Chapter II

POST - TRAUMATIC STRESS DISORDER
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Definition

The term post-traumatic stress disorder (PTSD) first appeared in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III) and is listed within the group of anxiety disorders. DSM-III (1980) defines PTSD as follows:

"The essential feature is the development of characteristic symptoms following a psychological traumatic event that is generally outside the range of usual human experience. The characteristic symptoms involve reexperiencing the traumatic event; numbing of responsiveness to, or reduced involvement with, the external world; and a variety of automatic, dysphoric, or cognitive symptom" DSM-III (1980).

The revision of DSM-III (DSM-III R. 1987) specifies the types of stressors that cause psychological trauma: "Serious threat to one's life or physical integrity; a serious threat or harm to one's children, spouse, or other close relatives and friends; sudden destruction of one's home or community or seeing another person who has recently been, or is being, seriously injured or killed as the result of an accident or physical violence. In some cases the trauma may be learning about a serious threat or harm to a close friend or relative, e.g., that one's child has been kidnapped, tortured, or killed".
This definition highlights the importance and severity of the stressor and recognizes that PTSD comprises a group of symptoms that can arise from a wide variety of severe, psychologically traumatic events.

DSM-III-R (1987) Defines PTSD as Follows:

"The essential feature of this disorder is the development of characteristic symptoms following a psychologically distressing event that is outside the range of usual human experience (i.e., outside the range of such common experiences as simple bereavement, chronic illness, business losses, and marital conflict). The stressor producing this syndrome would be markedly distressing to almost anyone, and is usually experienced with intense fear, terror, and helplessness. The characteristic symptoms involve reexperiencing of the traumatic event, avoidance of stimuli associated with the event or numbing of general responsiveness, and increased arousal. The diagnosis is not made if the disturbance lasts less than one month."

The trauma may be experienced alone (e.g., rape or assault) or in the company of group of people (e.g., military combat). Stressors producing this disorder include natural disaster (e.g., floods, earth-quakes, accidental disasters
(e.g., car accidents with serious physical injury, airplane crashes, large fires, collapse of physical structures), or deliberately caused disasters (e.g., bombing, torture, death camps). Some stressors frequently produce the disorder (e.g., torture), and others produce it only occasionally (e.g., natural disasters or car accidents). Sometimes there is a concomitant physical component of the trauma, which may even involve direct damage to the central nervous system (e.g., malnutrition, head injury). The disorder is apparently more severe and longer-lasting when the stressor is of human design.

Exposure to combat is an extremely stressful experience that can impair functioning (Selye, 1956 and Wolf, 1953). The most widespread manifestation of psychopathology on the battlefield is combat stress reaction (CSR), also known by such names as 'battle shock' and 'battle fatigue'. Combat stress reaction can be characterized by various features: psychomotor retardation, withdrawal, increased sympathetic activities, stuttering, confusion, nausea, vomiting, and paranoid reactions (Selye, 1956).

Combat stress reaction syndrome is marked by a sense of inadequacy in coping with a real and acute threat to life. The soldier experiences a feeling of loss of control accompanied by
overwhelming anxiety and a marked decline in functioning. Despite the extreme variability of this phenomenon, a common denominator can be identified: the soldier ceases to function and/or begins to function in an extreme manner that usually endangers himself and/or his comrades (Kardiner, 1987).

After the war, the most common and conspicuous manifestation of combat pathology are combat stress reaction (CSR) and post-traumatic stress disorder (PTSD). CSR refers to labile polymorphic manifestation affecting cognition, affect, and behaviour during battle. PTSD is characterized by reexperiencing the traumatic event after battles have ended, numbing of responsiveness to, or reduced involvement with, the external world and a variety of autonomic, dysphoric, or cognitive symptoms (DSM-III APA, 1980).

DSM-III accompanying its definition of PTSD, delineates what it terms 'associated features', psychiatric symptoms that are frequently observed in conjunction with PTSD. In addition to these psychiatric symptoms, the DSM-III points out, PTSD may result in occupational or recreational impairment and other difficulties in social functioning.
History of Post-traumatic Stress Disorder (PTSD)

Psychological reactions to stress have been studied with increasing sophistication during the twentieth century, although old fashioned ideas, prejudices, and blind spots from the wish to ignore the effects of trauma and stress remain to plague clinical practice in this area (Titchner and Ross, 1974).

Prior to understanding of the dynamics of traumatic neurosis, many writers cited theories which came close to the truth.

Sydenham (1624-1684) stated that "hysterical hemiplegia may proceed from some violent commotion of the mind or strong emotions", but he saw the cause of these as animal spirits.

William Harvey (1649) described a case history in which, he recognized revenge as a precipitating cause. He described a man overcome by hatred, spite, and passion after receiving an injury from someone more powerful than he (Alexander, Selesnick, 1966).

The earliest explanations, in the 1800s, of conditions which we now recognize as neuroses following trauma were ones which postulated "molecular disarrangement" or vascular changes.
in the spinal cord. John Eric Erichsen (1977) described this condition as "symptoms following (train) accidents which may assume the form of a traumatic hysteria, neurasthenia, hypochondriasis, or melancholia". He called this syndrome "railway spine", attributing it to organic causes and the idea persisted in literature as "Erichsen's disease" (Hoerr and Osol, 1956).

Hodges in 1880 and Page in 1883 did not go along with the molecular disarrangement thesis. Hodges (1880) suggested "vascular changes in the cord as causing the clinical picture usually encountered after railway accidents". Page (1883) said "many cases were due to intraspinal hemorrhage and to causes other than trauma such as syphilis". Page also recognized emotional factors, and cited fright and other psychic factors as causes (Strauss and Savitsky, 1934).

Brodie (1837) was probably the first physician to recognize that for some hysterical symptoms, "fear, suggestion, and unconscious simulation are primary factors" (Ziegler, 1962).

