POSTTRAUMATIC STRESS DISORDER: DIAGNOSIS, CLINICAL COURSE, AND CONCEPTUAL OVERVIEW

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HISTORICAL PERSPECTIVE

A. Shakespeare recognized the unique effect of trauma on memory: Out, Out damn spot...a flashback!!! And then a request for treatment.

> Canst thou not minister to a mind diseased,
> Pluck from the memory a rooted sorrow,
> Raze out the written troubles of the brain,
> And with some sweet oblivious antidote
> Cleanse the fraught bosom of that perilous stuff
> Which weighs upon the heart?
>
> --Macbeth to the Doctor of Physic requesting treatment for Lady Macbeth

B. Janet—Justine and flashbacks of seeing cholera victims as a child.

C. Vietnam and the development of DSM-III

D. TV dramatizations: MASH and ER

DSM-IV CRITERIA

POSTTRAUMATIC STRESS DISORDER

A. The person has been exposed to a traumatic event in which both of the following were present:
   (1) actual or threatened death or serious injury, or a threat to the physical integrity of self or others
   (2) response included fear, helplessness, or horror

B. The traumatic event is persistently reexperienced in at least on of the following ways:
   (1) intrusive recollections
   (2) recurrent dreams
   (3) acting or feeling as if the event were recurring

(4) psychological distress with exposure to cues of the event
(5) physiological reactivity to cues of the event

C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three of the following:
(1) avoidance of thoughts feelings, or conversation related to the trauma
(2) avoidance of people, places, or things that arouse recollections of the event
(3) inability to recall an important aspect of the trauma
(4) diminished interest in activities
(5) detachment form others
(6) restricted affect
(7) sense of foreshortened future

D. Persistent symptoms of increased arousal as indicated by at least two of the following:
(1) difficulty falling or staying asleep
(2) irritability or anger outbursts
(3) difficulty concentrating
(4) hypervigilance
(5) exaggerated startle response

E. Duration is more than one month

F. Distress or impairment in social, occupational, or other important areas of life function

Specify if acute (symptoms less than 3 months); chronic (greater than 3 months).

Delayed onset: at least 6 months after the stressor

ACUTE STRESS DISORDER
Added to DSM-IV to describe a limited response and possibly predict PTSD; DSM I—"Gross Stress Reaction"—immediate response following an extreme stressor—no longer term PTSD dx.

A. Event same as in PTSD

B. Three of the dissociative symptoms during or after the event:
(1) numbing, detachment, or absence of awareness
(2) reduction in the awareness of surroundings
(3) derealization
(4) depersonalization

(5) dissociative amnesia
These set it apart from PTSD and are problematic.

C. Persistent re-experiencing as in PTSD.

D. Avoidance of event or related stimuli

E. Anxiety or increased arousal

F. Significant impairment

G. Lasts 2 days to four weeks and occurs within 4 weeks of the event.

Unclear if the criteria are too stringent: dissociation may not be a good predictor of PTSD although it may be a risk factor for chronic PTSD. (Marshall, AJP, 11/99). People with ASD have higher levels of hypnotizability (somewhat tautological) (Bryant, AJP, 4/01).

NATURE OF STRESSOR

A. Changed from DSM-IIIR from “outside the range of normal human experience” to life threatening.
Clinical and forensic implications

B. Common stressors
Combat
Criminal Assault
Rape
Accidental Injury
Hostage/POW
Natural or manmade disasters
Witnessing assault, homicide of injury
Sexual abuse of child

C. Dose-response relationship
Development of symptoms linked to intensity of exposure
Veterans, natural disasters, intensity of assault
Critical threshold for single exposures

D. Subjective Response
Perceived threat or suffering
Low controllability

E. Rape as a particularly potent event

PREDISPOSING FACTORS FOR PTSD

A. Inability to "debrief" the trauma--veterans, disaster victims; unsupportive environment in abused children prevents debriefing. (Data on disaster debriefing is conflicting). Deahl's data (Abstract in PTSD Research Quarterly, 12/01) debriefing UN peace keepers found an impact on ETOH use.

B. Extent of the dissociation during and immediately after the trauma, more dissociation increases the risk for PTSD. (Shalev, AJP, 2/96—study of physical injury victims in an ER—25% with PTSD at followup—predicted by dissociation, anxiety, depression, and intrusion at 1 week).

C. Higher rates of childhood physical abuse in PTSD Vets seeking treatment which is confirmed in civilians exposed to assault.

