Chapter 16

CHRONIC POST-TRAUMATIC STRESS DISORDER

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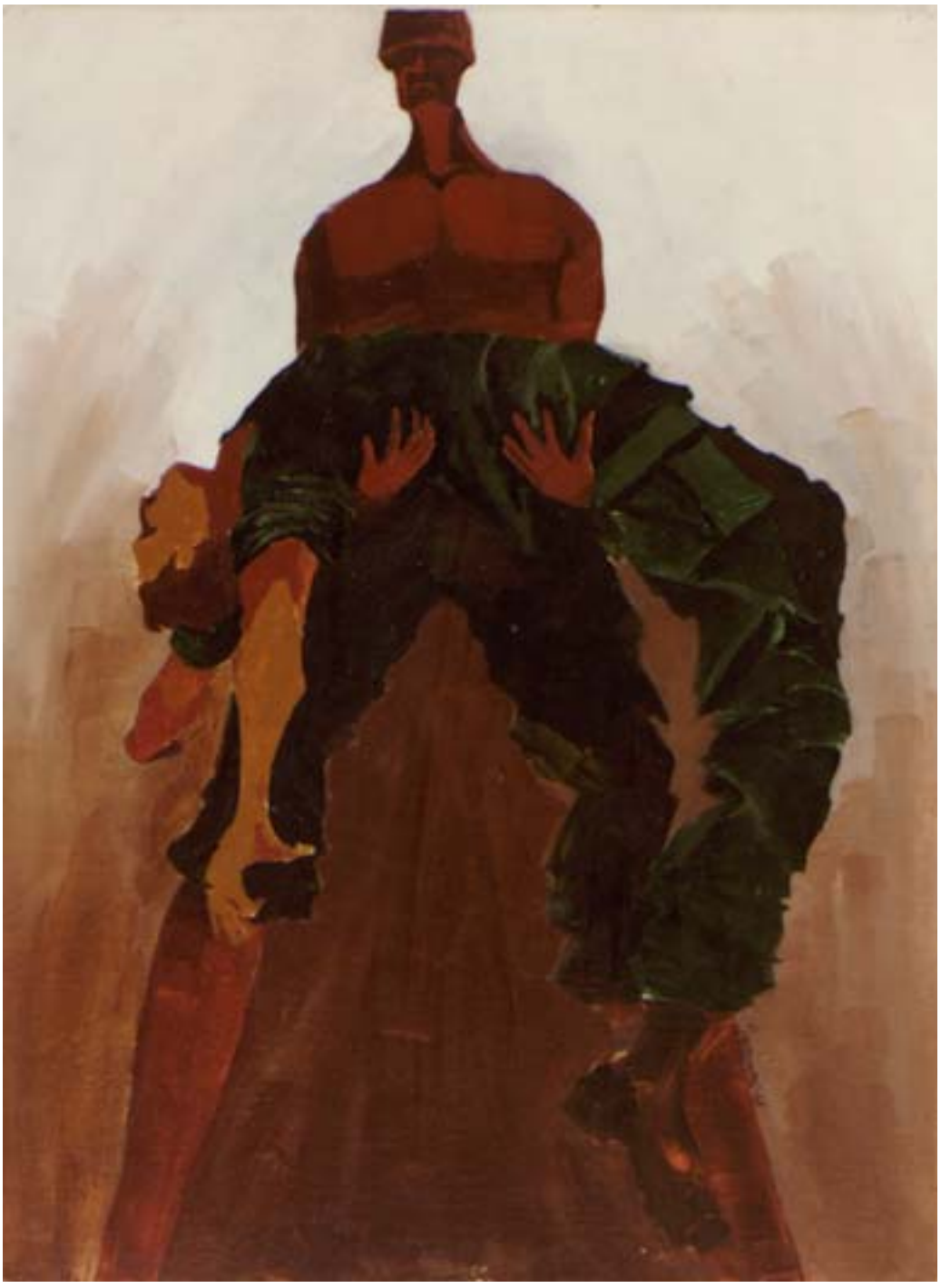

David N. Fairrington *Long Binh* 1968

David N. Fairrington was a member of the U.S. Army Artist Team #6 and was in Vietnam from February through June of 1968. In this striking visual presentation from that conflict, Fairrington captures the essence of every soldier's worst nightmare—carrying the body of his dead buddy and fearing that he himself may be the body being carried. Post-traumatic stress disorder is a complex of symptoms most often predominated by flashbacks and repetitive nightmares of this nature.

Art: Courtesy of US Center of Military History, Washington, DC.

INTRODUCTION

Post-traumatic stress disorders (PTSDs) comprise the majority of stress disorders associated with the trauma of combat, either of the acute, chronic, or delayed type. Combat fatigue may be considered a form of acute PTSD in its original understanding. Chapter 1, Psychiatric Lessons of War, describes this in greater detail. The chronic and delayed forms of PTSD have assumed considerable importance as sequelae of combat in Vietnam and in the 1982 Lebanon War.[1] The specific criteria for a diagnosis of PTSD, as delineated by the American Psychiatric Association's descriptive and nontheoretical *Diagnostic and Statistical Manual*, Fourth Edition (DSM-IV),[2] are presented in Exhibit 16-1.