The 1880s brought a flurry of discussion, and study of the question of psychogenic versus physical etiology. Oppenheimer (1880), in his study of the neuropsychiatric sequelae
following injury, recognized four conditions: hysteria, neurasthenia, or organic syndromes, and traumatic neurosis (Strauses and Savitsky 1934). However, he did not feel that these conditions were emotional. His explanation of the etiology of traumatic neurosis was that it was caused by molecular changes "due to electrical changes in the central nervous system" (Bennett 1964).

Charcot (1880) disagreed with Oppenham. He felt that the cause of traumatic neurosis was psychogenic (Bennett 1964). He noted that the symptoms of traumatic neurosis resembled changes seen during hypnosis.

Sigmund Freud, in his paper in 1920, sharply differentiated between the traumatic neurosis and the transference neurosis. He defined traumatic neurosis as the human ego defending itself from danger which threatens it from without, whereas in the transference neurosis, the enemy from which the ego is defending itself is actually the Libido whose demands seem to be menacing. Major factors in neurosis following traumas, Freud felt, were fright and surprise. The sudden flooding of the psychic apparatus with excessive stimuli is the start of traumatic neurosis. Following this, the psychic apparatus "endeavors to handle these stimuli to slowly reduce their effect". If the psychic apparatus exhausts its energy a neurosis develops (Dreyfuss, 1949).
Freud noted the advent of dreams after trauma. He wrote, "The dreams are endeavoring to master the stimulus retrospectively by developing the anxiety whose omission was the cause of the traumatic neurosis". This he attributed to an "obedience to the compulsion to repeat". He further stated that a severe injury would call forth narcissistic hypercathexis of the injured organ and this, he said, would bind the quantity of sexual excitation, which would develop because of the lack of preparation for anxiety, and as a response to the trauma (Fenichel, 1932).

Fenichel, in 1932, felt that accidents may release a neurotic reaction because they serve as temptations for aggression, with the subsequent development of defence mechanisms against the aggression. As did Freud, he further recognized that accidents are perceived either as castration or as threatening the loss of parental love or the favour of destiny (Fenichel, 1932).

Although wars are commonly thought of in terms of the havoc and unhappiness they cause, out of them come the understanding of traumatic neurosis.

During the civil war, De Costa (1871) defined a syndrome which is called DeCosta's syndrome. DeCosta's syndrome
(soldier's heart) was a clinical phenomenon observed in young soldiers who had irregularities in heart rate which we now recognize as anxiety symptoms. A combat soldier with palpitations and chest pains was felt to have a functional cardiac disturbance which DeCosta called "soldier's heart" or "effect syndrome" (DeCosta, 1871).

During World War I, traumatic reactions to combat conditions were called "shell shock", a term coined by a British Pathologist Col. Frederick Mott (1919) who regarded such reactions as organic conditions produced by minute hemorrhages of the brain. It was gradually realized, however, that only a very small percentage of such cases represented physical injury from concussion of exploding shells or bombs. Most of these people were suffering instead from the general combat situation with its physical fatigue, ever-present threat of death or mutilation, and severe psychological shocks (Hoch, 1934 and Strauss and Savitsky 1934).

Out of World War I came a noted psychiatrist named Abram Kardiner, who did exhaustive research with victims of traumatic neurosis in both World Wars. He developed a theory which primarily modified the Freudian theory of the time. He went to great lengths to discuss the functions of the ego and how they were affected by the sudden onset of trauma. He discussed the
adaptive processes of the organism and the attempts of the organism to achieve psychic equilibrium after trauma. "The traumatic experience can precipitate any of the well-known types of neurotic or psychic disorders" (Kardiner 1947).

Grinker and Spiegel (1945) in their book, "Men under Stress", emphasized the factor of ego psychology and the effect of trauma upon the ego. All individuals are susceptible to this condition, they state. How severe the neurosis will be depends on the persons' pretraumatic patterns (Alexander 1948).

During World War II, traumatic reactions to the continuing stressor of combat passed through a number of classifications, such as "operational fatigue" and "war neurosis", before finally being termed "combat fatigue" or "combat exhaustion" in the Korean and Vietnam War.

World War II brought further clinical experience not only with combatant but also with civilians: survivors of prisoner-of-war camps, Nazi death camps, and the atomic bombing of Japan. An early description of symptoms among civilians caught in the disastrous Boston Coconut Grove fire of 1941 (Adler, 1943) listed general nervousness, irritability, fatigue, insomnia, and nightmares. Both physical and psychological causes of the resulting disorder were stressed (Adler 1943).
Early investigator of the survivors of death camps described symptoms of anxiety, motor restlessness, hyper-apprehensiveness, difficulty in sleeping, night terrors, fatigue, phobic reactions, and a constant preoccupation with recollections of persecutory experiences. This became known as the concentration camp syndrome. Some investigators regarded this as the significant factor of an organic brain disease as a result of physical injury. The concentration camp syndrome occurred, however, so regularly without evidence of predisposition among such a high proportion of survivors that it became clear the symptoms were almost entirely the result of the psychological trauma itself (Eitinger, 1964, 1969, 1973; Sigal et al., 1971; Warnes, 1972; Ross, 1966; Hoppe, 1971; Chodoff, 1966). The existential factors involved surviving the concentration camp and were vividly described by Viktor Frankl (1959) in his book "From Death Camp to Existentialism".

Robert Lifton (1979) emphasized in "The Broken Connection" the death imagery with resultant symptoms among civilians after the bombing of Hiroshima.

The traumatic neurosis of war was described in terms quite similar to DSM-III's PTSD. Recognition of the neuroses of World War II veterans led to the category of gross stress reaction in the first edition of the Diagnostic and Statistical
Manual of Mental Disorder (DSM-I), 1952. Gross stress reaction is defined as a transient reaction to unusual, severe, or overwhelming combat stress. It listed five diagnostic criteria; (1) unusual stress; (2) previous normal personality; (3) reversibility; (4) possible progress to one of the neurotic reactions; (5) if persistent reaction, "this term is to be regarded as a temporary diagnosis to be used only until a more definite diagnosis is established" (DSM-I, APA 1952).

