outline: “An Introduction to Traumatic Stress: Recognition and Prevention”

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   2. Referral to psychiatrist for evaluation and treatment
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Part II — General Outline

1. Traumatic Stress (Trauma Response)

   A. Traumatic Events: Trauma has been a part of the human experience since the dawn of mankind. In the words of Billy Joel, “We didn’t start the fire. It was always burning since the world’s been turning.” War, devastation, famine and pestilence have occurred throughout human history. In today’s world, traumatogenic phenomena are ubiquitous. Some of the universally accepted traumatic stressors are:

   Accidents — Motor Vehicle, Other
   Disasters — Man-made, Natural and Technological
   Violence — Abuse(Physical, Sexual), Assault, Domestic Violence, Holocaust, Incest,
   Torture & Being Held Hostage, Rape
   War
   Witnessing someone being injured or killed

   B. Traumatic Stress (Trauma Response) Although there are literary descriptions dating back to ancient times about the effect on the psyche of traumatic events, the scientific study of the human response to trauma only spans the 20th century and part of the 19th. In the 1980’s the field of traumatology emerged as the investigation and the application of knowledge about the immediate and long-term psychosocial consequences of highly stressful events and the factors which effect these consequences. Within this field the validity of a response to trauma is universally accepted. A cardinal feature of this response is its biphasic nature which was first described by Janet (1889, 1909). Janet is also attributed with noting that dissociation is a key process in the reaction to overwhelming experiences and that traumatic memories are not synthesized but may be expressed as sensory perceptions, affect states or behavioral re-enactments. The trauma response is a dynamic concept in which there are alternating phases of intrusion (re-experiencing) and avoidance (psychic numbing) of the trauma. Freud (1939) observed, “The effects of trauma are of two kinds, positive and negative. The former are attempts to bring the trauma into operation once again - that is, to remember the forgotten experience, better still, to make it real, to experience a repetition of it anew...” [fixation to the trauma and compulsion to repeat] and “The negative reactions follow the opposite aim: that nothing of the forgotten trauma shall be remembered and nothing repeated.” [defensive reactions]. Abram Kardiner (1941) also noted these symptoms of repetition, including “repetitive tics and ceremonials,” and “inhibitory phenomena.” In addition, he focused on the somatic and autonomic symptoms. Thus, he coined the term “physioneurosis” in order to describe the fact that the response to trauma is physiological as well as psychological. For Kardiner, who emphasized an adaptive point of view, an external, sudden, overwhelming event causes an abrupt change in the person’s previous adaptation. Thus, the trauma response is a phenomenon of psychological and physiological adaptive changes necessary for mastery, restitution and the re-establishment of equilibrium. More recently, Horowitz (1978) has elaborated on the concept of the “stress response syndromes” and has developed multi-phasic models of a normal and a pathological stress response.

   The concept of a stress response derives from the work of Hans Selye who originated the study of stress. Building upon Claude Bernard’s concept of the internal milieu (all living beings maintain the constancy of their internal milieu) (1878) and Walter B. Cannon’s concept of homeostasis (the body’s tendency to maintain a steady state despite external change) (1929), Selye introduced the general adaptation syndrome (G.A.S.) (the manifestation of stress in the body which evolves in three stages - alarm reaction, stage of resistance, stage of exhaustion) (1936). Although the study of stress was a line of scientific investigation independent from the study of psychological trauma, it is interesting to note that Cannon’s work on the general function of the autonomic nervous system was derived from his research of traumatic shock (physical wounds) during World War I and much of what we know about psychological trauma comes from the study of war’s effect upon the psyche. Recently, there has been data demonstrating that the
hypothalamic-pituitary-adrenal axis functions differently in posttraumatic stress disorder compared to how it functions in stress. Therefore, one might argue that traumatic stress is either a different form of stress or not actually a stress disorder.

