

Traumatic Stress-An Overview of Diagnostic Evolution and Clinical Treatment?

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Abstract

This paper provides an overview of recent advances in the study of traumatic stress, with a special focus on the categorical and conceptual evolution in aetiological, diagnostic classification of post traumatic stress disorder (PTSD). A review update on psychological and pharmacological therapies relevant to the management of PTSD, and the trend towards a multi-layered, biopsychosocial approach is discussed. This review also includes an update of the risk factors and mediating factors associated with post traumatic responses.

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Introduction

Traumatic events are daily occurrences, despite our wishes. They can be classified by the nature of traumatic event (human-caused or natural), the effect of trauma on the individuals and groups, and the long-term responses to the event. The past decades have documented numerous heart wrenching traumatic events in Asia since the end of World War II. Events such as the Korean War, Vietnam War, Persian Gulf War, Tien An Men Square uprising, Tokyo Subway Sarin gas attack, political or ethnic cleansing and genocide in Sri Lanka and Cambodia, and communist insurgencies, have left many in South East Asia, especially those who went through the Second World War, with a constant reminder of the emotional and physical trauma the world can produce. These, coupled with the numerous natural disasters happening in the region (e.g. typhoons, hurricanes, earthquake and forest fires), serve as a reminder of our continued vulnerability to tragic events. With the rapid industrialization of the region, technical and industrial accidents have also become more common as local traumatic events.

The effects of trauma and disasters on individuals, communities and nations are also of substantial and increasing concern in our present economic-societal structure, which has evolved from the 1960s “baby

boom” era to the single-child or two-child nuclear family of today, which some have called the “precious baby” era. With a level of national security and street safety that our forefathers have not enjoyed before, the younger Singapore generation is being nurtured in an environment that some have mistakenly depicted as “sterile” of violence.

However, international terrorism and hostage-taking events, such as the 1992 hijacking of a Singapore Airlines plane by terrorists, can still happen and do. Industrial accidents resulting from human error recur despite stringent industrial safety regulations. Incidents such as the Cable Car Accident, the Collapse of Hotel New World, the “Spiros Explosion” (explosion of a ship under repair in a shipyard) are not unfamiliar news to all of us. Disasters do strike. Natural disasters like earthquakes and typhoons may seem remote to most Singaporeans, but we will probably not forget that our island is sited just outside the “fringe” of the volcanic and earthquake strip of Sumatra. We are reminded of this proximity each time we experience a tremor along the east coast of Singapore. Local environmental accidents seem insignificant when we think along the dimension of the Alaska Exxon oil spillage and the Kuwait oil field burning during the Gulf War, as most oil spillage in our waters usually has been efficiently managed and cleaned by the authorities before the news was

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even broadcast. But a major environmental disaster, like the 1994 seasonal Sumatra forest fire, which resulted in the regional haze entrapment, is an undeniable reminder to us of our closeness and vulnerability to disaster.

What is Trauma Psychiatry?

Trauma, as defined in the Webster's dictionary, is a wound inflicted by physical injury or emotional stress. As it suggests, Trauma Psychiatry denotes the clinical subspecialty branch of psychiatry that studies, and manages mental, emotional and behavioural disorders that result from a traumatic event, usually a disaster. It is far more than the study of post traumatic stress disorder (PTSD), but includes other Axis I and II disorders such as Major Depression, Acute Stress Disorder (ASD), Substance Abuse, Dissociative Disorder, Brief Psychotic Disorder with marked stressor(s).¹

With millions of lives affected by the many adverse traumatic events, it is not surprising that post traumatic stress syndrome has arrested the attention of the medical, behavioural sciences and legal specialists, and resulted in the active research and study of this age-old problem of mankind. This convergence of interest on post traumatic stress syndrome by the fields of neuroscience, experimental psychology, clinical psychiatry and sociology has resulted in the exploration of new areas of the impact of traumatic events, attempting to understand the intricacy of psychobiological processes that control reaction patterns, clinical symptom manifestations, and aspects of post trauma coping and adaptation.²

Prevalence and Incidence of PTSD

Many studies have examined the prevalence of PTSD in the general community, and in both military and non-military at-risk populations. In a review by Davidson et al,³ the lifetime prevalence of PTSD among American adults was reported to range from 1% to 1.3%, 0.5% in men and 1.3% in women.⁴ Following exposure to trauma, Breslau et al⁵ reported the rate of PTSD among a group of young adults was as high as 23.6%, yielding a lifetime prevalence of 9.2%, and they were also at risk for other psychiatric disorders. A recent study by Kessler et al⁶ based on a National Comorbidity Survey reported that post traumatic stress disorder is more prevalent in the United States than previously believed, with a lifetime prevalence of 7.8% in the total sample.

The incidence of PTSD in Vietnam combat veterans reported by Helzer⁴ was 6.3%, while for those who were wounded the rate was 20%. In perhaps the best designed study of PTSD in a military population, Kulka et al⁷ found current PTSD in 15.2% of male and 8.5% of female Vietnam veterans. The lifetime prevalence rates in these groups were 30.9% for males and 26.9% for

females. The rates were also higher in those with physical injury and with higher intensity of war zone stress. Preliminary studies on Persian Gulf War veterans have shown a low rate of PTSD immediately upon return,⁸ and 9% after the first year of returning.⁹

Traumatic Stress Syndromes-Historical Evolution of PTSD Diagnostic Criteria-Towards DSM IV

The study of the contribution of trauma to psychiatric disorders has waxed and waned over the past centuries. Attempts to establish the aetiological connection between war and post traumatic stress reaction were made as early as the sixth century. Subsequent acknowledgments of the impact of traumatic response to major disasters (large fires, train accidents) were documented in the seventeenth century. However, the psychological-trauma relationship phenomenon was not given a consistent attention until extensive work on trauma and memory were done by Pierre Janet, a student of Charcot in Paris in the early nineteenth century.¹⁰ Substantial interest throughout history has focused on what we now recognize as three primary symptom areas that occur after a traumatic event: intrusion/ unwarranted thoughts of the trauma; avoidance of reminders of the trauma often accompanied with emotional numbing; and hyperarousal.