D. Genetic predisposition in twin studies (True, AGP,1993)

E. Family history of psychopathology and PTSD

F. Women greater than men

G. Physiologic Response—greater at the time of the event correlates with increased PTSD risk.

H. ASD, early symptoms of PTSD are predictive: (Brewin, AJP, 3/99, assault victims: 19% had ASD, 20% PTSD; number of sx and full ASD diagnosis were predictive of PTSD at 6 months.) (Koren, AJP 3/99, Traffic accident victims-32% PTSD at 1 yr; PTSD at one year was predicted by higher symptoms immediately after the event, compared to no PTSD, and PTSD group had steady sx increase over first three months; PTSD sx immediately after accident were was better predictor than the severity of injury; premorbid psychopathology a risk factor)

COURSE OF POSTTRAUMATIC STRESS DISORDER

A. Posttraumatic stress symptoms (PTSS) that not disorder very common and fade shortly. Early CBT intervention may be helpful. (Bryant, AJP, 11/99)

B. PTSD itself is often chronic--presence of PTSD at three months predicts chronicity
POWs--47% at 40 years for W.W.II

Vietnam--15% with PTSD at 19 years
Rape--65% one month; 45% nine months; 40% three years
Childhood sexual abuse--50-90% during first year
in 2/6 studies. 4/6 studies found no PTSD

C. Breslau data (Breslau J Clin Psych Supp. 217 2001): Those with PTSD dx—82% at 3 months, 74% at 6 months, 50% at 1 year, 30% at 10 years (Kessler, Arch Gen Psych 12/95); Risks for chronicity included higher number of symptoms, higher rates of numbing and hyperreactivity, affective disorders, and co-existing medical conditions.

D. Intensity of trauma related to duration but not an absolute or unique factor

E. Memory deficits in Veterans and POWs; Neurologic soft signs in men(combat vets) and women (Abused as children)with chronic PTSD—also had lower IQs and more childhood ADHD symptoms compared to control groups with abuse but no PTSD: cause or effect?? (Gurvits, AGP,2/00)

F. Comorbidity—affective, substance abuse, poor role function; Sub threshold PTSD carries similar risk and suicidal ideation—(Marshall AJP 9/01); increase in somatic symptoms and poor role functioning (Breslau)

RESPONSES to WAR and DISASTERS

A. Responses to different types of Disasters (McMillen, JTS, 1/00).

1) Natural disasters—low rates 4-8%--property damage but less exposure to violent loss of life

2) Man-made disasters—technological and criminal disaster which resulted in multiple fatalities 29-54%

3) Higher rates in combat, POWs, Rwanda (no PTSD dx but high scores on the IES—intrusion and avoidance), and rape as above.

4) Children studied in Isreal after SCUD attacks: stress symptoms decreased in residentially stable children but increased in displaced children; younger children's responses were more closely tied to mother's response than older children, specifically mother's level of symptoms related to child's; family cohesion predictive of outcome. At 30 months 8% of children had severe PTSD sx. (Laor, AJP, 7/01).

B. Are PTSD responses normal??

1) Yehuda reviewed this question and concludes that those who meet the criteria for PTSD have high incidence of prior psychopathology, abnormal cortisol response, high comorbidity (50-90%). Development of PTSD is seen only in a

minority of people, making exposure not the only salient factor. (Yehuda, AJP 12/95)

2) High rates of B and D criteria fulfillment without C—Hurricane Hugo 85% "B" and 42% D, only 6% "C": thus Only 5% with PTSD. Study of 130 survivors 3 months post 1/94 Northridge, CA, earthquake: 13% had PTSD but 48% had "B" and "D" symptoms. Premorbid psychopathology predicted "C" and PTSD criteria but not "B" and "D". Suggests many suffer non-diagnostic symptoms, but also may recover more easily b/c they lack the withdrawal/numbing sx. (McMllen, JTS, 1/00)

SECONDARY POST TRAUMATIC STRESS DISORDER

A. Trauma Workers at Risk—National Organization for Victim Assistance

1) Immediate Responders (Police, firefighters, rescue workers)
2) Later responders (medical, emergency, paramedic, ambulance personnel)
3) Unexpected responders (passersby)
4) Body recovery, identification, and burial personnel
6) Crisis interveners (clergy, mental health and counseling workers)
7) Volunteers (Red Cross)
8) Remote responders (equipment maintenance)
9) Emergency support personnel (dispatchers, information services, telephone operators)