1. Characteristics of the Trauma Response (Traumatic Stress)

a. biphasic  
b. dynamic  
  re-experiencing  avoidance

c. positive features - re-experiencing; reliving; remembering; intrusiveness; repetitive thoughts, emotions, behaviors

d. negative features - avoidance, numbness, detachment, denial, amnesia, forgetting, repression

e. adaptive  
f. variety of emotions  
g. biopsychosociological components, e.g., physiologic - autonomic nervous system reactivity, importance of attribution and meaning, the response is modulated by social supports  
h. dissociative process

2. Functional Levels of the Trauma Response

normal psychodynamic process of the mind  natural reaction to a traumatic event  pathological state

C. Who has a pathological response? - If traumatic events are ubiquitous, why do we not all have a post-traumatic stress disorder? When more than one person is exposed to the same traumatic event, why do they not all develop PTSD? These questions lead to the stressor-stressee conundrum and endless debate. At one extreme is the view that any reaction to trauma is a normal response to an abnormal event; at the other extreme is the view that only individuals who are predisposed develop a disorder. Consistent with this latter hypothesis is a psychoanalytic theory that internal conflicts and fantasy are the determinants of pathology. Some argue that the disorder is due to stress; others argue that it is due to diathesis, i.e., constitutional predisposition. On the one hand, it is generally held that experiencing a traumatic event is a *sine qua non* in the causation of PTSD. Therefore, the nature of the traumatic stressor and the degree of exposure are variables in determining who develops PTSD. On the other hand, even when the traumatic event is very extreme not everyone who is exposed develops PTSD. Therefore, other variables appear to be necessary as co-variants with a traumatic event in order for PTSD or a pathological trauma response to develop. Hence, a stress-diathesis model (Zubin & Spring), i.e., an interaction between the environment and the individual, would explain the risk of developing PTSD. The variables (risk factors) that have been studied can be grouped under the categories of the features of the traumatic stressor, individual variables, and sociological variables. Using the categories of pre-traumatic, peri-traumatic and post-traumatic would be another way of classifying these risk factors. It should be noted that many of these variables require more study.

1. Features of the Traumatic Stressor

a. severity  
b. duration, degree of exposure  
c. proximity  
d. sudden onset & unexpected  
e. threat to life (Green *et al.*, 1990)
f. traumatic loss, bereavement

g. violence

h. exposure to the grotesque (Green et al, 1990)

i. physical injury

j. lack of uniformity of complex traumatic event for those involved in the event

2. Individual Variables

a. cognitive appraisal — process of cognitive interpretation, i.e., the meanings we assign to the world as it unfolds before us ("in the eye of the beholder"; one person's fear is another person's challenge)

b. attribution — the process by which one ascribes motives to their own or others' behavior, e.g., answering the question "Why me?" with self-blame — characterological self-blame (esteem-related, focuses on one's enduring qualities) vs. behavioral self-blame (control-related, commissions or omissions that effected outcome), e.g., "I never do anything right." vs. "I should not have gone alone."

c. resilience (Flach, 1977)

d. risk factors for developing PTSD - see ILA.3.c

e. genetic factors (True et al, 1983)

f. previous trauma

g. intelligence

h. peri-traumatic dissociation (Koopman et al, 1994; Shalev et al, 1995)

3. Sociological Variables

a. social support during the traumatic stressor

b. social support during recovery

II. Post-Traumatic Stress Disorders: PTSD and Acute Stress Disorder (ASD)

A. PTSD

1. Definition: A mental disorder in which there is the development of characteristic symptoms — persistent re-experiencing, avoidance and numbing, increased arousal — following exposure to an extreme traumatic stressor during which the person experiences intense fear, helplessness or horror. The symptoms must be present for more than one month and cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

