NEUROBIOLOGICAL ASPECTS OF POSTTRAUMATIC STRESS DISORDER

A Thesis Submitted For Partial Fulfillment
of Master Degree in
Neuropsychiatry

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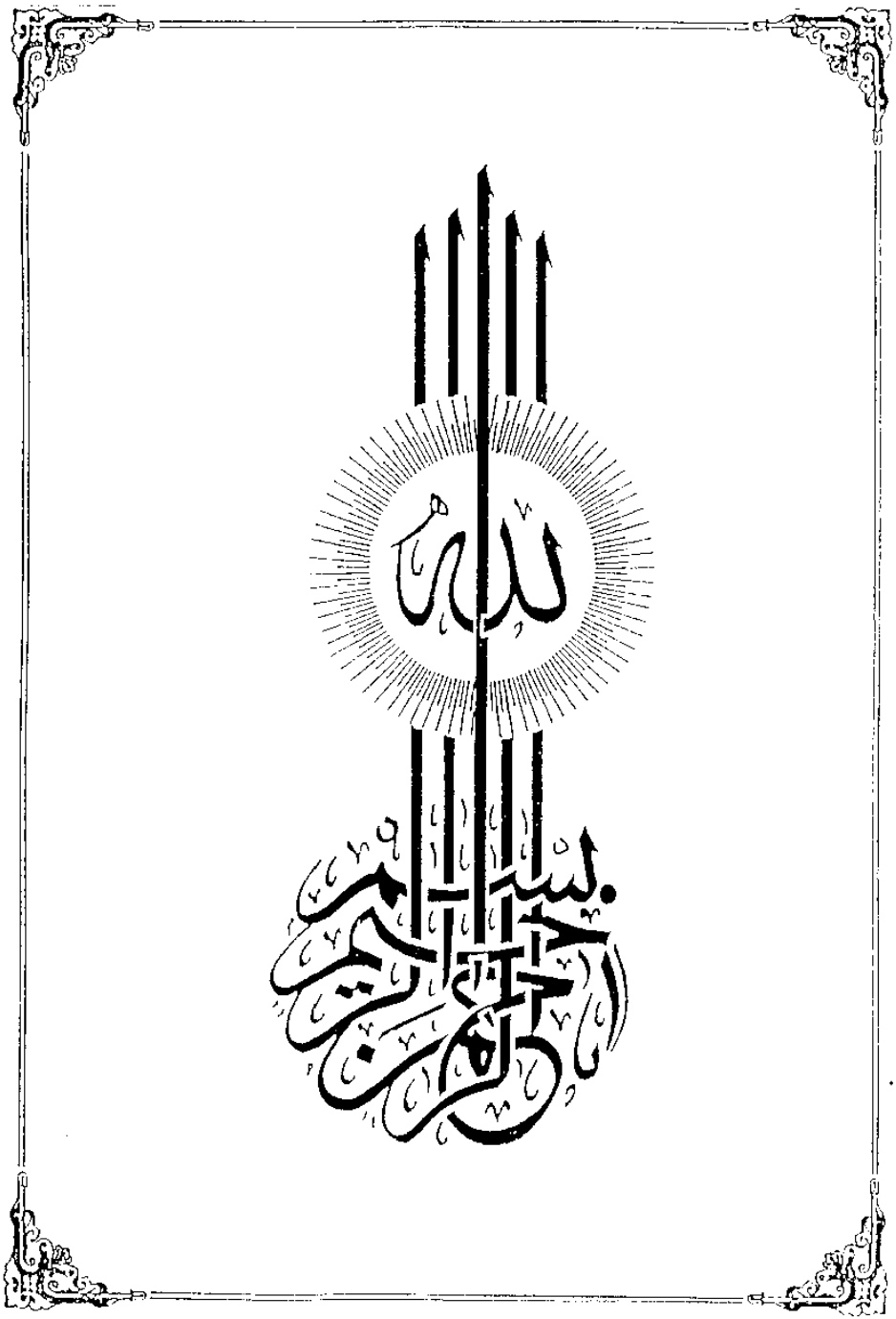

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Posttraumatic Stress Disorder (PTSD) is a disorder of considerable prevalence, often characterized by high morbidity, treatment resistance and a chronic course. The core symptoms of Posttraumatic Stress Disorder include persistent reexperiencing and reliving of the traumatic event in intrusive memories (flashbacks) or dreams, avoidance of stimuli associated with the traumatic event and autonomic hyperarousal, arising as a response to a stressful event of an exceptionally threatening or catastrophic nature *(Charney et al. 1993).*

It has been just over a decade since the inclusion of Posttraumatic Stress Disorder in the Third Edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM- III). During this period there has been a rapid growth of interest in this diagnosis among clinicians and researchers *(Pynoos 1993a).*

In the general population, Posttraumatic Stress Disorder (PTSD) has a 1% life time prevalence *(Helzer et al 1987).* Estimates among veterans of war are much higher, with 15% of Vietnam theater veterans currently meeting criteria of PTSD, and 30% meeting life time criteria *(Kulka et al. 1990).* Following the Mexican earthquakes, 32% of the victims displayed PTSD *(dela Feunte 1990).*

Despite the high prevalence of Posttraumatic Stress Disorder, there has been comparatively little research directed towards understanding its pathophysiology. Studies that focus on elucidating the neurobiological changes that occur in the brain following severe psychological trauma are only now being initiated *(Southwick et al. 1993)*. The pathophysiology of Post-traumatic Stress Disorder may involve dysfunction of several brain structures particularly the amygdala, locus coeruleus and hippocampus, as well as the noradrenergic, dopaminergic, opiate and corticotropin-releasing factor neurochemical systems *(Charney et al. 1993)*.