Pierre Janet: Trauma and Memory

In the study of trauma, Pierre Janet was particularly interested in the memory changes. He observed that while different temperaments exert great influence on the way people dealt and coped with the stresses in life, previous life experiences stored in the form of a memory structure also affected their coping styles. He considered the encoding and retrieval of memories being one of the key organizing functions of the mind, while the storage and categorization of incoming sensations into a matrix for proper integration of subsequent internal and external stimuli the basic function of the memory system. Janet believed that the successful categorization and integration of an event experienced into an existing collection of memories will allow the person to develop and add on to a series of subconscious, flexible mental schemes, that will in turn guide and prepare him to deal with subsequent challenges. He believed that it is the dynamic interplay of the memory system and temperament that determines the uniqueness and complexity of how a person responds to his environment.¹⁰

From the study of a group of patients, Janet suggested that traumatized people become "attached" to the trauma, i.e. unable to integrate traumatic memories. They lose the capacity to assimilate subsequent new experiences. Janet postulated that these traumatized patients were now unable to attend to both their internal processes and make adaptive use of their perceptions.

It was “as if” the evolution of their lives and their personalities were checked.

Janet proposed that these memory disturbances were largely due to the need to avoid thoughts about the traumatic event (phobia of “traumatic” memory) caused by the event that precipitated the emotion. This prevented the integration of traumatic events, and split the traumatic memory from ordinary consciousness. This dissociation state, together with the constriction of affect could persist in the traumatized subject as a way of coping with subsequent stresses in life. Contemporary studies concurred that memory processes the *quality* of experience and the *feelings* associated with it, and less on the vegetative aspect of the events.¹⁰ Holloway and Ursano¹¹ described traumatic memory recall as a metaphor to communicate the experiential qualities of the present life situation as well as recall the past. Current memory research work is an important part of understanding traumatic stress.

Janet noted that the initial emotional response to a traumatic event also determined the intensity of symptoms. He held that hyperarousal was responsible for the memory disturbances that accompanied traumatization. He believed it was the trauma associated physiological response that sustained this “emergency” response seen with subsequent stress.

Freud's Concept of Trauma and Neuroses, Hysteria

Freud's original view of neuroses was based on a post traumatic paradigm. Freud stated that there was a range of traumatic experiences and events which happened during childhood that could be profoundly distressing to the individual. As a result of the degree of threat experienced to the ego and the subsequent anxiety, the victim typically employed repression, forgetting, as an ego defense. Once repression was employed, various neurotic symptoms and behaviours would appear.”

However, Freud later shifted his emphasis away from a post traumatic theory to an instinct-driven Oedipal model which focus on intra-psychic mechanism. This shift in paradigm led to the formulation that the effect of traumatic events on self-structure was acute and transient in nature. If there was a prolonged reaction to trauma, this was usually attributed to the premorbid traits and psychodynamics of the individuals.

Nevertheless, Freud had clearly described several core PTSD symptom clusters which were incorporated in Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised, American Psychiatric Association (DSM III-R) criteria for Post Traumatic Stress Disorder. In their book, Freud and Breuer¹³ stated that “the traumatic experiences is constantly forcing itself upon the patient, and this is proof of the strength of the experiences; the patient is fixated on his trauma”. He noted that traumatic neuroses patients regularly repeat

the traumatic situation in their dreams (intrusive imagery phenomena), “as if they had not finished with the traumatic situation” (actively reliving the events), and showed physiological hyperactivity (hyperarousal).

Freud deduced that in both traumatic and war neuroses, when the psychic apparatus is overwhelmed, repression is employed as the central psychological defense. “The compulsion to repeat the trauma”, Freud believed was “itself a function of repression”, and as such, the patient was not able to “remember the repressed materials as something of the past, but as a contemporary experiences”.

Both Freud and Janet believed that the crucial factor in the repetition of trauma was unintegrated experiences. The “speaking out of one's emotional experience in language form” provides an important structure facilitating the translation of “emotional memory” into “narrative memory”, allowing the re-organization and integration of the memory schema.¹⁰ This theoretical framework continues as an important foundation of treatment of trauma victims using debriefing.

In Between the Wars

The First World War witnessed many acute responses to the battle stress resulting in many battle casualties, who were later labelled as Shell Shock. This term was attributed to the psychological disturbances that resulted from the concussion effects of artillery shells. But soon it was learned that great majority of the “shell-shocked” soldiers were the victims of transient stress-induced psychological disorders, and the name “war neurosis” was introduced. Thomas Salmon, the American Surgeon General, formulated a forward treatment policy employing the principles of proximity, immediacy, and expectancy, which has evolved into the current concept of forward combat casualty management [Proximity, Immediacy, Expectancy, Simplicity (PIES)].

A review of psychiatric literatures between the two world wars revealed few publications on trauma, but important works were reported.¹⁰ A notable paper by Ferenczi,¹⁴ studied child, victims of interpersonal violence. He observed that these children were helpless and terrified, and the predominant psychological defense employed was identification with the aggressor.