History

Modern theories of PTSD begin with the 19th century concept of traumatic neurosis. Railway accidents from the middle of the century had seen the development of increasing litigation by injured persons suffering from pain and paralysis. The new specialty of neurology initially attributed these apparent neurological deficits to spinal cord injury; however, clinical and autopsy evidence began to accumulate, revealing little correspondence between tissue destruction (usually absent) and degree of disability. It was recognized that "railway spine" was a functional disorder. Charcot's[3] demonstrations of the production of paralysis and other symptoms in "hysterical" women suggested to Freud in 1893 a psychological etiology of hysteria. Charcot retained his belief in a neurological cause of hysteria and its manifestations. This was the prevailing idea. In 1889 Charcot's student, Oppenheim,[4] coined the term "traumatic neurosis" to describe what he thought was a "molecular derangement" of nerve tissue. Initially Freud accepted this idea, postulating with Breuer in their classic work, *Studies in Hysteria*,[5] an organic "hypnoid state" that made one vulnerable to hysterical symptoms when stimulated by a traumatic event. Freud believed that the traumatic event in hysteria was sexual. Later, when evidence accumulated that cast doubt on the presence of actual sexual trauma, he postulated that a fantasized sexual trauma could produce hysteria.[6] Later Freud attributed war neuroses to conflicts in ego structures (ego, id, superego) and instinctual drives (libido, destrudo).[7]

The idea that psychological trauma could produce apparent physical disabilities became generally recognized, especially with the appearance of numerous "shell shock" casualties of World War I. The pendulum swung from considering those with traumatic neuroses as neurological cases to considering them to be of purely psychological causation. Eventually traumatic neurosis was mostly subsumed under conversion or somatoform disorders but a large group, whose symptoms took the form of mood and behavioral disturbances, did not fit this categorization.

The first edition of the American Psychiatric Association Diagnostic and Statistical Manual of Mental Disorders published in 1952 (DSM-I)[8] included combat reactions under Gross Stress Reaction that corresponded in the *International Statistical Classification*[9] 1948 revision to Acute Situational Maladjustment. In DSM-I Gross Stress Reaction was to be reserved for "conditions of great or unusual stress" in which "a normal personality may utilize established patterns of reaction to deal with overwhelming fear."[8(p40)] These were differentiated from neurosis and psychosis on the basis of "clinical history, reversibility of reaction, and its transient character."[8(p40)] In terms of prognosis the following was stated: "When promptly and adequately treated, the condition may clear rapidly. It is also possible that the reaction may progress to one of the neurotic reactions. If the reaction persists, this term is to be regarded as a temporary diagnosis to be used only until a more definitive diagnosis is established."[8(p40)] The diagnosis was stated to be "justified only in situations in which the individual has been exposed to severe physical demands or extreme emotional stress, such as in combat or in civilian catastrophe (fire, earthquake, explosion, etc.)."[8(p40)] In many instances this diagnosis applied to previously more or less "normal persons who have experienced intolerable stress."[8(p40)]

The second edition of the Diagnostic and Statistical Manual (DSM-II, 1968)[10] substituted the term Adjustment Reaction of Adult Life for Gross Stress Reaction. This was in the general category of Transient Situational Disturbances, which were defined as follows:

> This major category is reserved for more or less transient disorders of any severity (including those

EXHIBIT 16-1

APA DIAGNOSTIC CRITERIA FOR DSM-IV 309.81 POST-TRAUMATIC STRESS DISORDER

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> of psychotic proportions) that occur in individuals without any apparent underlying mental disorders and that represent an acute reaction from overwhelming environmental stress.[10(pp48–49)]

It is further stated in terms of prognosis that, *"If the patient has good adaptive capacity, his symptoms usually recede as the stress diminishes"* (author's emphasis). If, however, the symptoms persist after the stress is removed, the diagnosis of another mental disorder is indicated."[10(p49)]

This is a most unfortunate change because one gains the impression that therapy should be aimed at removing the individual from the stressful environment; in fact, a brief respite from the stressors is needed, but removal too far produces chronic symptoms, and the object of treatment is rapid return to the high-stress environment. Furthermore, it implies that outcome is dependent only on the individual's innate adaptive capacity rather than requiring therapeutic interventions to permit that adaptive capacity to recover. Contrast this impression with that given in DSM-I: "When promptly and adequately treated, the condition may clear rapidly."[8(p40)]

In the third edition of the Diagnostic and Statistical Manual (DSM-III),[11] published in 1980, the clinician may place the combat stress reaction in the Adjustment Disorder category specifying the presentation (depressed mood, anxious mood, etc.) or may choose the Post-Traumatic Stress Disorder category. In the former, outcome as in DSM-II is stated to be dependent on removing the stressor: "It is assumed that the disturbance will eventually remit after the stressor ceases."[11(p299)]

Post-Traumatic Stress Disorder, among other criteria, lists "a recognizable stressor that would evoke significant symptoms of distress in almost everyone."[11(p238)] By dividing these disorders into acute (duration of symptoms or onset of symptoms, less than 6 mo following the onset of the traumatic event), chronic (duration of symptoms 6 mo or more) and delayed (onset at least 6 mo after the traumatic event), the impression is given that one is dealing with a lengthy disturbance due to psychological trauma.