Studies of the neurobiology of Posttraumatic Stress Disorder, in addition to increasing our understanding of the underlying mechanisms of Posttraumatic Stress Disorder, will lead to advances in treatment *(Friedman 1988)*.

AIM OF THE WORK

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1.To review the Diagnostic Criteria of Posttraumatic Stress Disorder in the International Classifications of Diseases (ICD) of WHO and the American Diagnostic & Statistical Manual of Mental Disorders (DSM).

2.To review the Neurobiology of Posttraumatic Stress Disorder.

3.To review the recent approaches of management of Posttraumatic Stress Disorder.

HISTORICAL BACKGROUND

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Posttraumatic Stress Disorder has been known under various names, especially throughout military history, including the "irritable heart of the soldiers", "combat war neurosis", "traumatic neurosis" and "shell-shock". These terms represent a complex of symptoms making the soldier unfit for further military combat and undermining the striking power of army units *(Gersons and Carlier, 1992).*

Documented war experiences have provided an early description of the disorder among soldiers during the American Civil War (1861-1865). A combat soldier with palpitations and chest pain was said to have a functional cardiac disturbance which was called "soldier's heart" or "effect syndrome". *(Kinzie, 1989).*

During World War I (1914-1918), the British Army reported that no fewer than 7%-10% of the officers and 3%-4% of other ranks suffered mental breakdowns. In all, 80,000 shell-shocked troops passed through the army hospitals. They were diagnosed as suffering from "shell-shock". On the grounds of this diagnosis, 200,000 troops were exempted from further active service. The English psychiatrists working on home ground, with the English Channel separating them from the front line, came to the conclusion that the traumatized soldiers exhibited signs of hysteria and suggested that the best remedy was to ignore it and not to talk about it. Unfortunately, but not surprisingly, this led to consolidation of the symptoms. The shell-shock symptoms were anything but temporary, and the soldiers affected often simply disappeared into psychiatric institutions. In their despair at the unknown sickness before them, some doctors claimed that the cause lay in

micro-sections of exploded bombs having entered the brain (which is where the term shell-shock comes from). It was not long, however, before others came forward with data showing that shell-shock also occurred in soldiers who had never experienced any kind of explosion, which meant that this direct explanation had to be abandoned. Other researchers suggested that there was no question at all of a mental disorder but of simulation and it was even proposed that these so called patients should be imprisoned or summarily executed *(Gersons and Carlier, 1992)*.

Theories of the disorder's psychological etiology began to compete with physical causation theories in the early 1900's. Under the influence of the psychodynamic theory, war neurosis was viewed as the result of the reactivation of an unresolved conflict in a predisposed individual *(Kinzie, 1989)*. In other words the war trauma had served primarily to open a tin of libidinous worms, and had been reformulated in the terms of the patient's early childhood emotional war with members of the family *(Stone, 1985)*.

World War II (1939-1945) brought further clinical experience not only with combatants 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 night club "Coconut Groove" fire of 1941 with 500 fatalities, listed general nervousness, irritability, fatigue, insomnia and nightmares. Both physical and psychological causes of the resulting disorder were discussed *(Kinzie, 1989)*.

Kardiner, (1941) drew together the disparate reports describing the effects of war trauma and recognized a

common syndrome which he termed "traumatic neurosis of war" or "physio neurosis" and characterized this state as having the following five features:

1. A persistent startle response and irritability;
2. Continued pre-occupation with the trauma;
3. Explosiveness and aggressive outbursts;
4. Nightmares;
5. Constriction of interpersonal and social activities.

Since the pioneering observations of *Kardiner*, other investigators have described similar symptom clusters in civilian trauma victims. *Lindemann (1944),* published an article in which he presented a vivid account of the psychological consequences of the disastrous Boston night club "Coco-nut Groove" fire of 1941. He said that the survivor suffered from "Acute grief syndrome" which was a psychic disorder evident in people without any significant psychic problems who had previously managed to function quite satisfactorily. It seemed that many of the grief stricken individuals had various symptoms including:-

preoccupations with "visions" of the fire and the deceased
aggressive reactions
psychosomatic and behavioral disorders

none of which had been apparent in any of these people before. They also appeared to withdraw, react in a hostile manner to others, and engaged in actions harmful to themselves. Those grief symptoms were thought to arise as a direct result of confrontation with a catastrophe 'outside' the individual. *Lindemann (1944)* initially thought a period of four to six weeks might be enough for this process to take its course.