An important contribution during the Second World War was from Abram Kardiner, an American analyst who not only studied traumatic neurosis of war, but also drew attention to the study of traumatic neurosis in general, among non-war accident victims and rail road workers. Kardiner emphasized among the criteria for traumatic neurosis, the nature of the stressor, the *subjective meaning* of the trauma, which overwhelmed the host's defense. He described the symptoms of fixation on the trauma (with altered conception of self and of the

outer world), atypical dream life, contraction of general level of functioning, irritability and tendency to explosive aggressive reactions.¹⁵ During the Second World War, Kardiner's work was incorporated into field applications for the management of combat neuroses by American psychiatrists. Their observations soon confirmed Kardiner's earlier report of the persistence of conditioned biologic responses in traumatized patients. It was found that these patients remembered the somatosensory aspects of the traumatic experience in an altered state of consciousness. Hypnosis and other abreactive techniques were used, but the effects were often not lasting. It became apparent that "combat left a lasting impression on men's minds, changing them as radically as any critical experience through which they live, unlike the writing on the slate which could be cleaned and removed without leaving a trace".¹⁶ As a result, group psychotherapy was introduced and used in the treatment of traumatized combat soldiers.

Important progress in the management of combat neuroses was made through the studies of Vietnam war veterans in the seventies. These veterans presented initially with predominantly high incidence of alcoholism and somatization. The linkage of their problems with war trauma lead to the intensification of the treatment processes. Rap groups originated and were popular among the war veterans. These provided a unique group structure for the veterans to work through their own problems without being labelled as a psychiatric patient.¹⁰ Later studies in the 1970s of rape victims produced the description of rape trauma syndrome, with flashbacks and nightmares resembling the traumatic neurosis of war.¹⁷

Stress Response Syndrome-Horowitz's Model

Horowitz* made an important contribution to the study of responses to stress in the seventies. He identified and summarized 8 clinical themes reported by his patients: fear of repetition of the stressful event, shame over helplessness or emptiness, rage at the source of the stress, guilt or shame over aggressive impulses, fear of becoming aggressive, survivor guilt, fear of identification or merger with the victims, and sadness over loss. He called it the "Stress Response Syndrome". This provided an organized framework for the therapist to suggest appropriate strategies in the intervention and treatment of trauma victims.

Towards a Diagnostic Model in DSM IV: PTSD, Acute Stress Disorder

Post Traumatic Stress Disorder

During the span of 12 years which saw the conclusion of the Vietnam war, many returning veterans presented with clusters of emotional, physiological and behavioural disturbances associated with combat trauma.

The "Vietnam era" generated extensive studies in post-combat trauma which yielded greater understanding of the psychopathology of war trauma. Together with important contributions from civilian disaster studies, a specific and separate diagnostic entity, "Post Traumatic Stress Disorder" (PTSD), was created under the Anxiety Disorder group.

The fourth edition of Diagnostic and Statistical Manual (DSM IV, 1994)¹⁹ published by the American Psychiatric Association (APA), maintained the core triad of PTSD symptom clusters, i.e. (1) intrusive, re-experiencing the trauma event, B criteria; (2) avoidance responses to evidence of the trauma or generalized psychological numbing and isolation, C criteria; and (3) widespread physiological arousal not previously present, D criteria (Table I).

Post traumatic stress disorder was one of the few disorders that stated its aetiology as a prerequisite to the diagnosis. It required the "existence of a recognizable stressor that would evoke significant symptoms of

Table I: Diagnostic Criteria for Post Traumatic Stress Disorder*

- A. Exposure to a traumatic event involving actual or threatened death or serious injury, or threat to integrity of self or others, accompanied by intense emotional response of fear, helplessness or terror.
- B. Symptoms of re-experiencing the event: (one of the following)
 1. Recurrent and intrusive distressing recollections
 2. Recurrent distressing dreams of the event
 3. Acting or feeling the event recurring (flashbacks)
 4. Psychological distress at exposure
 5. Physiological reactivity on exposure to cues reminiscent of event
- C. Persistent avoidance of trauma associated stimuli or numbing of responsiveness (three or more of the following)
 1. Efforts to avoid trauma related thoughts, feelings or conversations
 2. Efforts to avoid activities, places or persons
 3. inability to recall significant aspect of memory (psychogenic amnesia)
 4. Diminished interest or participation in significant activities
 5. Detachment or estrangement
 6. Restricted range of affect
 7. Sense of foreshortened future
- D. Increased arousal (two or more of the following)
 1. Difficulty falling or staying asleep
 2. Irritability or outburst of anger
 3. Difficulty concentrating
 4. Hypervigilance
 5. Exaggerated startle response
- E. Minimum duration of symptoms for 1 month
Significant distress or impairment in social, occupational functioning

Acute : duration of symptoms less than 3 months

Chronic : duration of symptoms 3 months or more

Delayed onset : onset of symptoms at least 6 months after stressor

* Adapted from DSM IV (American Psychiatric Association)

distress in almost every one". It recognized the importance of the magnitude of stressor. The degree of stress had to be excessive as to affect almost every one. However, DSM IV also acknowledged that subject vulnerability contributes significantly to the development of PTSD symptoms in susceptible victims, which might explain why some victims of the same disaster or war were spared from PTSD and others were not.

The incorporation of a person's response as a prerequisite to the DSM IV PTSD diagnostic criteria, led to the broadening of the diagnosis of PTSD to include individuals who demonstrated extreme reactions to less than overwhelming trauma, and more explicit inclusion of those secondary victims who witnessed or were involved indirectly with the traumatic event. These underline the diagnostic importance given to the "perception" of threat or trauma in a case.

The duration of symptoms was used to classify PTSD into 2 subtypes: Acute and Chronic. Both these 2 diagnostic subtypes form a continuum, with Acute PTSD lasting from 1 to 3 months. Any patient with symptoms longer than 3 months will transit into the subtype of Chronic PTSD. This chronological distinction serves to differentiate 2 different prognostic and management patient groups, while delayed onset of symptoms give rise to a third subtype, Delayed Onset PTSD (onset of symptoms is more than 6 months after trauma). This group has generated much interest in studying the temporal relationship of trauma, memory mechanism and development of PTSD. It is not clear to what extent this group represents delayed onset versus delayed reporting of PTSD symptoms.