B. Selected Studies and Risk Factors

1) Personal factors—individual's history and makeup, organizational and community structures. (Eriksson, JTS, 1/01): 113 aid workers—10% with full PTSD and 19% 2/3 PTSD sx; overall 50% had sx in at least one cluster. Exposure directly or vicariously were strongest predictors.
2) Event factors—scope, duration, intensity relationship to trauma, identification with victim (Ursano, AJP, 3/99). Occupational exposure: (Wagner, AJP, 12/98): 318 German firefighters—18% with PTSD—risk factors were exent of exposure, number of distressing missions; PTSD associated with depression, somatic complaints, substance, and social dysfunction.
3) Stress Appraisal Factors—threat perception, service disruption.
4)Acute event: Uncued recollections soon after the event were associated with increased risk of intrusive thoughts and avoidance at 6-12 months in workers after an airline crash (Schooler, JTS, 10/99)

C. Secondary Traumatic Stress Disorder (Compassion Fatigue, Secondary Traumatization, Secondary Victimization, Burnout)

1) STSD—Exposure is as witness or through contact with traumatized victims resulting in development of re-experiencing, numbing/avoidance, and persistent arousal. Mechanisms include empathic bonding, sense of threat to self, revulsion and fear through contact with trauma, emotional contagion.

2)Burnout—a progressive response resulting from cumulative work exposure. Overlaps with STSD. Symptoms are physical (fatigue, somatization, illness) emotional(anxiety depression, irritability, guilt, helpless/hopeless), behavioral (anger, withdrawal, substance abuse) work related (diminished performance, absenteeism/lateness, leaving job) and interpersonal (loss of empathy, numbness and negativism).

D. Interventions

Multiple approaches have been designed incorporating common factors in either group or individual interventions:

1) Review of events and experiences
2) Emotional ventilation
3) Empathy/support from leader/therapist and other members (if group)
4) Psycho-educational phase—preparing for responses and normalizing them; discussion of self management and situation management strategies.
5) Closure/Assessment of needs—reviewing the intervention in positive frame, preparing for further intervention if indicated.

Interventions may be short term or long term depending on the nature of exposure (crisis v. ongoing), the individuals involved, and the system/organizational factors.

CONCEPTUAL MODELS AND NEUROBIOLOGY

A. Learning models

(1) Fear Conditioning--Conditioned stimulus is paired with an unconditioned stimulus to evoke the conditioned fear response and subsequently the CS provokes response without the UCS. Amygdala, locus ceruleus, thalamus, and hippocampus.

(2) Extinction--failure of extinction suggests the power of the learning trial (one trial learning)--animal models

show that extinction may not necessarily erase the learning but modify it or mask it. Sensory cortex and amygdala.

(3) Sensitization--overall increased arousal to stimuli and substances. Nucleus accumbens, striatum, hypothalamus, and amygdala.

(4) "The body keeps the score," van der Kolk--the amygdala keeps the score: Charney.

B. Learned Helplessness

(1) No perceived control
(2) Passivity: No task involvement
(3) Disruption of basic life style
(4) Withdrawal from social supports
(5) Mood: depressed

Treatment involves reversal of these patterns

C. Biochemical systems

(1) Opiates--numbing and pain reduction veterans and survivors survivors. "Addiction" to abuse or trauma hypothesis not well-established. Possible role of naloxone.

(2) Dopamine system--hypervigilance and paranoia.

(3) Noradrenergic--panic, anxiety, startle responses. Use of benzodiazapines, alcohol, opiates.

(4) **Corticosteroids**--exaggerated cortisol responses to stress but lower baseline levels (Yehuda). Appear to have hypersensitive negative feedback. Cortisol responses in PTSD are not an exaggeration of normal. Possible effects on learning and hippocampal function. (Other studies by Putnam in teenage victims found elevated cortisol levels but these may not be comparable to chronic PTSD).

D. Adaptive defensive reactions

(1) Defensive responses: freeze, flight, or fight
(2) Circa-strike defenses: analgesia, emotional numbing, startle-explosive
(3) Post-strike behavior: pain, recuperation

DIAGNOSTIC APPROACH

A. Thorough psychosocial and psychiatric evaluation
Affective and substance abuse

B. Trauma history

C. PTSD and Dissociative symptom review—CAPS, IES, Dissociative Experiences Scale and SCID-D, TSI.

TREATMENT STRATEGIES (Dissociative Disorders)

Levels of Care

I. Inpatient treatment
   A. Acute decompensation, usually affective with suicidality/self-destructive behavior
   B. Substance abuse.
   C. Triggers: acute trauma, rediscovery of trauma, contact with perpetrator or change related to perpetrator, legal entanglements, re-exposure to relevant cues which are inescapable
   D. Development or presence of more serious dissociative condition, esp. considering data re: predisposing factors

II. Partial Hospital/IOP
   Suitable when patient not in danger but needs significant support because of recurring symptoms and inability to fulfill role demands