In DSM III-R,[12] the 1987 revision of DSM-III, and DSM-IV,[2] the 1994 edition, there is a requirement for symptoms to last longer than a month. Presumably this was intended to make a distinction from transient adjustment disorders; however, this requirement introduces an unnecessary disjunction to the clinical and theoretical understanding of PTSD as an exaggeration of normal responses to psychic trauma. It does underscore the fact that the eventuation of chronic symptoms suggests perhaps persistent biological changes.

DSM-IV adds a new category, Acute Stress Disorder, for similar symptoms that occur during or soon after the trauma, last for at least 2 days, and cause clinically significant distress or impairment. If this persists beyond 4 weeks, it becomes Acute PTSD. This category corresponds reasonably well to those stress (battle fatigue) casualties who require "restoration" at medical holding facilities (clearing stations) for 2 to 3 days. It also covers those who require "reconditioning" for 7 to 14 days (or up to 4 wks) further to the rear. The DSM-IV also notes that "some symptomatology following exposure to extreme stress is ubiquitous and often does not require any diagnosis." This could apply to those battle fatigued service members who can remain in their own small unit or be given 1 to 2 days of rest in a nonmedical support element, or who recover and return to duty with only 24 to 36 hours of treatment at a forward medical (clearing) company. The DSM-IV has also shortened the onset time of chronic PTSD to 3 months post-trauma, although "delayed onset" is still after 6 months.

In summary, DSM-I, having been published shortly after the Korean conflict and based in large part on the U.S. Army nomenclature growing out of World War II experience, retained the correct concept for battle fatigue, which was placed under Gross Stress Reaction. In fact, the description of Gross Stress Reaction was almost an exact reiteration of that for Combat Exhaustion given in TB Med 203, the War Department Technical Bulletin, Nomenclature and Method of Recording Diagnoses, published 19 October 1945.[13] DSM-I was heavily influenced by the psychobiology of Adolf Meyer and the experiences of World War II psychiatrists.[14] DSM-II, however, was published in 1967, over 15 years after the end of the Korean conflict, the last conflict in which large numbers of battle fatigue casualties were seen. The Vietnam conflict was in its early stages; however, very few battle fatigue cases were produced primarily because of the low-intensity nature of combat and other factors.[15,16] Consequently the treatment lessons implicit in labeling did not have an urgent, emotional reality to the authors of DSM-II.

The authors of DSM-III were well aware of the effect of labeling as can be seen in the use of "schizophreniform" instead of "schizophrenia" but lack of familiarity with battle fatigue cases again led to a failure to understand the treatment implications of the labels involved.

Some of the thought involved might have been due in part to the need to view Vietnam veterans as having been damaged by their experiences in Vietnam and as appropriate recipients of psychiatric care. Such concern might have led to the acceptance of post-traumatic stress disorder (PTSD) and delayed post-traumatic stress disorder (DPTSD) for a larger cohort of behaviors (such as addictive and aggressive acts) than had previously been considered as sequelae of psychological trauma. Unfortunately, such labels hold potential untoward consequences for the perception, diagnosis, and treatment of the varied stress disorders that can be generated in combat and that are amenable to rapid intervention, very brief therapy, and quick restoration to duty. DPTSD must be seen as a special case that undoubtedly involves more complex historical factors both pre- and post-combat than the usual stress responses to the trauma of combat.

The DSM-IV category of Acute Stress Disorder helps to restore a distinction between the transitory reactions to extreme stress and more persistent symptoms, "Acute" may also have fewer negative connotations than DSM-I's use of "Gross," although "gross" does imply more than a trivial response.

Proposed Nomenclature for the Military

The following guidance was given when the author was Psychiatry and Neurology Consultant to the U.S. Army Surgeon General. It encapsulates ideas on proper nomenclature for combat psychiatric casualties, drawing from the *Manual of International Statistical Classification of Diseases, Injuries and Causes of Death*[9] (also known as ICD-9):