Acute Stress Disorder (ASD)

In addition, DSM IV¹⁹ added a new diagnosis to cover the period within 1 month of a traumatic event. The addition of this diagnosis, Acute Stress Disorder (ASD—Table II) under Anxiety Disorder category serves to document the long described acute stress response. This is parallel to the combat stress reaction documented in military psychiatry. Its close relationship to PTSD is evident by the use of identical stressor definition, and the common PTSD symptoms of re-experiencing the traumatic event, avoidance of trauma related stimuli, increased arousal, physiological response and impaired functioning. Besides the short timing of the onset and durations of symptoms (2 days to 1 month), the presence of significant dissociative symptoms set it apart from the main PTSD diagnosis. The presence of depersonalization, derealization, being in a daze associated with amnesia denote a very severe, acute, "decompensating" response to stress. A lot of interest has been generated on the aetiology of ASD and the relationship between ASD and PTSD, i.e. whether ASD predicts a poorer outcome and later PTSD.²⁰

Table II : Diagnostic Criteria for Acute Stress Disorder*

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| A. | Exposure to a traumatic event involving actual or threatened death or serious injury, or threat to integrity of self or others, accompanied by intense emotional response of fear, helplessness or terror. |
| B. | Symptoms of dissociation during or after the traumatic event: (three or more of the following) <ol style="list-style-type: none"> 1. Subjective sense of numbing, detachment or absence of emotional responsiveness 2. Reduced awareness of environment (in a daze) 3. Derealization 4. Depersonalization 5. Dissociative amnesia |
| C. | Symptoms of re-experiencing the event (one or more of the following): Recurrent images, thoughts, dreams, illusions, flashback or sense of reliving the event. |
| D. | Marked avoidance of stimuli: thoughts, feelings, conversations, activities, places or persons |
| E. | Increased arousal or symptoms of anxiety: Difficulty sleeping, irritability, poor concentration, hypervigilance, exaggerated startle response, restlessness |
| F. | Significant distress or impairment in social, occupational functioning |
| G. | Minimum duration of symptoms for 2 days, maximum 4 weeks, and occurs within 4 weeks of traumatic event |
| H. | Disturbance not due to drug or substance abuse, not adequately accounted by Brief Psychotic Disorder, or exacerbation of pre-existing Axis I & II disorder |

* Adapted from DSM IV (American Psychiatric Association)

Other Psychiatric Manifestations in Response to Trauma: Co-morbidity of PTSD

Controlled studies examining the relationship between psychiatric disorders and war and other traumatic events have shown that many trauma victims exhibit a range of other psychiatric disorders that coexist with PTSD.^{1,5} These include major depression, anxiety and panic disorders, substance abuse (alcohol and drugs) disorder, adjustment disorder, psychosomatic disorder and antisocial behaviour. Davidson and Fairbank³ reported a higher incidence (62%) of lifetime co-morbidity of psychiatric illness in PTSD patients as compared to a control group (15%). The severity and duration of exposure to trauma (dose effect) are also reported to correlate with higher prevalence of stress related disorders (major depression, generalized anxiety disorder), besides correlating with the severity of PTSD symptoms.²¹ In a study of a population of fire fighters exposed to a natural disaster, co-morbidity also appeared to be an important predictor of chronic PTSD.²²

Traumatic Stress among Medical and Surgical Patients

The negative psychological consequences of medical illness and surgical procedures have attracted research attention in recent years. Documented clinical reports have identified invasive medical procedures (e.g. having tubes in the nose and mouth, being stuck with

needles), physical pain, isolation and “not being in control of oneself” in an intensive care unit (ICU) as constituting sufficient stresses to precipitate an acute stress response among vulnerable ICU patients.²³ A survey of 500 women who underwent obstetric and/or gynaecological procedures revealed a PTSD incidence rate of 6%. These subjects attributed their traumatic experience to a feeling of powerlessness during the procedures, lack of information given, experience of physical pain, perceived unsympathetic attitude on the part of the examiner, and a lack of clearly-understood consent for the procedure.²⁴ In clinical settings, there are many patients who may not satisfy the criteria for the diagnosis of PTSD may, nonetheless, require psychological intervention for their subclinical symptoms.

Conversely, medical illness has also been shown to exacerbate post traumatic stress disorder symptoms. The potential for the worsening of PTSD symptoms with concurrent medical illness (e.g. myocardial infarctions, diabetes, hypertension, arthritis, and cervical fracture) has prompted greater awareness among healthcare givers of this unique psychological vulnerabilities in PTSD patients.²⁵

Personality and Vulnerability Factors-Resilience Hypothesis

Post traumatic stress disorder is one of the few psychiatric illnesses in which there is a clear aetiological precipitant required, i.e. the disorder is not possible without the occurrence of a traumatic event. But not all victims of a traumatic event develop PTSD. There must be an interaction of both personal vulnerability factors and resilience factors as well, although disagreement exist in general, on the exact role of pre-existing personality and interpersonal variables in the production of illnesses following trauma.²⁶

From a clinical perspective, a question commonly asked in the assessment of PTSD patients is, why certain patients fail to reintegrate successfully, to “digest” or to “metabolize” the trauma, with or without professional help. Flach²⁷ proposed that one should look into deficiencies in psychological, biological and environmental components of resilience that a person lacks which result in a dysfunctional state. He identified a list of psychological strengths which are positive attributes in conferring resilience on individuals in coping with trauma. Among these are “insight into oneself and others; a supple sense of self-esteem; the ability to learn from experience; a high tolerance for distress; a low tolerance for outrageous behaviour; open-mindedness; courage; personal discipline; creativity; integrity; a keen sense of humour; a constructive philosophy of life that gives meaning; a willingness to dream dreams that can inspire us all and give us genuine hope”.