Gross stress reaction category was not included in the second edition of the Diagnostic and Statistical Manual of Mental Disorder (DSM-II) in 1968, however, despite the syndrome's being well described in multiple settings, both civilian and military (DSM-II, APA 1968).

Work on reactions to trauma was continued, particularly, one influential report described a phasic reaction of intrusive responses alternating with avoidant behaviour and denial (Horowitz et al., 1979). According to Horowitz et al., 1979, the psychological sequelae of trauma can be summarized in two major intrapsychic manifestations: intrusion and avoidance. Intrusion refers to the penetration into consciousness of thoughts, images, feelings, and nightmares about the war, and to a variety of repetitive behaviours. Avoidance reflects the tendencies of psychic numbing, conscious denial of meaning and
consequences, behavioural inhibition and counter phobic activities related to the stressful event. Intrusion is generally the initial phase, followed by avoidance. Intrusion and avoidance may then alternate in the course of the post-trauma period, according to the individual's idiosyncratic pattern, until working through occurs (Horowitz, 1982).

The increasingly obvious problems of Vietnam veterans plus clinical work with victims of multiple disasters made clear a need for a post-traumatic stress category in DSM-III. The symptoms were primarily based on the varieties of clinical reports described above, and so the syndrome was placed with anxiety disorders. Unlike most other disorders in DSM-III, PTSD did not undergo prior extensive field or interrater reliability studies, resulting in some controversy over its validity. Studies done after the appearance of DSM-III, however, generally confirmed the disorder's validity. (Green et al., 1985).

The appearance of the operationally described category increased interest in studying and comparing reactions to such diverse disasters and personal calamities as incest, rape, child-kidnapping, terrorism, the Vietnam war (Figley, 1979; Horowitz, 1976; Terr, 1983; Shore et al., 1989). Three miles Island nuclear accident (Bromet, Schulberg, Dunn, 1982).
The Mt. St. Helens volcano explosion (Shore et al., 1986), Cambodian Concentration Camps (Kinzie et al., 1984) and children viewing the murder of parents (Eth and Pynoos, 1985). As clinical and research experience mounted, it became necessary to modify the criteria, and DSM-III-R was published in 1987 (Williams, 1980; Van der Kolk et al., 1982; Van Kampen et al., 1986; Friedman, 1981; Frye and Stockon, 1982).

DSM-III had four main criteria for PTSD: (1) evidence of a recognizable stressor that would evoke significant symptoms of distress in almost everyone, (2) reexperiencing the trauma, (3) numbing of responsiveness, and (4) at least two (from a list of six) associated symptoms that were not present before trauma. It recognized that some of the reactions were chronic or delayed.

Comparative Nosology

In the ninth revision of ICD (ICD-9) 1975, the only similar disorder is acute reaction to stress, defined as "a very transient disorder of any severity and nature which occurs in individuals without any apparent mental disorder in response to exceptional physical or mental stress such as natural catastrophes or battle and which usually subsides within hours or days".
The temporary nature of the disorder and the specification that it occur in normal personalities are the primary differences from DSM-III's description of PTSD.

DSM-III-R in 1987 makes major changes in the emphasis on symptoms in PTSD and thus reflects some conceptual change. The stressor, which is the first criterion, is explicitly listed in the definition, and examples are given of psychologically traumatic events.

The Diagnostic Criteria for Post-traumatic Stress Disorder

A. The person has experienced an event that is outside the range of usual human experience and that would be markedly distressing to almost anyone, e.g., serious threat to one's life or physical integrity; serious threat or harm to one's children, spouse, or other close relatives and friends; sudden destruction of one's home or community; or seeing another person who has recently been, or is being, seriously injured or killed as the result of an accident or physical violence.

B. The traumatic event is persistently reexperienced in at least one of the following ways:
1. Recurrent and intrusive distressing recollection of the event (in young children, repetitive play in which themes or aspects of the trauma are expressed),

2. Recurrent distressing dreams of the event,

3. Sudden acting or feeling as if the traumatic event were recurring (includes a sense of relieving the experience, illusions, hallucinations, and disassociative (flashback) episodes, even those that occur upon awakening or when intoxicated),

4. Intense psychological distress at exposure to events that symbolize or resemble an aspect of the traumatic event, including anniversaries of the trauma.

C. Persistent avoidance of stimuli associated with the trauma or numbing of general responsiveness (not present before the trauma) as indicated by at least three of the following:

1. Efforts to avoid thoughts or feelings associated with the trauma,

2. Efforts to avoid activities or situations that arouse recollections of the trauma,
3. Inability to recall an important aspect of the trauma (psychogenic amnesia),

4. Markedly diminished interest in significant activities (in young children, loss of recently acquired developmental skills such as toilet training or language skills),

5. Feeling of detachment or estrangement from others

6. Restricted range of affect (i.e., unable to have loving feelings),

7. Sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a long life).

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by at least two of the following:

1. Difficulty falling or staying asleep,

2. Irritability or outbursts of anger,

3. Difficulty concentrating,

4. Hypervigilance,

5. Exaggerated startle response,
6. Physiologic reactivity upon exposure to events that symbolize or resemble an aspect of the traumatic event (e.g., a woman who was raped in an elevator breaks out in a sweat when entering any elevator).

E. Duration of the disturbance (symptoms in B, C and D) of at least 1 month.

Specify delayed onset if the onset of symptoms was at least 6 months after the trauma.

The other three groups of symptoms are: (1) persistent reexperience of the traumatic events, (2) persistent avoidance of stimuli associated with the trauma, or numbing of general responsiveness (at least three of seven symptoms) must be present, (3) persistent symptoms of increased arousal (at least two of six symptoms), (4) duration of the disturbance of at least 1 month. All these symptoms must occur during the same period for at least 1 month. The total number of possible symptoms has increased to 17. Compared with DSM-III-R places more emphasis on the avoidance of stimuli associated with the trauma and less on numbing of responsiveness; it introduced symptoms of persistent increased arousal as a necessary criterion and removed survivor guilt as an associated symptom.
DSM-III recognizes two subtypes of PTSD: the acute form with an onset of symptoms within 6 months of the trauma and a duration of symptoms of less than 6 months, and the chronic or delayed forms with a duration of symptoms of 6 months or more (chronic) or with an onset of symptoms at least 6 months after the trauma (delayed). The acute and chronic subtypes are not listed in DSM-III-R but the diagnosis should specify whether there is a delayed onset (i.e., onset of symptom of at least 6 months after the trauma).