2. Historical

a. Civil War (1861-65)
   "Irritable Heart" — 1871 — Jacob Da Costa, M.D. — Da Costa’s Syndrome — Civil War veterans — palpitations, chest pain, tachycardia, shortness of breath, tremulousness, sweats, fatigue, dizziness, phobias, nightmares, headache, parathesias, digestive disorders, nervousness — pathophysiology: disturbance of sympathetic nervous system — similar to but independent from descriptions from Crimean War (1853-56)

b. Railroad Accidents
   1) "Compensation Neurosis" — 1879 — Rigler
   2) "Railway Spine" — 1882 — Erichsen ["Erichsen’s Disease" — 1889 — Clevenger]
   3) "Nervous Shock" — 1885 — Page
   4) "Traumatic Neurosis" — 1889 — H. Oppenheim
5) summary: controversy over whether claims of injuries were malingering (1 above), a valid clinical entity with organic basis (2.) or a valid clinical entity which is psychological in origin (3)

c. WWI (1914-18)
2) “Shell Shock” – 1917 – Mott – due to physical brain lesions caused by carbon monoxide or changes in atmospheric pressure secondary to exploding shells
3) “Soldier’s Heart and the Effort Syndrome” – 1919 – Lewis – chest pain, palpitations, tachycardia, fatigue
4) “Neurocirculatory Asthenia” – 1918 – Oppenheimer
5) “Shell Concussion” -- 1940 -- Meyers -- psychical causes
6) “Traumatic (War) Neurosis -- “physioneurosis” -- 1941 -- Kardiner

d. WWII (1939-45)
1) “Combat Stress” -- 1945 -- Grinker & J. Speigel
2) “War Stress” -- 1947 – Kardiner & H. Speigel
3) other -- “Battle Fatigue,” “Combat Fatigue”

e. DSM
1) I – “Gross Stress Reaction” -- 1952 -- transient reaction in a “normal” person to deal with overwhelming fear after exposure to severe physical demands or extreme emotional stress (combat or civilian catastrophe)
2) II – “Adjustment Reaction of Adult Life” -- 1968 -- reduced to a minor status

f. Vietnam War (1964-75) and Other
1) “Survivor Syndrome -- 1968 -- Niederland -- survivors of the Holocaust
2) “Rape Trauma Syndrome” -- 1974 -- Burgess & Holmstrom
3) “Post-Vietnam Syndrome,” “Delayed Stress Syndrome,” “Postcombat Syndrome” -- “Combat Stress Reaction” -- sociopolitical movement of the Vietnam era brought about recognition, Congressional legislation and funding of research and treatment programs

g. DSM
1) III – “Post-traumatic Stress Disorder” -- 1980 – a mental disorder caused by a traumatic event became firmly established – Post-traumatic Stress Disorder became official name for this disorder.
2) III - R – 1987 – PTSD -- changes in diagnostic criteria were made: a) “Outside the range of usual human experience” was added to modify the stressor criterion (Criterion A). b) Intense psychological distress was added as a sub-criterion to the re-experiencing cluster of symptoms (Criterion B). c) The avoidance/numbing cluster of symptoms (Criterion C) was expanded from three to seven sub-criteria. d) The third cluster of symptoms (Criterion D) which was not labeled was labeled as “increased arousal” symptoms. e) Survivor guilt was deleted as a sub-criterion. f) The sub-criterion of “irritability or outbursts of anger” was added to the arousal symptom cluster. g) The sub-criteria of memory impairment and avoidance of activities were removed from the 3rd symptom cluster (Criteria D) and placed in the avoidance/numbing symptom cluster. h) “Hyperalertness or exaggerated startle response was separated into two sub-criteria in the arousal symptom cluster and hyperalertness was changed to hypervigilance. i) The temporal criterion of one month duration of symptoms was added (Criterion E). j) Subtypes were changed from acute and chronic or delayed to only delayed onset. k) The minimum number of sub-criteria of the avoidance/numbing symptom cluster required for the diagnosis was changed from one to three.

3) IV – 1994 – PTSD – Major Changes: a) stressor criterion (Criterion A) – Subjective
criteria was added to the objective criteria and “outside the range of usual human experience” was eliminated as a modifier of the traumatic stressor. b) The clinical significant criterion (Criterion F) was added. c) Minor Changes: (1) The physiological reactivity sub-criterion was moved from the arousal symptom cluster (Criterion C) to the re-experiencing symptom cluster (Criterion B). (2)) The specifiers Acute and Chronic were reintroduced and redefined.