> Psychiatric combat casualties consist of a unique group of military patients for whom the diagnosis has strong possibilities for adversely affecting recovery. The term "battle fatigue" is ideal in that it suggests a nearly normal response, is relatively nonspecific in allowing for labeling of the great variety of symptom syndromes known to occur, and most importantly conveys an expectancy of rapid resolution. The disadvantage is that many psychiatric casualties occur so soon in combat that fatigue cannot reasonably be presumed to be a factor. Policy will be that patients in whom fatigue can reasonably be considered a factor will continue to be diagnosed as battle fatigue while those in whom fatigue cannot be so considered will be diagnosed transient battle reaction. Both terms should be considered roughly equivalent, should be treated similarly and will be coded with ICD-9 number 308.4 (mixed disorders as reaction to stress). Avoidance of technical terms that could be regarded as diagnoses (eg, "anxiety," "conversion," "paralysis") is desirable. Two examples follow:
>
> 1. Unwounded soldier presenting with tremor, tachycardia, sweating, paralysis of right arm, and glove anesthesia of right hand ten minutes after observing a friend killed in the first hour of battle:
> *(Axis I) 308.4 Transient battle reaction* manifested by numbness and weakness of right arm and hand, sweating, and rapid pulse.
> 2. Unwounded soldier developing fatigue, tremor, tachycardia, sweating, paralysis of right arm, and glove anesthesia of right hand following 36 hours of sustained combat exposure.
> *(Axis I) 308.4 Battle fatigue* manifested by fatigue, numbness and weakness of right arm and hand, sweating, and rapid pulse.

Subsequently, in current doctrine, the distinction between battle fatigue and transient battle reaction was abandoned. The rationale is that fatigue, by definition, is impaired performance due to doing something too long *or* too hard. As S.L.A. Marshall observed, fatigue or exhaustion can be brought on very rapidly by extreme fear. Anticipatory anxiety, as well as physiologic strain can bring on battle fatigue even before the battle starts. Therefore, the one term, battle fatigue, suffices. As operations other than war, such as disaster relief or peacekeeping in high stress conditions have increased while combat has decreased, the terms "contingency fatigue" and "conflict fatigue" have also been proposed.

ETIOLOGY

Psychiatric theories of etiology generally derive from the cultural or scientific zeitgeist. Ancient Egyptian healers, noting almost exclusive incidence of hysteria in women and being well-versed in anatomy, assumed that the multiple somatic symptoms of hysteria were due to migration of the uterus, a theory that held sway until the medieval Catholic Church, emphasizing the conflict between Satan and God, attributed hysteria to possession by evil spirits. After Isaac Newton revolutionized science with his theory of universal gravitation, Anton Mesmer began treating hysteria with magnets

thought to have effects similar to planetary bodies. In a contest with the renowned exorcist Father Gassner, Mesmer demonstrated the superiority of his "scientific" approach over the older theory of possession.[17]

Likewise, in an era in which the intelligentsia accepted Charles Darwin's concepts of the evolution of increasingly complex structure and behavior based on the survival of animals with the best instincts, Freud saw various neuroses as resulting from instinctual drives clashing with reality. Thus, those with hysteria suffered from unfulfilled sexual wishes and those with obsessions and compulsions suffered from expressions of or defenses against anal eroticism and aggression. In this view, psychological trauma could cause anxiety symptoms due to the activation of unacceptable sexual and aggressive wishes. While temperament varied, the psychological conflict was considered paramount. Until the late 1970s psychological explanations of PTSD etiology, usually based on psychoanalytic or learning theories, predominated.

Gradually, perhaps presaged by the Watson-Crick discovery of the molecular structure of DNA, biological explanations of causality in mental disorders have gained hegemony. The concept of hysteria has almost disappeared except as a cluster of personality traits; and obsessive-compulsive disorders are viewed by many as the survival in some persons of instinctual grooming and other social behaviors of our mammalian ancestors, often best treated with medications.[18] Concerning PTSD, this biological supremacy has emphasized the physiological and neural aspects.

Biological Models of PTSD

Patients with chronic PTSD present with "positive" symptoms such as anxiety, tachycardia, muscle tension, shortness of breath, insomnia, irritability, and exaggerated startle response, which have been postulated as arising from conditioned autonomic activation to innocuous stimuli.[19–23] PTSD is also characterized by "negative" symptoms such as diminished interest in formerly significant activities, interpersonal detachment, restricted affective range, and a feeling of foreshortened future. These symptoms have been likened to the animal model of learned helplessness.[24]

Biological models of PTSD have emphasized the role of noradrenergic systems in the brain (primarily the locus ceruleus and its projections), which are activated by situations of alarm or trauma, the fight-flight reaction of Cannon.[25] Such "trauma centers" in the brain could be conditioned by threatening environmental events or stimuli associated with threat (conditioned fear stimuli) to respond to innocuous situations with PTSD symptoms.[26]

Drugs that inhibit noradrenergic brain systems have been used to treat stress symptoms including those of PTSD. These include clonidine, β-adrenergic blocking agents (propranolol), antidepressants (which downregulate β-adrenergic receptors), and benzodiazepines (GABA facilitators).[26] Many substances abused by persons with PTSD may be attempts at self-treatment because they share the ability to inhibit noradrenergic systems, at least temporarily. These include alcohol, benzodiazepines, barbiturates, and opiates.[26] The effectiveness of serotonergic attenuating agents (such as buspirone, a partial mixed serotonin 1A/1B receptor agonist) in treating anxiety disorders suggests serotonergic excess theories of anxiety as well.