III. Outpatient therapy
   Suitable when able to resume some of role demands

Modalities

I. Individual Therapy
   A. Re-establish basic safety
   B. Affect management: relaxation, diary keeping
   C. Address cognitive distortions
   D. Develop narrative
   E. Affect processing
   F. Reintegration of experience; reintegration in role demands

II. Group therapy
   A. Psychoeducational overview
   B. Principles to re-establish safety
   C. Affect management techniques,
   D. Identification of cognitive distortions,
   E. Skills development--avoid premature rechallenge and exposure
   F. Avoid narrative work or processing beyond basic outline
   G. Self-help for substance abuse but not for PTSD **per se,** Victims' Services

Evidenced Based Treatments for PTSD

A. Classification Scheme for studies of PTSD (Foa, 10/00 ISTSS Journal)

1) A=Randomized, well-controlled studies
2) B=Well-designed studies without randomization or placebo.
3) C=Naturalistic studies

4) D=Clinical practice not subjected to empirical study
5) E=Clinical practice by a small group of clinicians
6) F=Recently develop Rx without empirical basis

B. Review of Psychotherapies

1) CBT: Exposure: "A" Generation of narrative and repeated rehearsals. Best data of the CBT studies.
2) CBT: Systematic Desensitization: B/C
3) CBT: Stress Inoculation Training: "A"
4) Psychological debriefing: Prevents future PTSD "B"; Positively received by recipients "A"; lowered alcohol use in soldiers returning from Bosnia (Deahl, JTS, 7/01)
5) EMDR: "A/B" Effective in several controlled studies compared to wait list or routine but not compared to other PTSD treatments. The only comparison against PTSD specific CBT found CBT/EMDR equal in symptom reduction but not as well tolerated and CBT had longer lasting effects. Further, studies of EMDR without eye movements compared to with found **no difference,** suggesting that EM may not be the critical factor. (See PTSD Research Quarterly 12/99)
6)Imagery Rehearsal for Nightmares—controlled trial reduced nightmares and decreased PTSD scores; (Krakow, JAMA, 10/1/01)

C. Review of Pharmacotherapies

1) SSRIS's: "A" Sertraline; "B" for fluoxetine, paroxetine, fluvoxamine. Global improvement and reduction of all symptoms groups (although the studies vary).
2) MAOIs: "A/B" Good for cluster B symptoms.
3) Benzos: "B" Good for arousal symptoms.
4) Anticonvulsants: "B" Different drugs show variable effect on each of the three symptom clusters.

Details of Pharmacotherapy Review: No "magic bullet"

A. Antidepressants SSRI’s, TCAs, SNRI, TZD, NEF may be helpful in decreasing activation and avoidance/depressive symptoms; some intrusive symptoms may respond as well; CBT or exposure still more powerful for re-experiencing.
B. Mood stabilizers: anticonvulsants—-positive results for both arousal and intrusive symptoms
C. Benzodiazepines used carefully for hyperarousal; 2-4 mg clonazepam may be helpful
D. Clonidine reduces intrusive symptoms; propranolol only in open trials for flashbacks
E. Neuroleptics
F. Correct sleep with trazadone or low potency neuroleptics

Zoloft study—Brady, et al JAMA 2000

12 week placebo-controlled study of 187 outpatient with a primary dx of PTSD of at least 6 month duration (mean was 10 years) with the usual exclusions for active substance abuse, past psychosis, and suicidality. 60% were victims of physical or sexual assault. Sertraline given as indicated with mean dose of 133 mg/day. Results showed global improvement in total CAPS score, CGI, HAM-D, and Davidson PTSD scales compared to placebo but IES score did not reach significance (more weighted toward intrusive symptoms). CAPS avoidance and arousal improved but not intrusion. Improved on all measures of function. 53% responders v. 32%with placebo.

Zoloft Study—Davidson Arch Gen Psych 5/01

12 weeks placebo controlled 100 subjects, 108 placebo, ^ month PTSD on CAPS. 60% victims of physical or sexual assault. Mean dose 146 mg. Equal numbers of active v. placebo subjects discontinued treatment due to s/e's. 60% responders v. 38% for placebo. Improvement in CAPS, IES, CGI, Davidson but not in HAM-D,A, nor sleep measures (speculated to take longer than 12 weeks). All symptom clusters improved as well.

Paxil study—Marshall, et al J Clin Psychophar 2/98

Open label 12 wk study of 13 completers. PTSD primary. Utilized only Davidson and IES (self reports). Improvement in all symptom areas with hyperarousal and avoidance improving by 4 weeks but intrusive sx not improving until 8 weeks.

SUGGESTED READINGS

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