“Acute Stress Disorder” (ASD), a new diagnostic entity, was added. (See II.B. Acute Stress Disorder.)

h. Lessons from History
1) dialectic of collective recognition vs. denial of the horror of trauma and its effects, e.g., the horror of war is forgotten until the next major war when the traumatic disorder is rediscovered and given a new name
2) bias that PTSD is not a valid clinical entity but rather malingering
3) dichotomies of organic vs. functional, physical vs. psychical cause, biological vs. psychosociological are giving way to integrative, systems and multi-variant models

3. Epidemiology

a. prevalence — depends upon population at risk
1) National Vietnam Readjustment Study (Kukla et al, 1990) men 15.2% women 8.5%; lifetime: men 30.6% women 26.9%
2) urban young adults *
   lifetime prevalence = 9.2%
3) Buffalo Creek -- (Green et al, 1990) -- 14 year follow-up 25% met criteria for PTSD
4) National Institute of Mental Health -- Epidemiological Catchment Area Study
   (NIMH-ECA) general population lifetime prevalence: St. Louis 1% (Helzer et al, 1987),
   North Carolina 1.3% (Davidson & Fairbank, 1993)
5) Rape Victims -- 70% (Bownes et al, 1991)
6) NIMH Comorbidty Study – Lifetime Prevalence: 7.8% ( female 10.4%, male 5%  
   (Kessler et al, 1995)

b. risk of exposure to a traumatic event — urban young adults — low education, male, early conduct problems, extroversion, family history of mental illness/substance problems *

c. risk factors for developing PTSD — female sex, neuroticism, family history of instability and deviance, early separation from parents, pre-existing anxiety or depression, family history of anxiety *

*(Breslau et al, 1991)

4. Phenomenology

a. DSM-IV Criteria

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 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

b. TRAUMA Mnemonic

T r au ma (Criterion A. - Stressor Criteria)
R eexperiencing (Criterion B. - Symptom Cluster) need 1 of 5 sub-criteria
A r ousal (Criterion D. - Symptom Cluster) need 2 of 5 sub-criteria
U nable (Criterion F. - Clinically Significant Distress or Impairment)
M onth (Criterion E. - Temporal)
A voidance (Criterion C. - Symptom Cluster) need 3 of 7 sub-criteria
c. Course — onset any age; duration/prognosis: approximately 25% spontaneously remit within one year but 40% persist beyond six years (NIMH Comorbidity Study - Kessler et al, 1995)

d. Differential Diagnosis

1) Natural Trauma Response vs. Acute Stress Disorder or PTSD
2) Other Disorders Developing after a Traumatic Stressor
   a) Adjustment Disorder
   b) Anxiety Disorder
   c) Brief Psychotic Disorder
   d) Conversion Disorder
   e) Dissociative Disorder
   f) Exacerbation of a preexisting mental disorder
   g) Major Depressive Disorder
   h) Panic Disorder with or without agoraphobia
   i) Phobia
3) Co-morbid Disorders
   a) Dissociative Disorder
   b) Major Depression
   c) Panic Disorder with or without agoraphobia
   d) Psychoactive Substance Use Disorders
4) Trauma: Physical and/or Psychic
   a) Traumatic Brain Injury (TBI) (Mental Disorder Due to a General Medical Condition – Head Trauma)
   b) Postconcussional Syndrome (PCS) (Posttraumatic Syndrome) — a condition resulting from head trauma consisting of headache, dizziness, fatigue, mood changes, attention and memory difficulties — note: because of similar terms this is often confused with PTSD
   TBI or PCS may be present instead of ASD or PTSD or may be co-morbid with ASD or PTSD
5) Mutually Exclusive Criteria
   a) Adjustment Disorder (if symptoms of PTSD are present but the stressor is not extreme, i.e., not life-threatening, no threat of or actual injury, e.g., being fired, marital separation)
   b) Malingering
   c) Normal Bereavement or Pathological Grief
   d) Obsessive-Compulsive Disorder (recurrent intrusive thoughts are not related to trauma)
   e) Psychosis (Schizophrenia, Psychotic Disorder due to General Medical Condition, Major Depression with Psychotic Features, delirium, Substance-induced disorder, Other Psychotic Disorders) — flashbacks, hallucinations, illusions of PTSD must be distinguished from the perceptual disturbances in these disorders
6) Disorders Associated with Childhood Trauma
   a) Borderline Personality Disorder
   b) Disorder of Extreme Stress Not Otherwise Specified (DESNOS) — not a DSM diagnosis
   c) Eating Disorders
   d) Multiple Personality Disorder