Stress-mediated changes in neuronal structures of lower animals suggest that PTSD could be associated with fundamental and long-lasting modifications, including alterations in neuronal structure and gene expression.[26] Treatment, therefore, must often be intensive and prolonged and preventive measures should be the first approach.

While traumas cannot be prevented in conflicts, it is noteworthy that not all those exposed to severe traumas develop PTSD. In animal experiments of inescapable shock or stress (ie, the learned helplessness model of PTSD and depression)[27] those animals that could gain control over stress presentation and the severity, duration, and repetition of the aversive stimulus did not develop learned helplessness. The presence of a supportive peer and previous escape experience have protective effects in animals though biological and social vulnerabilities are factors.[26]

Studies also revealed that animals given antidepressants, clonidine, and benzodiazepines did not develop learned helplessness when exposed to inescapable stress.[27] Substances often abused by PTSD sufferers (stimulants, barbiturates, ethanol, and chronic use of benzodiazepines) were ineffective in reversing learned helplessness once it developed; however, antidepressants, clonidine, and buspirone, had a normalizing effect in animal studies.[26]

In summary, while older theories emphasized psychological trauma or conflict and conditioning aspects of PTSD etiology, more recent investigators have emphasized lasting neuronal changes and behavior in traumatized animals, postulating a hyperadrenergic state with hypercortisolism and physiological arousal to innocuous stimuli that re-

semble the original stressor. Others[28,29] have pointed out the aspect of repetition of the trauma manifested by intrusive thoughts, nightmares, and even hallucinations, thus implicating memory systems as paramount. The author has emphasized a multifactorial etiology or biopsychosocial model of chronic PTSD.[30]

A Biopsychosocial Model of Etiology

Chronic PTSD symptoms develop in those with social and biological predispositions in whom the stressor is meaningful when social supports are inadequate and the symptoms are maintained because of subsequent inadvertent reinforcement of the maladaptive behaviors.

Following both World War I and World War II, large numbers of combat veterans were treated in Veterans Administration hospitals for chronic "war neuroses." Many of these former soldiers had broken in combat and had been evacuated, never to rejoin their comrades. In many such soldiers a dynamic was set up that produced increasing disability. The dynamic developed as follows: (*a*) the soldier was conflicted over almost instinctual urgings to leave the combat arena to secure personal survival, battling with his own concepts of duty, honor, and responsibility to his comrades requiring him to remain in combat, (*b*) medical symptoms developed offering an honorable route out of combat, (*c*) the symptoms were accepted as a legitimate reason for leaving and the soldier was evacuated, (*d*) the soldier experienced guilt for abandoning his comrades because at some level he did not accept the legitimacy of his symptoms, (*e*) the symptoms became strengthened and exaggerated because of the soldier's need to prove to others and himself that he was really disabled and legitimately left combat, and finally, (*f*) any reproach either from internal guilt or from external doubt as to the medical necessity for his symptoms resulted in further strengthening of the symptoms.

Such a dynamic would explain the development, progression, and persistence of symptoms in the improperly treated acute post-traumatic stress casualty who becomes chronically disabled; but how can one explain the development of delayed PTSD symptoms, often occurring years after combat exposure? Such cases suggest that the dynamic described may represent only a special case of a more pervasive condition. Based on his study of psychiatric casualties in the 1973 Yom Kippur War and the 1982 Israel-Lebanon War, Belenky[31] has postulated that psychiatric casualties form a spectrum ranging from immediate ("battle shock") through acute ("combat fatigue") to late (chronic and delayed PTSD) combat stress reactions. In each case the etiopathogenic element is combat stress. The distinctions are based on certain intrinsic (personality, prior adjustment) and extrinsic (degree and quality of trauma, presence of ameliorating influences) factors. This conceptualization complements Marlowe's battle ecologies scheme, with the latter focusing on environmental factors (combat intensity).[32]

In Belenky's conceptualization, delayed PTSD results from the traumatic process itself, depending on degree of trauma, and develops somewhat independently of subsequent events. The author's view of the development of delayed PTSD is slightly different. While he agrees that a psychologically traumatic event will result in PTSD symptomatology, he would emphasize the contingent nature of the maintenance of, or delayed appearance of, disabling symptoms. As with the dynamic described earlier, *acute* post-traumatic symptoms are maintained and become chronic by their reinforcing value in preventing guilt or admonishment for the soldier's evasion of combat responsibilities. This has sometimes occurred because of improper or absent treatment.

The *delayed* PTSD syndrome, however, has a slightly different history. In these cases the soldier has experienced a traumatic event with variable degrees of subsequent symptoms that eventually disappear and may not even be remembered. Often, these soldiers performed without obvious impairment at the time, perhaps by denying fear or grief. After a symptom-free interval, the former combatant again experiences environmental stress. Such stress may or may not resemble the stress of combat; however, it evokes anxiety symptoms that usually are similar to those of combat. This similarity evokes memories of combat trauma and even produces in some instances reaction patterns similar to combat. These symptoms are reinforced in a variety of ways, including the concern of friends, justification for acting out otherwise unacceptable feelings, sustaining the patients' indignation over being abused by society, and monetary reinforcement (Veterans Administration [VA] pension).