Epidemiology

The definition of PTSD requires that it follows unusual and severe stress. Therefore, it can occur only when the patient has undergone stressful events. The incidence of PTSD increases after disasters involving large numbers of people. Only since the DSM-III definition was formally accepted have comparable epidemiological studies been made. Data on PTSD in the general population and among victims of specific disasters are 50 to 80 per cent of survivors.

Early reports, using various criteria for PTSD gave incidence of the disorder that increased with the severity of stresses. Eighty-five per cent of victims of Nazi death camps were reported to have the concentration camp syndrome, and none
was without pathology (Chodoff, 1963). Fifty-seven per cent (26 out of 46) of patients in the 1941 Coconut Grove night-club disaster developed psychiatric complications involving multiple symptoms (Adler, 1943). Traumatic-neurotic reactions were found in 80 per cent of the survivors of the 1972 Buffalo Creek disaster (Kubler, Ross, 1980). Fifty-nine per cent of soldiers in the 1982 Lebanon war developed PTSD (Solomon et al., 1987).

Newer studies using the DSM-III criteria offer a better comparison. PTSD was recently added to the diagnoses studied in the St. Louis Epidemiologic Catchment Area (ECA) 1987 survey (Robins et al., 1984). In the general population it is a rare disorder, occurring in 0.5 per cent of men and 1.2 per cent of women. In a study following the volcanic eruption of Mt.St.Helens, a lifetime prevalence of PTSD was 2.9 per cent for males and 2.3 per cent for females (Shore, Tatum and Vollmer, 1986). Sixty per cent of the disorders were unrelated to the Mt.St.Helens experience. In both studies the most common stressors in men were combat experience. Being assaulted was the most common stressor in women in the St. Louis study and second only to the experience of the explosion itself in the Mt.St.Helens disaster.

A survey among Vietnam veterans found that after active duty, 26 per cent in heavy combat, 17 per cent in average
combat, and 7 per cent not in combat met the criteria for PTSD. A follow-up study 6 to 16 years after the men returned home showed 36 per cent of those in heavy combat, 24 per cent in average combat, and 17 per cent not in combat suffering from chronic PTSD (Laufer, 1985; Allerton, 1970).

Structural interviews with former World War II prisoners of war (POWs) revealed that 67 per cent had a PTSD after their release. Of those affected, 24 per cent had moderate residual symptoms, and 8 per cent (5 per cent of the total) had marked chronic PTSD 40 years later (Kluznik et al., 1986). Using a structured interview with Cambodian children who had endured 4 years of forced labour, starvation, witnessing executions, separation from their family, and dislocation to a new country, it was found that after 5 years, 50 per cent (of 40) had full PTSD diagnoses (Kinzie et al., 1984; Bocchnlein, 1987).

Etiology

The etiology of PTSD combines the interactions of many factors, including the type of stressor, the personality of the individual involved, and the social environment of the traumatic and post-traumatic period (MacFarlane, 1989). Unlike most other diagnoses in DSM-III, the definition of PTSD
includes a stressor of such severity as to produce significant symptoms of distress in most people. Though necessary, the stressor by itself is usually insufficient to develop the disorder. That is, aside from extremely prolonged catastrophic conditions such as living in a death camp, most individuals experiencing a trauma do not develop the disorder.

**Stressors**

Stressors of different types and durations contribute to the etiology of PTSD. The stress must be outside the range of common experiences, which excludes simple bereavement, chronic illness, business losses, marital conflict, or divorce. The trauma includes rape or assault, military combat, natural and manufactured disaster (airplane crashes, auto accidents, industrial accidents) and deliberate violence (torture and death camps). The stressor may cause a physical injury such as head trauma or malnutrition, but as stated in DSM-III-R, it must also cause a psychological trauma such as a serious threat to one's life or family, the destruction of one's home, or the sight of dying or mutilated bodies. The victim of physical violence is one of the clearest examples and most frequently suffers PTSD. The severity of the stressors is recorded on Axis IV (DSM-III-R, 1987) is usually extreme (physical or
sexual abuse) or catastrophic (concentration camp or devastating natural disasters). (DSM-III-R, APA 1987).

Traumatic events vary in intensity and duration, may affect individuals or groups, or can have manufactured or natural causes. All of these factors influence the development of the disorder and the chronicity of its course. Some events such as an isolated physical assault occur in a single brief episode. Others, such as combat experiences, involve several episodes over a longer duration. Death-camp and some POW experiences are of severe intensity over a prolonged time. Those of greater severity and longer duration regularly produce a high occurrence of PTSD with a more chronic course.

Group trauma is often more complicated. The large number of people involved in a catastrophe can mean seeing bodies, witnessing mass destruction, and watching (or reading) media coverage. Grieving family members or friends complicate the primary disorder; loss of family or community support prolongs the trauma. Catastrophe (earthquakes, floods) that wipe out entire communities, or a genocidal death camp may destroy the entire social and cultural fabric of a community, greatly impairing the ability of individuals or the social group to recover. Manufactured disasters regularly produce a
higher prevalence of PTSD than do natural disasters. (DSM-III, APA 1980). Symptoms of guilt concerning others, rejection by others, and humiliation are more common in manufactured disasters than in natural disasters. Manufactured disasters can result from acts of both commission and omission. Acts of commission are direct, conscious, violent acts. Acts of omission such as toxic flooding from ill-maintained dams or poor safety standards at industrial sites are the result of attempts to save money or time and often result in slower recognition of the event, ill-defined anger at "the system", and prolonged, complicated legal processes (Grinker, Spiegel, 1945, Horowitz, 1974).