5. Psychobiology and Biopsychosociological Models

a. Data — Biological Alterations
   1) heightened sympathetic activity
   2) excessive adrenergic activity
   3) serotonergic abnormalities
   4) increased dopaminergic activity
5) GABA/benzodiazepine receptor changes
6) exaggerated startle response
7) provocation of panic attacks and flashbacks
8) opioid system dysregulation
9) HPA (hypothalamic-pituitary-adrenal) axis abnormalities
10) elevated thyroid function
11) suppression of immune response
12) reduced hippocampal volume
13) increased blood flow right-sided limbic, paralimbic, visual areas; decreased blood flow in left inferior frontal and middle temporal cortex [PET & Script-Driven Imagery]

b. Theories

1) Biological
   a) Septohippocampal (Gray, 1992) novel, unpleasant environmental cues => SH system =>
      response (inhibition of motor system, increased arousal, hypervigilance)
   b) Locus Ceruleus LC = central control mechanism of sympathetic nervous system (SNS)
      responds to threatening stimuli
   c) REM Sleep Disturbance (Ross et al, 1989)

2) Psychological
   a) Psychoanalytic (Freud, 1920) Economic Model traumatic stimuli => breakthrough of stimulus
      barrier => increased libidinal excitation => repetition-compulsion (reexperiencing) =>
      decrease of excess energy (mastery)
   b) Behavioral
      1) Classical Conditioning (Pavlov, 1927) repeated aversive stimuli/environmental threat
         [unconditioned stimuli (UCS)] + cues associated with the trauma [Conditioned Stimulus
         (CS)] => defensive reaction [Conditioned Response] (CR), hence CS => CR
      2) Operant Conditioning: avoidance behavior reduces anxiety and fear => reinforcement
         of avoidance behavior
   c) Cognitive Reprocessing (Horowitz, 1978) traumatic event => incongruence with internal
      schemata => active memory (reexperiencing) vs. completion tendency + information
      processing => integration of traumatic event and schemata (self and world images)

3) Biopsychological
   a) Physioneurosis - (Kardiner, 1941) see LB.
   b) Inescapable Shock (Van der Kolk et al, 1985) based on learned helplessness (Seligman et al,
      1968) inescapable shock (trauma) => transient catecholamine depletion + endogenous
      opioid release (numbing) => stress induced analgesia, hence re-exposure =>
      paradoxical calm alternates with cessation of stimuli => opioid withdrawal +
      noradrenergic hypersensitivity => physiological hyperactivity [repetition + mastery]
   c) Neuropsychological (Kolb, 1987) excessive emotional stimulation => stimulus overload =>
      perceptual discrimination & cortical defensive structural processes cannot process information
      => sensitization of neurophysiology => synaptic change or if chronic high intensity stimuli
      => depression of synaptic processes & of habituating learning => impaired cortical
      control of limbic structures => aggressive expression + sleep-dream cycle disturbance
   d) Kindling (Post, 1984, 1992) stressor => triggers 1st episode => stressor sensitization + episode
      sensitization => subsequent spontaneous episodes Sensitization encoded on level of gene
      expression: stressor => neurotransmitter => 2nd messenger => induction of
      transcription factors => mRNA induction => protein synthesis [neuropeptides] => alteration
      of, e.g., CRH (corticotropin-releasing hormone)
   e) Stressor-Induced Oscillation (Antelman et al) intermittent exposure to stressor =>
      neurochemical & behavioral oscillation or bi-directional cycling

4) Sociological
   a) cultural variants, e.g. survivor guilt
b) traumatic environment
c) recovery environment

c. Psychological Themes: fear of repetition, fear of merger with victim, shame and rage over vulnerability, rage at the source, rage at those exempted, fear of lose of control of aggressive impulses, guilt or shame over aggressive impulses, guilt or shame over surviving, sadness over losses. (Horowitz, 1986)