Within the past decade the belief has developed that delayed and chronic PTSD are more common following unpopular conflicts. This belief is based on experience with U.S. veterans of the Vietnam conflict and more recently with Israeli veterans of the 1982 Lebanon War. In the Vietnam instance, estimates as high as 700,000 or 25% of Vietnam veterans were given as suffering from chronic

PTSD.[33,34] Other estimates as high as 60% of combat veterans have been given.[35] A more accurate figure for Vietnam appears to be 17%,[36] less than the 25% psychiatric disabilities given for World War II veterans. In the Lebanon instance studies[37] have revealed that two thirds of Israeli psychiatric casualties have been of the chronic or delayed PTSD type. This is exaggerated because all Lebanon War veterans reporting to a mental health facility were labeled as suffering from PTSD even though they had prior psychiatric diagnoses including manic-depressive disorder.[38]

Despite the possible exaggeration of PTSD prevalence, some relationship between unpopular wars and chronic and delayed PTSD seems to exist. Goodwin[33] has identified the following variables as producing chronic PTSD in Vietnam veterans: it was a teenage war (average age 20 for combatants); there was a fixed tour (unrealistic expectations after return to states); the ideological basis of the war was unclear (saving the corrupt South Vietnamese "democracy" from the North Vietnamese, not Soviet or Chinese Communists); the enemy was hard to identify (sometimes the enemy appeared to be civilians—including women and children); there was widespread use of illicit drugs (especially heroin and cannabis); tranquilizing drugs were first used in combat (may have suppressed symptoms of stress); administrative discharges were frequent (often for drug abuse) and were only temporary solutions to stress; and the rapid return to the United States did not allow for decompression, with the returnee often being met with a hostile or indifferent homecoming. This may be a reflection of the lack of societal support for wars of this type. They are frequently ambiguous with a large percentage of the population indifferent or hostile to the war. Such wars are usually prolonged beyond the expectations of the initiators of combat and the patience of the populations, and they often include actions against civilians.

PTSD can be conceptualized as a special case of a broader mechanism by which the mental apparatus handles aversive stimuli. Freud's postulate, described shortly after World War I, of a repetition compulsion in which the organism replays the psychic trauma in an attempt to gain mastery, may play a role, especially with the early symptoms.[7] This replaying may take the form of dreams or nightmares, recurrent memories, or even hallucinatory "flashbacks" of the traumatic event. The sufferer may be preoccupied with "if only I had (or hadn't…)" thoughts. Other mechanisms such as positive reinforcement (secondary gain in Freud's model) seem more important in the chronic maintenance of symptoms. The emergence of delayed symptoms may be explained on the basis of an association between the current situation and an aspect of the traumatic situation. Ullmann and Krasner[39] have used the term "redintegration" for such evocation of behavior more appropriate to an earlier life event. Whatever the theory of causation, PTSD symptoms appear to be relatively universal given a severe enough stressor; however, it is not the presence of symptoms but the psychological purposes they serve that determines the degree of disability.

PTSD PRESENTATIONS

Diagnosis, treatment, and prevention of PTSD will be considered in the context of the following cases, which illustrate some of the features of chronic and delayed PTSD. Diagnosis, treatment, and prevention of *acute* PTSD have already been described in Chapter 1, Psychiatric Lessons of War, and Chapter 2, Traditional Warfare Combat Stress Casualties, in terms of combat fatigue or combat stress reactions. The following two cases, known to the author, illustrate some of the features of *chronic* and *delayed* post-traumatic stress, ranging from normal memories which are not a disorder to disabling PTSD.

Case Study 1: Just Bad Dreams

June K., now 60 years old, has never sought mental health care despite mild, chronic post-traumatic stress symptoms. Typically she develops nightmares after seeing a war movie or when undergoing unusual psychological stress such as the death of a family member. The nightmares awaken her and her husband who reassures her; then she falls asleep without further incident.

The psychic trauma that she experienced occurred at the age of 18 when she was captured by the North Koreans when they invaded Seoul in 1951. From a prominent South Korean family, she and her parents and siblings had been targeted for capture, torture, and death. Knowing this, the family had dispersed throughout Seoul. June K., the oldest child, had found work under an assumed name in order to buy food for her family (forced rationing and use of North Korean money prevented purchase of food with family resources). She was turned in to the North Koreans by a collaborator who had recognized her from newspaper photographs. After several days' captivity, which included occasional beatings, she escaped when the compound was bombed by U.S. planes.

She hid for several weeks until Seoul was recaptured by UN forces.

June K. served as a laboratory technician with U.S. forces until the war ended; then she came to the United States on a Fulbright Scholarship. Other than rare nightmares about her war experiences, there are no other symptoms and no apparent secondary gains.