Participation in abusive violent acts (defined as unlawful behaviour) was found to be related to the denial pattern of PTSD but not to the reexperiencing symptoms. Witnessing abusive violence resulted in the reexperiencing symptoms but not the denial. (Laufer and Collegious, 1985).

**Individual Factors**

The idea that a person is predisposed to PTSD harks back to the concept of traumatic neurosis in which a patient's response to a current trauma was considered to be a reactivation of prior unresolved conflict. But establishing premorbid
personality functioning is difficult after a trauma has occurred, thus making predisposition difficult to determine (Horowitz, 1986). In severe trauma such as death-camp experiences, the disorder is so common that the syndrome seems to be the result of the trauma itself. In a group of Vietnam POWs whose precaptivity personalities were known, the post-traumatic psychiatric problems did not correlate with pre-existing problems (Hunter, 1978). A study of rape victims, however, showed that poor long term recovery was related to poor premorbid adjustment (KilPatrick, 1989). In a study of five fighters exposed to a bush fire disaster, the intensity of the exposure or losses sustained did not predict development of PTSD. However, introversion, neuroticism, and past history were premorbid factors associated with chronic PTSD (McFarlane, 1989).

Trauma in early life may increase the symptoms in response to later trauma. Psychiatric patients have an increase in symptoms in response to trauma; however, this usually takes the form of the previous disorder -- not a new PTSD per se (Brill, Beebe, 1955). The individual's defensive style determines how a specific trauma is perceived and what coping mechanism is used. Similarly, for combatants, the subjective perceptions of the meaning of combat influence the
perception of realistic dangers and reactions to war (Solomon et al., 1987). A sense of personal responsibility, either real or exaggerated can promote or maintain PTSD symptoms. Among families of patients with chronic PTSD, familial psychopathology was found in 66 per cent, with alcoholism, depression, and anxiety the most common. The probands with anxiety showed more similarity to the patients than did the probands with depression, indicating that chronic PTSD may be genetically related to generalized anxiety (Davidson, Swartz, Stork et al., 1985).

PTSD can occur at any age (DSM-III, 1980 and DSM-III-R, 1987), with the young adult years the most prevalent. Several studies now indicate that the syndrome exists in very young children, even those who are pre-verbal, but they tend not to exhibit psychic numbing or visual flashbacks. Reenactment occurs in post-traumatic play and such children, often have a foreshortened view of the future (DSM-III, 1980). An earlier traumatic event may become apparent only in old age. There are several reports of World War II veterans in their 60s who, probably as a result of age, illness, or losses, developed PTSD from traumatic events that occurred more than 30 years earlier (Solomon, Weisenberg, Schwarzwald et al., 1987).

Many soldiers in their formative adolescent years
developed strong group cohesion in the military. Because such groups can be severely disrupted by death and injury, the younger soldiers were more vulnerable to PTSD. One study found that soldiers who developed PTSD had an average age of 18.3, whereas the average age of a control group without PTSD was 21.5 (Solomon et al., 1987).

Other work with veterans found that those with high PTSD scores also had high hypnotizability scores and high imagery scores, indicating that people with excellent hypnotic potential may be susceptible to PTSD (Spiegel et al., 1988).

The associated features of PTSD, especially among Vietnam veterans, include behavioural abnormalities such as aggressive-impulsive behaviour and substance abuse. Some evidence indicated that these disorders were common in those with PTSD before enlistment and were not related to PTSD and the war experience (Sierles, Chen, McFarlane et al., 1983).

Social Factors

Often trauma can affect a large group of people and their social environment, which further impairs recovery. Disasters such as airplane crashes or floods imply multiple trauma, with sights and sounds of death and destruction. The
media's attention and publicity can lengthen the trauma but also mobilize public support and sympathy for the victims. When the social environment is destroyed, as in mass flooding with severe loss of life, the loss of the community adds further demoralization. The normal helping community network disintegrates, and individuals are often left to cope on their own (Merbaum, 1977). Some traumas are so severe (Nazi death camps, Cambodian concentration camps, or civil wars of attrition) that the entire cultural structure breaks down. Traditions, cultural models, or leaders can no longer handle the processes of grief, and an existential or religious meaning to life can also be lost.

In mass disasters, especially natural disasters, some groups find short-term cohesion and acceptance by others. If help and intervention materialize slowly, doubts about society's genuineness may cause disillusionment and social disintegration, with further individual withdrawal. After a disaster, family and friends tend to be protective and often limit contact — even that which may be beneficial. This group solidarity offers social support but also limits necessary treatment.

More social support leads to a better outcome for the victims. Some highly symptomatic victims with PTSD had smaller social networks, fewer social
contacts outside family circles, and more negative emotions towards family members. The specific symptoms of PTSD, however, such as a diminished interest in activities or feelings of detachment (numbing), may mean that a traumatized individual does not perceive or use the available social support (Barrett and Mizes, 1988; Solomon et al., 1988).

Society's attitude towards the victims trauma may play a large role in their recovery. Some events evoke positive attitudes towards victims (hostages), whereas other events are received by the public with disinterest or apathy (refugees of the Central American conflict). Still other events induce public hostility (perceptions of some Vietnam veterans about the Vietnam War). Negative attitudes of society or friends can exacerbate the original trauma (Kinzie et al., 1984).

Psychological Factors

Sigmund Freud (1939) and other early analysts made several attempts to explain the symptoms and course of traumatic neurosis. An early formulation contended that trauma revived the original childhood neurosis through regression. Later an energy model was postulated in which strong external trauma caused a disturbance in the organism's energy. The "stimulus barrier" or "protective shield" was exceeded.
Defensive mechanisms, such as repression of the event and undoing (in dreams and compulsive repetition of the trauma), were the ego's attempt to cope with the event and to drain off excess energy. Fixation on the trauma was important to this theory. Severe trauma with chronic course and poor response to treatment may lead to two unmodifiable ego changes: ego exhaustion and changes in the ego-superego boundary as a result of overwhelming of guilt and shame (Freud, Moses and Monotheism, 1939).