In their work on war time survivors, Arthur and

McKenna²⁸ found that “a differential emphasis on the good, hope, the ability to engage in regressive behaviour from time to time, and a serious will to live enabled the prisoners of war to survive the most demeaning and brutal experiences of their lives”. They also noted that those who “best handled severe trauma, as in airplane accidents, had insight into the emotional impact of their recent traumatic experiences, and were able to express their feelings to another immediately following the event”. Although we know little about protective factors and predictors of relapse, we do know that the more severe the trauma, the greater the risk of PTSD.

PTSD Psycho-pharmacotherapy Update

Biology of PTSD: Basis for Drug Therapy

Besides the environmental stressor and personality interactions, recent studies on the neurobiology of PTSD have yielded evidence to suggest a significant role in the contribution of biologic factors at neuro-biochemical pathway level. Research findings appear to suggest that patients with PTSD may display abnormalities in sympathetic nervous system arousal, hypothalamic-pituitary-adrenal-cortical function, endogenous opioid system and in the sleep/arousal physiology. A number of these models are reviewed here, and some of the systems are inter-related.

Noradrenergic dysregulation: Van der Kolk²⁹ suggested a theory of possible catecholaminergic mediation of PTSD symptoms. The initial mobilization and subsequent depletion of norepinephrine seen in animals under extreme stress (in an inescapable shock situation) may suggest a catecholamine mechanism of intrusive and avoidance symptoms of PTSD. This may well correspond to the hypersensitivity and hyposensitivity to catecholamines. Krystal et al³⁰ advanced this model and suggested that the locus coeruleus, the anatomical core of the physiological arousal system, plays a pivotal role in the genesis of fear behaviour similar to the alarm-like reaction in PTSD. Some drugs that have shown some effectiveness in PTSD also prevent the development of learned helplessness in animals exposed to inescapable shock when infused directly into the locus coeruleus or other adrenergically innervated areas. These drugs include tricyclic antidepressant (TCA), monamine oxidase inhibitors (MAOI), benzodiazepines, and clonidine.

A long history of psycho-endocrine research has suggested that 24-hour secretions of catecholamines (norepinephrine and epinephrine) and cortisol provide an integrated sample of metabolism in the sympathetic nervous system, adrenal medulla and adrenal cortical activity respectively. Kosten et al³¹ found raised 24-hour urinary norepinephrine and epinephrine secretion among combat veterans with PTSD as compared to other psychiatric diagnoses; cortisol levels were lower,

and the norepinephrine/cortisol ratio was elevated.

Altered serotonergic functioning: Animal studies have demonstrated that the ascending serotonergic pathways play an important role in the control of anxiety-related behaviour associated with suppression by punishment, and the suppression of behaviour is reversed by serotonin receptor blockers. The importance of serotonin in regulating impulse control and association of low cerebrospinal fluid (CSF) 5-hydroxyindoleacetic acid (5 HIAA) with suicidal, destructive or aggressive behaviour are consistent with earlier findings that serotonin antagonists also cause increased aggression in response to stress, and hyperactivity to stimuli.³² The introduction of serotonin re-uptake inhibitors (SSRI) (fluvoxamine, fluoxetine and gepirone) has demonstrated the reversal of such "emergency" responses and repetitive behaviours in animals following stress. Clinical empirical evidence supports the efficacy of fluoxetine in some patients with PTSD, with marked improvement of both intrusive and avoidance symptoms. Facilitative effects on trauma-focused psychotherapy were also observed.³³

Hypothalamic-Pituitary-Adrenal (HPA) axis hypofunction: The decreased 24-hour urinary free cortisol level seen in PTSD patient, and the enhanced suppression of cortisol following dexamethasone and increased glucocorticoid receptor activity within the HPA axis suggest that HPA hypofunction is associated with PTSD³⁴ and support the hypothesis of an altered negative feedback within the HPA system, i.e. an increased central norepinephrine activity inhibits the release of corticotropin-releasing hormone (CRH), which in turn inhibits the entire HPA system.

Opioid neuromodulation: The opioid model suggests that the presence of distressful and repetitive behaviours in PTSD, e.g. "self-inflicted pain, re-exposure to danger, or emergence of distressful recollection of the trauma" is due to an inappropriate release of endogenous opioids during stressful events. Van der Kolk et al³⁵ demonstrated an increased opiate peptides release in response to stress, leading to stress-induced analgesia (SIA) among Vietnam veterans with PTSD. Naloxone, an opiate antagonist was demonstrated to reverse the SIA among PTSD subjects.³⁶ It was also postulated that this may account for the higher incidence of co-morbidity of opiate drug abuse and dependence among the PTSD Vietnam veterans.

Sleep and dreaming: Following trauma, PTSD patients often have difficulty initiating and maintaining sleep. This is often associated with nightmares and anxiety-dream disturbances. The abnormal sleep architecture observed in PTSD patients includes decreased REM (rapid eye movement) sleep latency, increased stage 1 and 2 sleep, decreased REM and stage 4 sleep, reduced

REM sleep percentage, and decreased sleep efficiency.³⁷ Although there are controversies especially with regards to REM latency and total REM percentage,³⁴ the nightmares in PTSD appear to be unique to PTSD because they are neither REM dream anxiety attacks nor non-REM night terror/nightmares. Common themes also arise from both.³⁸ Ross et al³⁹ suggested that these phenomenon may be seen as "reflections of a repetitive, unsuccessful attempt to reconstitute psychological defenses". Clinical pharmacological reports suggest some usefulness of phenelzine (monoamine oxidase inhibitors), tricyclic antidepressant, and lithium carbonate on sleep disturbances and nightmare symptoms of PTSD.⁴⁰ All these have marked or significant effect on REM sleep reduction. This further supports viewing PTSD as essentially an arousal disorder.