Comment: This person exhibits typical mild chronic post-traumatic stress symptoms that do not appear to serve any adaptive role in her current functioning. Her symptoms are evoked by associations with the traumatic event.

Case Study 2: The Assassination Witness

While the author was consulting in the psychiatric unit of a military hospital in an Arabic country in 1985, he interviewed a 54-year-old army major, formerly a Warrant Officer, with 32 years of active duty. The major was married with five children. He dated the onset of his symptoms to an incident several years earlier when he was present at the assassination of the leader of his government, having been invited by his oldest son to see him march in the parade.

Sitting near the leader, he initially thought that the firing of weapons was part of the normal demonstration of support for him. When he recognized the hostile nature of the firing, which killed the head of state, the patient ran in a panic about 3 miles, collapsing at the gate to his military unit. He stated that ever since that time he has had headaches, nightmares, and giddiness. Physical exams and neurological studies revealed no apparent physical basis for his symptoms.

The symptoms persisted roughly a year at which time he was admitted to a hospital where he remained for another year. In the hospital he was found to be severely depressed but not suicidal. He also had severe anxiety, trembling, elevated heart rate, complaints of irritability, and dreams of airplanes bombing. He had also become angry with his oldest son, who had urged him to attend the ceremony. On one occasion he had even started choking him. Psychometrics revealed that he had average intelligence and neurotic symptoms, primarily a depressive disorder with hysterical personality. He was treated with thioridazine, lorazepam, and psychotherapy. His request for a medical separation from the military was denied.

Past history revealed that he had similar symptoms in the 1956 Arab-Israeli War when, as an officer candidate at the military academy, he was in a building that was bombed, causing it to collapse. He remained terrified underneath his bed for hours until he was dug out. Shortly thereafter, a bullet from a second air raid narrowly missed him. Afterwards he collected bullets as souvenirs. He had a period of nightmares and anxiety following these episodes, but this did not persist beyond a few weeks.

A decade later he was an advisor to the Yemen Army during their civil war. He stated that for a period of about 6 months he was constantly panicked. Several of his fellow soldiers were ambushed and their bodies mutilated; however, he did not witness these episodes. He felt isolated, alone among foreigners. A psychiatrist who served at about the same time verified that such incidents did occur but that the biggest problem was material deprivation in an inhospitable climate. Following that tour he again developed symptoms of anxiety and nightmares that lasted for several months, but then they dissipated and he experienced no particular problems until the assassination.

Currently, the patient appeared as a middle-aged man with gray hair who was anxious and sweating profusely while recounting his symptomatology. He presented my fellow psychiatrists, some of whom had treated him, with a letter that detailed his current complaints. He could not tell us why he had persistent symptoms after the assassination, but did not after the barracks bombings in 1956 and after his Yemen tour. He did not participate in the 1967 or the 1973 wars with Israel.

The following were the patient's complaints: giddiness, especially when walking, with a tendency to be worse on the right side; tinnitus leading to irritability and sometimes violent actions toward his wife or children; numbness or tingling in his scalp, "like I had a helmet on"; general paresthesias all over the body; sleep disturbances (early morning awakening, fatigue after having nightmares, and frightening awakenings). During the day he often felt tense as if his "head were full of blood" and as if the blood in his head were "boiling." He stated that he would become irritable if in crowds or if he heard loud noises, especially shooting. He also stated that he had profuse sweating of the right hand. He described sexual difficulties (impotence for the past 3 years); pessimistic outlook (that he hadn't achieved anything in his life); withdrawal and suspiciousness of other people; impulsiveness, depression, and sadness (in contrast to formerly being friendly and energetic); anorexia with mild weight loss; and smelling something burning. (The author's colleagues pointed out that this is a common symptom among PTSD sufferers who were exposed to napalm or burning flesh in various wars; however, this patient was not exposed to those conditions, so the etiology of this symptom is obscure. Perhaps it may have been modeled after the symptoms of other PTSD patients on the wards; such "contagion" of symptoms among suggestible battle fatigue cases is common.)

During the interview the patient was very energetic and animated, sweating profusely at times when recalling his symptoms; but, at the same time he seemed to derive some relief or even pleasure from sharing those symptoms and his suffering. His physician said that this patient, despite the diagnosis on psychological testing of a hysterical personality, actually had many features of an obsessive-compulsive personality (having been meticulously clean, very organized, very attentive to details, and somewhat rigid in interpersonal relationships).

Etiologically, this patient appeared to have all of the elements of a post-traumatic stress disorder: a severe stressor (the assassination in which he could have been

killed) and recreation of this and earlier traumas in nightmares and perhaps the smelling of something burning (which could have been reminiscent of the gun powder at the time of the assassination—an intrusion of the trauma into the present). He also displayed the explosive irritability and aggressiveness as well as the withdrawal from social contact that are often found in PTSD. The irritability in the presence of loud noises, particularly the firing of weapons, has some components of a startle reaction.