Other analysts revised the concept of a stimulus barrier, from a passive to an active total attempt by the ego to protect against traumatization: The trauma must be understood in terms of the individual's psychic reality and how the person interpreted and reacted to the experience. Psychic trauma may result in the individual's being overwhelmed with emotion and becoming terrified of the uncontrollable element of the emotion. The central role of affect in the theory explains such phenomena as affective blocking and alexithumia or chronic depression (Titchener and Ross, 1974; Gediman, 1971).

An information processing model has been proposed that regards human cognition as having the central role. The concept of information overload can replace energy overload: a person will remain in a state of stress until the information
has been processed. Emotions are seen not as drives but as responses to ideational incongruities and motives for defence and coping behaviour. Overload symptoms can occur in two phases, alternating with each other: the intrusive phase (unhidden and flooded images, hypervigilance, startle reactions, labile emotional behaviour, and compulsive repetition predominating after a breakthrough of the difficult-to-complete stress-related information process) and the numbing phase (a defence against these images and its anxiety, characterized by denial, selective inattention, constriction of thought and sense of numbness, and social withdrawal). This reduces the cognitive processes and anxiety but may not prevent the return of the intrusive phase. A premorbid personality style greatly influences the defences used in coping, for example, but hysterical or obsessive styles offer much different responses (Kaplan, Sadock, C.T.P., 1989).

One investigator working with victims of severe disasters involving death used a paradigm that emphasized symbolization in human cognition (McFarlane, 1988). Severe trauma interrupts the individual's participation in life activities and affects that person's ties to the human community. Mental life is strongly related to symbols of death anxiety and death guilt (self-condemnation for surviving). These death imprints prompt other images of destruction and
impairment of symbolization, resulting in psychic numbing —
decreased capacity to feel and maintain the continuity of life. At a more philosophical level, disaster epitomizes the tenuous-
ness of life and always-present possibility of death (Kaplan, Sadock, CTP, 1989).

The psychological theories of PTSD have evolved as a
response to the evolution of psychodynamic theory in general
and recognition of the complexity of the symptoms and the
chronicity of course. Clinicians working with mild or discrete
trauma have emphasized the dynamic, cognitive, and defensive
aspects of the response to trauma. Others who have worked with
victims of massive trauma have emphasized more permanent ego
changes, affective regression, and psychic numbing as core
psychological reactions (Kaplan, Sadock, CTP, 1989).

Biological Factors

The symptoms of PTSD are combination of biological and
psychological factors. The earliest symptoms following or
during a trauma are the result of a large autonomic sympathetic
discharge to a realistic fear. Hyperactivity, increased heart-
rate, increased respiration, sweating, muscle tension,
vigilance, and overwhelming anxiety occur acutely and can
persist if the stress is extreme or constant. Increasing
evidence has shown that a variety of biological factors as well as autonomic activity are an important part of the disorder's chronic symptoms.

Reactions to a traumatic event may represent a conditioned emotional response. Studies of patients with chronic PTSD when confronted with combat sounds showed regressed, highly emotional responses. This conditioned response of fear, rage, or despair was accompanied by increased pulse rate, systolic blood pressure, and muscle tension, all suggestive of central adrenergic hyperactivity. Patients with PTSD, on hearing personal scripts of combat experiences, had higher physiological responses measured by heart rate, skin conductance, and frontalis electromyogram than control subjects (Pallmeyer, Blanchard and Kolb, 1986). This is supported by other studies that show a sustained urinary, catecholamine elevation in patients with chronic PTSD (Roger et al. 1987 and Blanchard et al., 1991, Pitman et al., 1987).

Sleep studies of PTSD patients, although a small sample, have shown long sleep latencies, shorter latencies to the first rapid eye movement (REM) sleep period, decreased deep sleep, and a high REM density during REM sleep. These resemble primary depression. Traumatic nightmares, in comparison with lifelong nightmares, tended to occur earlier in the sleep cycle,
were replicas of actual events, and were accompanied by gross body movements. They occurred in varying stages of sleep and were not confined to REM stage. The autonomic arousal in some stages of sleep may be associated with the affect of the trauma and may be responsible for the replicative traumatic nightmares (Wilmer, 1986; Burstein, 1985).

Endogenous opioid peptides, which have anxiolytic action and reduction of aggression and feelings of inadequacy properties, may be produced as part of a biological response to psychological as well as physical trauma. Reexposure to the trauma may produce an endogenous opioid peptide response which result in a subjective sense of calmness or control. This may also explain why some victims continuously seek out situations that remind them of the trauma. When the traumatic stimulus is stopped, there may be a subsequent reduction in endogenous opiates that produce symptoms of opiate withdrawal. These symptoms, probably mediated by CNS noradrenergic hyperactivity, include anxiety, irritability, hyperalertness, insomnia, and startle response. Thus, endogenous opiates as well as CNS noradrenergic hyperactivity may be involved in the combination of intrusive and hyperactive symptoms of PTSD. From the same line of reasoning, clonidine (Catapres), an $\alpha-2$ adrenergic agonist that has been used to treat opiate withdrawal, has
been useful in some symptoms of PTSD (Van der Kolb, 1984; Kolb, 1987).

Urinary free cortisol levels were found to be low and stable in patients with PTSD. This was surprising, as the patients displayed overt signs of anxiety and depression and had signs of chronic increased sympathetic nervous system activity, as shown by elevated urinary catecholamines, it was thought that specific psychological defence mechanisms, especially denial, caused a selective inhibitory effect on the pituitary adrenal cortical system. This finding points the way for future research and offers a possible hormonal criterion as an adjunct to clinical diagnosis (Mason, Giller and Kosten et al., 1986).