Kindling model: Kindling is a process by which neuroanatomical structures become increasingly sensitized following repeated exposure to electrical stimulation. When observed in the limbic system, kindling could explain the development of progressive neurophysiological abnormalities such as seizures and abnormal repetitive behaviours. Van der Kolk⁴¹ suggests that repeated traumatization as in child abuse, or experiencing one trauma followed by intrusive re-experiencing could lead to lasting neurobiological abnormalities and behavioural changes via kindling phenomena. The possible mechanism could be through chronic central sympathetic arousal in PTSD, mediated by the locus coeruleus, kindling limbic nuclei, producing a stable biological abnormality which would explain the "stability" of PTSD when left untreated. This model also supports the use of antikindling drugs such as carbamazepine, valproate and lithium in PTSD.⁴²

Pharmacotherapy

Controlled studies: Sutherland and Davidson⁴³ reviewed 6 reported placebo-controlled drug trial studies on PTSD patients; five used antidepressant and one used benzodiazepines. Three of the antidepressant studies used tricyclic antidepressants (TCAs), two on phenelzine (MAOI) and one selective serotonin reuptake inhibitor (SSRI) fluoxetine.

Of these, trials with amitriptyline, imipramine, phenelzine, and fluoxetine demonstrated positive drug effects in PTSD relative to placebo. This contrasts with desipramine, and alprazolam (benzodiazepine group) trials which produced significant improvement of the IES (Impact Event Scale) over the placebo. It was identified that the positive trials were related to the use of drugs with prominent serotonergic effects, whereas the negative outcome trials included a mixture of catecholaminergic and GABAergic effects (desipramine, alprazolam). The dosage and duration of drug therapy also influenced the outcome. Those with

high drug dose (e.g. phenelzine 45-75mg/D, imipramine 50-300 mg /D, amitriptyline 50-300 mg / D, and fluoxetine 20-40 mg/D) for an 8-week period was related with better outcome against those associated with lower doses for shorter time.

Clinical studies also found that tricyclic antidepressants (such as imipramine) and monoamine oxidase inhibitor (phenelzine) significantly reduced specific *intrusive and hyperarousal anxiety symptoms* of PTSD, but had less effect on the avoidance symptoms and emotional numbing,⁴⁴ whereas serotonergic drugs (e.g. fluoxetine) and amitriptyline have been demonstrated to be useful in the improvement of *avoidance symptoms*. Another key symptom of PTSD, *impulse control problem*, has been shown to improve with the use of carbamazepine and lithium. Therefore, it is not too surprising that many different drugs may be useful, and at times, multi-drug therapy may be adopted for some patients but not the others. And symptoms (intrusion, anxious arousal, avoidance, sleep and impulsivity) relief provided by pharmacotherapy facilitates the patient's participation in behavioural, individual or group psychotherapy treatment programmes, which in turn best help the negative symptoms (phobic avoidance and withdrawal) associated with chronic PTSD.

The role of pharmacotherapy: Overall, PTSD is a common disorder with high co-morbidity and a tendency towards chronicity. It can respond slowly to treatment or show, in acute form, rapid recovery.⁴⁵ It was not until the mid 1980s that there was greater research interest and evidence to support a biological profile consisting of alterations in sympathetic arousal, neuroendocrine and sleep/dream cycle, as different from both major depression and panic disorder.⁴⁶ Prior to that, pharmacotherapy of PTSD was mainly focused on symptom relief, and as an adjunct to psychological treatment (intra-psychic or behavioural). Although the causative trauma may differ, e.g. natural disaster, combat and rape, the symptomatology and response to treatment is similar. Also, studies have found that different medications appear to affect different target symptom clusters in PTSD, i.e. intrusive, avoidance and generalized hyperarousal symptoms.

Despite these optimistic directions, pharmacologic treatment of PTSD is primarily directed at symptom control rather than the disorder. The role of pharmacotherapy in PTSD has been described by Vargus and Davidson⁴⁷:

1. To provide relief from distressing and intrusive symptoms (nightmares, flashbacks, daytime thoughts or images related to trauma)
2. To improve avoidance symptoms
3. To bring under control the generalized autonomic hyperarousal, hypervigilance, irritability,

insomnia, and increase startle response

4. To relieve depression, anhedonia, and suicidality
5. To gain greater control over violent impulses and aggressivity
6. To control acute dissociative and psychotic features when present
7. To facilitate psychotherapy

Psychological Treatment of PTSD: A Second Dimension

Psychological treatment has evolved to be the main stay of treatment for PTSD over the past decade, and copious literature reporting positive outcome based on various behavioural, cognitive and psychodynamic treatment models for different types of trauma cases have surfaced since. Many of these claim specific relief for certain PTSD symptom clusters while having a limited effect on the rest.⁴⁸ However, as McFarlane⁴⁹ noted, there is a paucity of sophisticated outcome literature offering validated conclusions on the treatment of PTSD versus many assertions to their claims. He noted that this was further complicated by treatment and ethical constraints in the implementation of controlled study for certain severe trauma groups such as survivors of torture, child abuse and severely abused adult victims. The historical competitive relationship among the schools of therapy has also made it more difficult for the pursuit of a more eclectic approach to treatment studies as indicated by the knowledge. Nevertheless, there has been encouraging outcome results from recent studies on war veterans and rape victims with PTSD treated with behavioural-cognitive and psychodynamic therapy programmes.