6. Treatment

a. Treatment Guidelines

The Expert Consensus Guideline Series Treatment of Posttraumatic Stress Disorder
Editors: Foa, E.B., Davidson, J.R.T and Frances, A
J. of Clinical Psychiatry, 1999 Supplement 60:16

Effective Treatments for PTSD Practice Guidelines from the International Society for Traumatic Stress Studies (ISTSS)
Editors: Foa, E.B., Keane, T. M. and Friedman, M. J.
The Guilford Press, New York, 2000

b. Consensus Guidelines

Acute PTSD (1-3 months duration: mild: start psychotherapy
moderate-severe: start combination (psychotherapy and psychopharmacotherapy)
Chronic (> 3 months): start combination (opinion of psychopharmacologists)
start psychotherapy if necessary add medication (psychotherapists)

Comorbidity: use combination

Psychopharmacotherapy: first line – selective serotonin reuptake inhibitor (SSRI), Zoloft (sertraline) FDA approved December 1999; Paxil (paroxetine) FDA final approval pending

Psychotherapy: Psychoeducation and Individual Psychotherapy: Cognitive Therapy, Behavior Therapy (Prolonged Exposure) Anxiety Management if necessary

c. Other

1). family/group — adjunctive
2). hypnosis — caveat regarding use for recovered memories
3). Eye Movement Desensitization and Reprocessing (EMDR) (F. Shapiro, 1989) —
ISTSS = found to be an effective treatment but use of alternating movements
including eye movements not proven

4) psychodynamic
5) in-patient treatment
6) psychosocial rehabilitation
7). Thought Field Therapy (TFT), Traumatic Incident Reduction (TR), Time-Limited Therapy (T-LIT), Visual/Kinesthetic Dissociation (V/KD) — all not proven

B. Acute Stress Disorder

1. Definition: The development of characteristic anxiety, dissociative and other symptoms that occur within 1 month after exposure to an extreme traumatic stressor.
2. Phenomenology

DSM-IV Criteria

A. Same as PTSD
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 effect of a substance (e.g., a drug of abuse, medication) or a general medical condition, in not better accounted for by a Brief Psychotic Disorder, and is not merely an exacerbation of a preexisting Axis I or Axis II disorder.

[The underlying is mine.]

III. Prevention

Major role for all physicians but especially important for primary care physicians

A. Primary Prevention

1. safety
2. reduction of violence and accidents
3. preparedness for disasters
4. diagnosis and treatment of psychoactive substance use disorders
5. diagnosis and treatment of sleep disorders
6. early detection and intervention of abuse (domestic violence, child abuse)
7. suicide prevention

B. Secondary Prevention

1. early detection of trauma-response
2. emphasis on natural response (accident, disaster, rape victims; rescue workers, physicians and other health care workers)
3. restoring safety
4. emotional support
5. group experience
6. critical incident stress management (CISM) and debriefing (CISD) (J. Mitchell, 1983)
7. social/cultural rituals
8. counseling
9. identifying populations at risk, e.g., individuals with pre-existing mental illness

C. Tertiary Prevention

1. early detection of pathological trauma-response - - ASD, PTSD - - Other Disorders
2. referral to psychiatrist for evaluation and treatment
Traumatic Stress and Posttraumatic Stress Disorder Recognition and Prevention

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PTSD: Biological Data

- Heightened sympathetic activity
- Excessive adrenergic activity
- Serotonergic abnormalities
- Exaggerated startle response
- Provocation of panic attacks and flashbacks
- Opioid system dysregulation
- HPA axis abnormalities
- Elevated thyroid function
- Reduced hippocampal volume
- Script driven imagery PET scan changes

Traumatic Stress and PTSD Educational Objectives

At the conclusion of this introduction to this topic, you should be able to recognize the manifestations of traumatic stress, diagnose PTSD, be respectful to those who suffer from psychic trauma, and know how to prevent traumatic stress and PTSD.