The new preliminary research of the biological aspect of PTSD offers new hypotheses for understanding the syndrome. Highly emotional arousal states can be conditioned from the original traumatic stimulus and reactivated by a stimulus that resembles the original trauma. This may be mediated by CNS nonadrenergic and sympathetic nervous systems. The arousal state that occurs in times of emotional distress or sleep could also bring back the traumatic images. Endogenous opiate withdrawal could help maintain the syndrome. Specific
psychological defences may inhibit the pituitary adrenal axis (Van der Kolb, 1984).

Biological research which has offered some understanding of complex psychophysiological interactions, has suggested specific treatments, and may offer a new diagnostic test.

Clinical Sign and Symptoms

PTSD can begin anytime after the occurrence of the stressor, but the full syndrome does not usually occur immediately. Anxious or depressed affective states may develop soon after acute trauma, and emotional constriction may predominate in chronic trauma. Typically, weeks, months, and sometimes years follow the trauma before the full syndrome is expressed.

The most typical feature of PTSD is the persistent reexperiencing of the trauma. This usually includes intrusive thoughts or dreams that are unwanted and often fearful and are often replications of the traumatic events with the full, associated emotional reactions. The distressing recollections occur without an environmental stimulus of the event. The dreams wake up the patient in terror sometimes several times every night. The reexperience may be a sudden acting or feeling as
if the traumatic event were recurring and can include a feeling of reliving the experience. Illusions, hallucinations or dissociative (flash back) episodes are examples of reliving in trauma.

The patient may have a physiological reactivity or intense psychological distress at exposure to a stimulus that resembles an aspect of the original trauma. Examples of this are the reaction of a German concentration-camp victim on seeing a swastika or a Vietnam veteran on seeing a television news report on fighting in Indochina.

For a diagnosis of PTSD, either persistent avoidance of stimuli associated with the trauma or numbing of responsiveness—neither of which was present before the trauma—always occurs, and in addition, two of the following symptoms must occur: deliberate avoidance of thoughts or feelings associated with the trauma, deliberate avoidance of situations that would arouse memories of the trauma, numbing experienced by psychogenic amnesia, diminished interest in significant activities, feelings of detachment or estrangement from others, or restricted range of affect with inability to have loving feelings. The avoidance of traumatic stimuli may be a problem in the original interview, as the patient may avoid giving any history about the trauma. Psychogenic amnesia may also be
present and make it impossible to get full history of the trauma. Numbness can severely impair interpersonal relationship with families, and marriages may disintegrate without overt concern by the patients. Indeed, many state that they receive no enjoyment from any activity or have any emotion towards it. Sometimes avoidance and construction of responsiveness are the most obvious signs of chronic PTSD.

In addition to reexperiencing the trauma and avoiding stimuli or numbing, two persistent symptoms of arousal must be present for a diagnosis of PTSD: There can be difficulty falling asleep, irritability or angry outbursts, difficulty concentrating, hypervigilance, and exaggerated startle response. The sleep disorder is often accompanied by terrifying nightmares. The irritability and anger often further compound the social impairment caused by numbing. Difficulty concentrating and complaints of cognitive problems such as poor memory and attention are often found in those who suffered severe trauma in death-camps, where head trauma and malnutrition compounded the psychological trauma. The startle reaction can be extremely disabling when any unexpected noise will produce an involuntary motor movement.

Other symptoms have also been associated with PTSD. Although survivor guilt was listed in DSM-III but not in
DSM-III-R, persistent guilt feelings associated with surviving a trauma in which others died or suffered severely are often present. In some cultures the concept of shame is a more public sense of dishonour or disgrace and more adequately describes the emotion. The recognition that certain events or places may evoke intrusive symptoms can result in public behaviour that greatly restricts the person's life. Depressive symptoms are common. Some symptoms such as sleep disorders, difficulty concentrating and decreased interest in previously pleasurable activities are included in the diagnostic criteria of major mood disorder. In many reports, including those of traumatized refugees, the two diagnoses frequently coexist.

Symptoms of anxiety with tremors and restlessness are common. In some disaster studies, generalized anxiety disorder commonly coexists with PTSD. The irritability and anger may increase to the point of aggression and violence. Explosions of violent behaviour have been reported in war veterans, although it is unclear how much this is related to their prewar experiences (Feldmann, 1988; Yoger, 1976; Yesavage, 1983; Rosenheck, 1984). The increased drug and alcohol use reported in Vietnam veterans has been described as self-medication to decrease symptoms and autonomic hyperactivity (Boman, 1986; Behar, 1987; Behar, 1984). There are dramatic reports of war veterans reenacting or reliving the war experiences, sometimes
involving a dissociative state with weapons and violent acts duplicating actual battle events. A malignant form of PTSD involves violent, explosive behaviour, social ostracism and isolation, extreme self-loathing, and a vivid and persistent reexperiencing of psychological war trauma. This has been associated with high degrees of death immersion in Vietnam and a family background in which affective experience was discharged through action.

PTSD can occur in very young children, but some features may be different from those in adults. Children tend not to exhibit psychic numbing but may show a diminished interest in significant activities. Children may not be able to report on this, and so such information must come from other observers. Although visual flash-backs are rare, disturbing dreams may occur soon after the trauma and may generalize into terrifying nightmares. Young children do reexperience the past but reenact the trauma through action in repetitive post-traumatic play. A unique symptom of PTSD in children is a foreshortened view of the future. This may include the feeling of dying young, or pessimism about getting married or having a career. Omen formation, a belief in the ability to prophesy negative events, has also been noted.
Differential Diagnosis

Although PTSD can follow a severe trauma, not all trauma victims suffer from PTSD. Indeed, other psychiatric disorders have been more commonly reported after some disasters. Although the diagnosis can be suspected in victims of a disaster, other diagnosis must be considered. The symptoms are subjective, require a careful history, and overlap with those of other major psychiatric diagnoses. The presentation may be complicated by secondary problems such as drug abuse, alcoholism, chronic pain or rage, and violent attacks. The latter may result in severe legal problems during which a psychiatric evaluation is requested (Laufer et al., 1985).