The Behavioural Model

The two-factor behavioural model (Mowrer's) employs classic and operant conditioning learning theory to explain the development and persistence of the acute PTSD symptoms associated with the trauma.⁵⁰ Subjects who reacted to a traumatic event (unconditioned stimulus) with fear and arousal (unconditioned response), when exposed to cues/stimuli (conditioned stimulus) associated with the original trauma will experience the same "conditioned response" (fear and arousal symptoms). This simple learning is followed by operant conditioning which sustained the "learned response" by rewarding avoidance behaviour (of internal and external cues) with relief or reduction of emotional distress associated with the trauma. And reinforcement of such avoidance would prevent extinction of the learned response, and in turn allow the expansion of the secondary and tertiary cues.

Behavioural therapy is designed to unlink the conditioned response to conditioned stimulus that has been paired with the trauma. Established behavioural tech-

niques include *gradual desensitization* or *massive flooding* re-exposure to the conditioned stimulus either *in-vivo* using live object or situation relating to the trauma, or *in-vitro* using mental imaging exposure of the trauma.

A review of four controlled studies by Shalev et al⁵¹ on PTSD in war veterans indicated that *flooding* improved PTSD symptoms. A mixture of desired positive effects from flooding exposure include a significant reduction of re-experiencing anxiety, depression, improvement on personal treatment of sleep disturbances, association with a positive adjustment and a reduction of physiological responses to the traumatic imaginary.^{48,51} On the other hand, a report by Pitman⁵² showed that flooding therapy could induce an exacerbation of depression, panic anxiety, increased alcohol consumption and mobilization of negative appraisal.

Controlled trials using mental imaging desensitization were associated with a reduction in nightmares, flashbacks, muscle tension and improvement of phobic symptoms. One main disadvantage of desensitization therapy noted by Shalev is its limitation to the focus of a single traumatic event (e.g. rape and traffic accident), whereas in many other instances PTSD is the result of prolonged exposure to a chain of events, such as war, years of physical or sexual abuse or torture. And it has been shown that "desensitization of one traumatic incident does not extend to other traumatic incidents engendered by the same event".⁵¹

However, some reports have cautioned about generalizing flooding desensitization techniques in all treatment cases, as adverse effects of increased intrusion and avoidance symptoms were reported post treatment. The Koach project, a controlled live exposure therapy study⁵³ designed to treat PTSD among Lebanon War veterans, showed that participants fared worse than controls with their initial psychometric scores both immediately and at 9 months after the programme. Overall, as suggested by Shalev, "studies of behavioural treatment in PTSD report a significant but partial improvement, suggesting that the pathophysiology of PTSD encompass more than just learned conditioning".

Cognitive Model

The cognitive model of PTSD focused on the meaning and psychological representation of the trauma within the subject's cognitive framework. This involves the integration of information processing and the cognitive appraisal with threat perception, its predictability and controllability of the trauma. Traumatic events not only challenge the validity of previously held belief that the world is safe, benevolent and meaningful, and the victim invulnerable and strong, but alter the appraisal of the world as a treacherous and dangerous place, and transform this self-schemata into one of incompetence and vulnerability. The accentuation of a negative cogni-

tive self-schemata may then lead to dysfunctional thinking and therefore to depression, anxiety and PTSD.

These associated maladaptive assumptions and traumatic meanings are dealt with using cognitive restructuring, which involve examining whether the catastrophic outcome could have been predicted, and if the trauma could have been modified by any action of the subject, and the issue of individual responsibility.⁴⁹ The need to activate traumatic memory and introduce new information which are incompatible with the previous maladaptive assumption is important and facilitates the formation of a new memory which is more acceptable to the memory schemata.⁵⁴

Shalev⁵¹ reviewed three controlled outcome studies on cognitive treatment of rape and sexual assault PTSD subjects. All reported significant improvement of PTSD symptoms immediately and were maintained at follow-up of 3 to 6 months later. Significant reduction in symptoms of distress, depression, avoidance, intrusion and improved self expression and self concept were reported by Resick and Schnicke.⁵⁵

A review by McFarlane⁴⁹ on the comparative outcomes of cognitive and behavioural sensitization treatment techniques, found that there were more similarities than differences. Studies⁵⁵ on the treatment outcome of PTSD rape victims found no statistical difference between the two treatment groups. It has been suggested that a cognitive behavioural approach might be preferred for certain trauma groups to employ the exposure strategy to reduce the intrusions and hyperarousal symptoms, while cognitive restructuring worked towards the reintegration of meaning of trauma into a new memory schemata, and promoted the development of this new memory into a new life experience.

Psychodynamic Psychotherapy

Based on the concept of traumatic neurosis discussed above, extreme trauma is believed to alter the rules of mental functioning and damage the ego ideal. The PTSD patient is thought to have impaired self healing capacity, and is subjected to the dysfunctional set of mental processes. Psychodynamic psychotherapy focuses on interpretation of the meaning of traumatic event in relation to the ego, and its relation to the associated symptoms and behaviour. Through gaining insight into both the conscious and unconscious meaning of the symptom with respect to him or herself, it aims to help the patient master his inner experiences, and repair and restore the integrity of life.⁵⁶ This approach of reinterpreting the trauma experience needs to be augmented by the provision of ego support within the therapeutic context, helping the patient develop realistic appraisals of his affects and responses, and developing coping strategies to contain the anxiety arising from confronting the traumatic experiences

during therapy.⁶⁰

In a comparison study by Brom et al,⁵⁷ psychodynamic psychotherapy was found to have similar treatment outcomes as with desensitization, hypnotherapy treatment groups, and had significant symptom improvement over the control group. Lindy et al⁵⁸ in a study of 21 PTSD war veterans who received intensive psychoanalytic therapy reported reduction of general psychiatric symptom and intrusion, but no effect on avoidance. There were however numerous case reports which emphasized important treatment techniques required to produce good treatment success, and these include understanding the origin and adaptive function of re-living experiences in PTSD, and focusing on self-concept, working alliance and counter transference.⁵⁹

Other Treatment Modalities

Group therapy has been reported to be an effective treatment modality among groups of victims with common exposure experiences, especially the war veterans and post disaster victims like in flood or hurricanes. Vietnam war veterans met in "rap groups", which consisted of disclosure of common experiences, validation of feelings, and the sharing of existential distress.⁵¹ This allows the victims to address the issues of trust, anger, survivor guilt and personal social reintegration, which are all common to PTSD. Group therapy also attempts to reduce isolation, helplessness, enhance social competence and anger management through support from within the group.