1. Why is it important for you to be knowledgeable about the impact of traumatic events on human beings?
2. How does one recognize traumatic stress and PTSD?
3. What is your role in the prevention of traumatic stress and PTSD?

Traumatic Stress and PTSD Topics

1. Diagnostic criteria
2. Protective/Risk Factors
3. Clinical case
4. Prevention

Trauma Mnemonic

<table>
<thead>
<tr>
<th>TRAUMATIC STRESSOR</th>
<th>A</th>
</tr>
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<tbody>
<tr>
<td>REEXPERIENCING</td>
<td>1 OF 5</td>
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<tr>
<td>AROUSAL</td>
<td>2 OF 5</td>
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<tr>
<td>UNABLE</td>
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<td>MONTH</td>
<td>E</td>
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<tr>
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</tr>
</tbody>
</table>

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PTSD: DSM-IV Criteria - 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
Lifetime Prevalence of Trauma Experience

Differential Diagnosis

Normal trauma response or acute stress disorder
Other disorders after a traumatic stressor
Trauma: physical and/or psychic
Co-morbid disorders
Mutually exclusive criteria
Disorders associated with childhood trauma

Traumatic Stress Screening Question

"Was there ever a time when you thought you were about to die or your life was threatened or you came close to death?"

Lawrence C. Kolb, M.D.

Trauma Response Characteristics

- Biphasic
- Dynamic
- Positive/negative features
- Adaptive
- Variety of emotions
- Biopsychosociological components
- Dissociative process

Functional Level of the Traumatic Stress Response

Prevalence of PTSD

<table>
<thead>
<tr>
<th>STUDY</th>
<th>LIFETIME PREVALENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breslau et al, 1991</td>
<td>9.2%</td>
</tr>
<tr>
<td>NIMH-ECA Davidson &amp; Fairbank, 1993</td>
<td>1.3%</td>
</tr>
<tr>
<td>Helzer et al, 1987</td>
<td>1.0%</td>
</tr>
<tr>
<td>NIMH-Comorbidity Kessler et al, 1995</td>
<td>10.4% M 5%</td>
</tr>
<tr>
<td>Resnick et al, 1993</td>
<td>F 12.3%</td>
</tr>
</tbody>
</table>
Problems

- Why doesn’t everyone who is exposed to a traumatic event develop PTSD?
- Why do some individuals develop the symptoms of PTSD after exposure to a life stressor or low magnitude event?

Variables Related to the Development of PTSD

- Traumatic Stressor
  - Definition
  - Features
- Individual variables
  - Pre-traumatic
  - Peri-traumatic
- Environmental variables
  - Pre, peri and post-traumatic

Disaster Studies: Post Disaster Body Handlers

<table>
<thead>
<tr>
<th>Investigator</th>
<th>Disaster</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Taylor et al</td>
<td>N.E. Disaster</td>
<td>Initial trauma? Problems 20% of 300 of N.E. fol, follow-up of stress</td>
</tr>
<tr>
<td>Jones</td>
<td>Boston Bombing, 1993</td>
<td>Short term symptoms, 7% of 565, &lt; 36 to 6 months, + exposure</td>
</tr>
<tr>
<td>McConaghy et al</td>
<td>Close Proximity, 1993</td>
<td>Pre-disaster, CA: Experience + Anticipation, AA</td>
</tr>
<tr>
<td>JTS, 1993</td>
<td>Pre-disaster</td>
<td>Anxiety: Exposure to companions + Anticipation, AA</td>
</tr>
<tr>
<td>Sisano et al</td>
<td>Various Disasters, 1990</td>
<td>Anticipatory anxiety + gastrointestinal symptoms of exposure, emotional, EPA + victim, personal threat to handler</td>
</tr>
<tr>
<td>Sisano et al</td>
<td>Various Disasters, 1990</td>
<td>Vicarious exposure, lack of support, stress, + exposure, emotional, EPA + victim, personal threat to handler</td>
</tr>
<tr>
<td>Sisano et al</td>
<td>USA War, 1990</td>
<td>Exposure, 1990</td>
</tr>
</tbody>
</table>