Many studies have indicated that a high percentage (upto 80 per cent) of patients with PTSD had one or two concurrent psychiatric diagnosis. Depression has been commonly found, as many symptoms of PTSD overlap with those of depression (Anderson, N.C., 1985; Nace, E.P., 1977). When the diagnostic criteria are met for a mood disorder, that diagnosis should also be made.

Anxiety symptoms are common following a trauma (Laughlin, 1956; Riply Wolf, 1941 and Kluznick and Speed et al., 1986). Even though PTSD is listed as an anxiety disorder,
if the criteria for generalized anxiety or phobic disorder are met this should also be listed.

Many patients with PTSD have difficulty with concentration and attention and have memory problems. These could be the results of severe physical trauma (head injury, starvation) accompanying the psychological trauma, and, therefore, a diagnosis of organic mental disorder should be considered (Frenzi, 1969). Organic mental disorder differs from PTSD in that no organic factors can be found to explain the cognitive disturbance. In cases in which the history and examination indicate the presence of an organic brain disorder (OBD), both diagnoses should be given.

Adjustment disorders are a major differential diagnostic consideration. In adjustment disorders, the stressors are less severe and more in the range of common human experiences. The symptoms of reexperiencing are usually absent in an adjustment disorder, and the course is less chronic, persistent and debilitating than in PTSD (Horowitz, 1986).

Because the symptoms of PTSD are subjective and have received some publicity, and with the Vietnam War fading in clinicians' memory, it is not surprising to have reports of
factitious Vietnam PTSD (Sparr, L., Pankratz, L.D., 1983). Some involve elaborate stories of combat by people who were never in Vietnam. Some cases represent factitious medical illness (Munchausen's syndrome). Other motives are expectations of monetary compensation, relief from criminal responsibility, and guilt about not serving in Vietnam. In some ways this diagnosis is similar to compensation neurosis, which contains the belief that an injury or accident should result in financial compensation. In contrast, PTSD is outside the patient's conscious control and persists despite any compensation received (Laughlin, 1956).

Some psychological testing may be helpful, but not decisive, in making the diagnosis of PTSD. The Minnesota Multiphasic Personality Inventory (MMPI) has shown several typical scale elevations in veterans with PTSD, including schizophrenia (SC) and depression (D) as the highest scales and high elevations in the frequency (F) and psychasthenia (Pt) scales. But these profiles may suggest more severe psychopathology than actually exists. A 49-item MMPI subscale also was developed to evaluate combat-related PTSD. The Impact of Event Scale (IES), a self-rating psychological test, was developed to rate the intrusive and denial symptoms following a traumatic event (Keane et al., 1984; Merbaum, 1977).
Prognosis

Although DSM-III-R does not divide PTSD into acute and chronic categories, such a dichotomy probably has clinical uses. Acute disorders have symptoms that either develop within 6 months or do not last longer than 6 months. Both instances tend to offer a more favourable prognosis. Most acute symptoms usually clear spontaneously after a mild stressor such as an auto or industrial accident (DSM-III, APA 1980). Studies of the Mt.St.Helens Volcano disaster showed that the number of disorders greatly decreased between the first and second year. Recovery is aided by a healthy premorbid personality, good social supports, and the absence of ongoing physical or medical problems (Shore, Tatum and Vollmer, 1986).

When symptoms are present for more than 6 months, the disorder is chronic; the prognosis is less favourable and may result in severe impairment. Impairment is not necessarily related to the present symptoms or type of symptoms. Some individuals with marked symptoms seem to function well in many areas of life, but others with few overt symptoms are socially and vocationally impaired. The avoidance behaviour may appear phobic and cause patients to avoid activities or events that even symbolically resemble the original trauma, thereby
severely restricting the patient's personal and professional life. Emotional constriction and depression may lead to social isolation, drug abuse, or suicidal actions. The degree of impairment is noted on Axis V of DSM-III-R, which specifies the global assessment of functioning.

With severe stressors, more people tend to develop chronic PTSD. According to some reports, the incidence has increased several years after combat or the catastrophic event. Many reports have indicated that chronic reactions to trauma are enduring. A 20-year follow-up World War II combat stress-reaction patients showed that their symptoms of startle reactions, nightmares, irritability, and depression had worsened since the end of the War (Archibald and Tuddenham, 1965).

World War II and Korean War follow-up studies showed persistent psychoneurosis sequelae in many POWs. Depression was found to be significantly higher, 40 years after World War II in POWs than in controls (Farber, Harlow and West, 1956).

Many studies have reported persistent effects of the Nazi concentration-camp experience. These effects include most symptoms of PTSD as well as depression and, in some, chronic aggression. Even for those who have seemingly made a
successful life adjustment, the success was based on a single-minded pursuit of their goal, but with little satisfaction (Chodoff, 1963, Sigal et al., 1973). In a 40-year follow-up of POWs that used DSM-III criteria, of those who had PTSD, 29 per cent had fully recovered; 39 per cent still reported mild symptoms; 24 per cent had improved but still had moderate residual symptoms; and 8 per cent had not recovered or had deteriorated (Kluznik, Speed et al., 1986).

The author's work with Cambodian refugees who were severely and chronically impaired by PTSD found that a majority had improved significantly (most on the intrusive symptoms) in 1 year. Over the next several years, however, everyone had at least one major relapse with a full return of symptoms. After treating over 50 patients for 2 to 4 years, it was clear that PTSD symptoms are cyclical. Some symptoms such as nightmares, irritability, startle reaction, depression can improve, and the patient seems much less impaired, although avoidance symptoms remained. But after a loss (death of family or friends or increased stress, the symptoms usually return fully for a time (Kinzie et al., 1984; Boehnlein, 1987).

Others have reported symptoms reactivated or delayed 30 years after the original trauma. After the trauma, there is often a chronic relapsing in which some or all symptoms are
latent but can be reactivated by further trauma, loss, or stress. This may make the patient's response to treatment temporary at best or refractory in the severe cases (Kolb, 1983).