Family or couple therapy helps to assist family or spouses whose interactions have been affected by the family member with PTSD. In particular anger, irritability, poor impulse control, mistrust and alienation often deteriorates one's level of social functioning and aggravate the already strained relationships. These further diminish the much needed support and understanding that PTSD patients lack.

Hypnosis therapy allows the PTSD patient to confront the trauma and decrease the triggered psychosomatic response in a less ego-anxiety provoking stage. In Shalev's review,⁵¹ some interesting studies found that while Vietnam veterans were more hypnotizable than controls, Shalev's Lebanon war veterans resisted suggestion as a way to remain in control, and some responded with severe dissociation upon induction. As we know, remaining in-control is a key issue in the psychopathology of PTSD. Nevertheless, it has been reported that subjects showed improvement of avoidance and distress symptoms over the control group.⁵⁷

Eye movement desensitization (EMD) was first reported by Shapiro.⁶⁰ This procedure involves eliciting saccadic eye movements in the patient while mentally maintaining the most salient aspect of a traumatic memory, and

describing the event and its associated feelings. Shapiro reported that the technique resulted in a lasting reduction of anxiety, changes in the cognitive assessment of the memory and cessation of flashbacks, intrusive thoughts, and sleep disturbances. One controlled study report indicated that the procedure can be extremely effective in just one session, although multiple sessions were necessary for some combat veteran cases. Despite this promising outcome, studies are still ongoing to determine in a controlled manner, whether EMD is effective. It is likely that any results can probably be explained through the "semi-hypnotic" state induced by the saccadic eye movement process, rather than any new mechanism of treatment.

Debriefing following traumatic exposure has been used in groups exposed to traumatic events as a stress management technique.⁶¹ This is a group oriented intervention in which major elements of a trauma are reviewed by the participants shortly after the event. Post combat briefing, a method first developed by Brigadier General SLA Marshall (1944),⁶² was applied in World War II, the Korean War and the Vietnam War by the US Army. These debriefings entail precise reconstruction of the battle in a chronological path by the combatants in a group. Marshall described the psychological impact of debriefing on the group as a "spiritual purge". He noted that this emotional response led to increased self-esteem and relief among the soldiers. A similar model, Critical Incident Stress Debriefing (CISD) was developed by Mitchell and Everly⁶³ to help high-risk emergency-response workers deal with their work related trauma. This seven-step protocol provided the rescue workers with a framework to explore and work through their traumatic experiences. Mitchell and Everly believes that CISD derive its effectiveness through early intervention, as well as providing an opportunity for catharsis. The "finite" behavioural structure and group support also provided an environment that decreased apprehension and distrust, facilitating emotional and information sharing, amidst the background of chaos in a disaster. Despite a growing number of applications, there are few empirical studies and controlled research that examine the immediate and long term outcome variables of this technique.

Principles of Management of PTSD

In the management of PTSD, almost every medical and psychological treatment modality has documented some success, but none was able to claim absolute superiority in treatment outcome, probably because the treatment populations were different. Studies are just now addressing differences in treatment for Acute versus Chronic PTSD and differences necessitated by the different trauma experiences (e.g. war, disaster and rape). However, a flexible combination treatment pro-

gramme consisting of pharmacotherapy and psychotherapy appears to have a synergistic effect.”

A conceptual summary of an approach to treatment of PTSD⁶⁵ will be a useful reminder that different treatment modalities may be required for the different dimensions of PTSD, addressing both the central role of trauma in causing intrusive thinking and avoidance symptoms, personality and interpersonal contributions to PTSD and other co-morbid disorders that may be present.

- Establish a therapeutic alliance, engage the patient and allow the accurate medical assessment of physical symptoms that may be the patient's primary preoccupation.
- Evoke a sense of hope and provide an explanation for the distress of the PTSD; these by themselves are important elements for the improvement process to begin.
- Identify and treat PTSD component symptoms:
 - explore and understand the nature of intrusive experiences
 - focus separately on both intrusive painful memories and the feelings of loss and bereavement
 - relief of hyperarousal symptoms, using adjunct pharmacotherapy or behavioural relaxation techniques
 - identification of the patient's emotional constriction, encouraging involvement and re-engagement in activities and relationships
 - family or group therapy to focus on interpersonal and social problems related to PTSD
- Treat co-morbid disorders, e.g. depression, panic disorder, alcohol abuse and impulse control problems.
- Examine the context of illnesses and assess the impact of trauma on other family members, who could be another victim of the same event which may complicate the recovery process.
- Consider perpetuating factors as PTSD may develop into a chronic process, and address issues that interfere with the recovery process.
- Important for the therapist to confront the reality of the trauma within the victim's individual context, discerning the level of impact on the victim, the interpersonal meaning of the trauma, the intensity and duration of the trauma exposure, the nearness to death experienced, and possible violation of trust and infringement of human dignity.

Conclusions

The evolution of the diagnostic classification of traumatic stress syndromes has been long and tedious, in part because of the complex process of traumatization

affecting the biological, psychological and social functioning of an individual. The repeated revisions of DSM diagnostic criteria further serve to affirm this complex process as our understanding of psychotraumatology continues to unfold each day. Our new understanding provides a multi-dimensional frame work critical to the formulation of a biopsychosocial multi-layered management approach to post traumatic stress disorder, and we can continue to give the many victims of post traumatic stress disorder hope of a brighter tomorrow.

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