Protective / Risk Factors

- Features of the Traumatic Stressor
  - Severity
  - Degree of exposure
  - Proximity
  - Sudden onset
  - Threat to life
  - Traumatic loss
  - Violence/atrocities
  - Exposure to the grotesque
  - Physical injury
  - Lack of uniformity

Individual Variables: Genetic Factors

- Monozygotic versus dizygotic
- Total twin pairs = 4,042
- Vietnam veterans
- Controlled for combat exposure
- Variance due to genetic factors
  - 13-30% Reexperiencing symptoms
  - 30-34% Avoidance symptoms
  - 28-32% Arousal symptoms

True et al, 1993

Protective / Risk Factors

- Individual Variables
  - Attribution
  - Cognitive appraisal
  - Genetic factors
  - Experience
  - Intelligence
  - Peri-traumatic dissociation
  - Personality
  - Previous trauma
  - Resilience
  - Other factors
Protective / Risk Factors

- Environmental Variables
  - Early life environment
  - Social support during the traumatic event
  - Social support after the traumatic event

Lessons from History

- Dialectic: collective recognition versus denial
- Bias: not a valid clinical entity
- Dichotomies are replaced by integrative, systems and multi-variant models

Top 10-Causes of Disability and Premature Mortality Worldwide

<table>
<thead>
<tr>
<th>Rank</th>
<th>Disease or Injury</th>
<th>Disability-Adjusted Life Years*</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Major Depression</td>
<td>15,664</td>
<td>27,461</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Tuberculosis</td>
<td>16,361</td>
<td>8,542</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Road Traffic Accidents</td>
<td>13,096</td>
<td>7,605</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Alcohol Use</td>
<td>11,040</td>
<td>7,895</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Self-inflicted Injuries</td>
<td>10,957</td>
<td>4,453</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Bipolar Disorder</td>
<td>7,899</td>
<td>6,415</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>War</td>
<td>7,800</td>
<td>6,800</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Violence</td>
<td>6,746</td>
<td>5,805</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Schizophrenia</td>
<td>6,446</td>
<td>6,297</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>Iron-Deficiency Anemia</td>
<td>6,094</td>
<td>6,125</td>
<td></td>
</tr>
</tbody>
</table>

* (in thousands) used to quantify the burden of disease in terms of premature mortality and living with disability for people aged 15-64 in 1990

Prevention: Primary

- Safety
- Reduction of violence and accidents
- Preparedness for disasters
- Diagnosis and treatment of psychoactive substance use disorders
- Diagnosis and treatment of sleep disorders
- Detection and Intervention of abuse
- Suicide prevention

Detection: Abuse of Woman

- 22-35% of women seeking care for any reason in emergency departments
- 19-30% of injured women seen in emergency departments
- 25% of women utilizing psychiatric emergency service
- 25% of women who attempt suicide
- 23% of pregnant women seeking prenatal care
- 14% of women seen in internal medicine clinics

AMA Diagnostic and Treatment Guidelines on Domestic Violence

Prevention: Secondary

- Early detection of trauma response
- Emphasis on natural response (accident, disaster and rape victims, rescue workers, physicians, others)
- Restore safety
- Emotional support
- Group experience
- Critical incident stress debriefing
- Social and cultural rituals
- Identifying populations at risk
Prevention: Tertiary

- Early detection of pathological traumatic stress (ASD, PTSD, other disorders)
- Referral to psychiatrist for further assessment and for treatment

Chronicity of PTSD

PTSD Treatment: Effect Sizes

<table>
<thead>
<tr>
<th>Treatment Type</th>
<th>Effect Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychological</td>
<td>.82</td>
</tr>
<tr>
<td>Pharmacological</td>
<td>.41</td>
</tr>
<tr>
<td>SSRIs</td>
<td>.77</td>
</tr>
<tr>
<td>MAO-I</td>
<td>.39</td>
</tr>
<tr>
<td>TCAs</td>
<td>.32</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td>.25</td>
</tr>
</tbody>
</table>

Panagia, S.J. et al, Depress Anxiety, 4:240, 1994-97