In terms of treatment, group psychotherapy should be considered in this case although the lack of patients with PTSD symptoms from the assassination episode would weaken this approach. Usually group therapy works best when all group members have been exposed to a similar stressful situation.

Past individual psychotherapy apparently had focused on ventilation and supportive treatment. It had also emphasized work because he was denied compensation and a medical separation. Such an emphasis on "here and now" issues is desirable.

Comment: This patient exhibited a plethora of symptoms that, while typical of chronic PTSD, are seldom all found in one person. His symptoms appear to serve a current adaptive role in his functioning in making him a focus of attention and sympathy and excusing him from some military duties. Pharmacotherapy in such cases is often quite rewarding. The patient had received some anxiety relief from the thioridazine and lorazepam; however, he continued to be troubled by a multiplicity of symptoms. Recent studies have shown that the use of benzodiazepines beyond a few months may actually be countertherapeutic due to the development of tolerance effects.[40] He had not yet been treated with antidepressants, particularly monoamine oxidase inhibitors (MAOI) or the tricyclics. Although the mechanism of action of these agents is unknown (their effectiveness might be related to suppression of dreaming or REM (rapid eye movement) sleep (and thus nightmares), due to a general antidepressant effect or due to a specific anxiolytic action related to downregulation of β-adrenergic or serotonin 1-A receptors), these antidepressants often produce dramatic relief of symptoms. The author recommended a trial of phenelzine (a hydralazine-type MAOI—Nardil) in a dose of up to 90 mg per day. If there were problems with his use of MAOI, perhaps dietary restrictions not being enforceable, or other problems such as hypotension, then a trial with a tricyclic, probably imipramine, was recommended. At the time, selective serotonin reuptake inhibitors (SSRI) were not available. Currently, a trial of an SSRI might be helpful.

The following case, provided by Dean A. Inouye, M.D., reveals an aspect of personality, alexithymia, that may play a role in chronic PTSD. Alexithymia (literally "inability to read emotions") was described as a characteristic of some patients who appear unable to properly interpret emotions in themselves and others.[41]

Case Study 3: Chronic PTSD and Alexithymia

First sergeant (1SG) MC is a 43-year-old married white male with 22 years of service, self-referred for feelings of distress following his involvement in a shooting incident. In July 1991, 1SG MC and a security officer had attempted to evict an occupant from the company barracks. The occupant produced a gun and, without warning, shot 1SG MC in the chest and killed the security officer. The patient underwent surgery, which revealed no injury to vital structures. His post-operative course was unremarkable.

Past medical history revealed that the patient experienced three injuries during his two tours as a medic in the Republic of Vietnam: 1969, gunshot wound to right thigh; 1970, fragment wounds to right leg and head; 1971, burns to right hand and head. He had no other significant past medical history or current medications. He rarely used alcohol and denied use of tobacco or caffeine. He had no known drug allergies.

Social history revealed that he was adopted in infancy with a fraternal twin brother into an upper middle class family. He was close to both parents, did well in school, graduating from high school. He had no behavior problems and joined the Army at age 20 after 2 years of business college, "because I always wanted to." His military history was exemplary with many awards and citations.

Course: The patient first presented to outpatient psychiatry 1 month after his injury, complaining of rumination about the shooting incident, decreased appetite, and early insomnia with multiple awakenings, which he stated was "no different than when I was shot in Vietnam." He appeared anxious but his mental status exam was otherwise unremarkable. He was given a diagnosis of adjustment disorder with mixed emotional features. He was briefly tried on lorazepam (Ativan) 1 to 2 mg at bedtime to improve the insomnia. The medication was discontinued when the patient terminated treatment after three visits.

The patient returned to psychiatry 6 months after his injury complaining of rumination with depressed mood, pan-insomnia, increased appetite with 22 lb weight gain, lack of energy and initiative, pervasive anxiety, sudden crying spells and angry outbursts, decreased self-esteem and social withdrawal, and a feeling of loss of control of his emotions.

He had experienced the death of friends during the war but denied survivor guilt. After returning from Vietnam he had complained of prominent generalized anxiety; pronounced startle reaction without hypervigilance; pan-insomnia with multiple awakenings; nightmares of battle scenes and ambush; difficulty concentrating; angry outbursts; fear of flying in helicopters but no vivid recollections while awake. He received no specific treatment for his symptoms except diazepam (Valium). His symptoms gradually abated after a few years although he continued to have occasional nightmares. He believed that his present PTSD symptoms were worse than those after the Vietnam conflict. He was anxious and tearful during his exam. He was given a provisional diagnosis of post-traumatic stress disorder. He was started on doxepin

(Sinequon) 25 mg at bedtime to improve his anxiety and insomnia. Six weeks later, his treatment was terminated due to his unwillingness to attend his appointments.

The patient returned to psychiatry a year after his injury, 3 weeks before the anniversary date of the shooting incident. He complained of vivid recollections and nightmares of the shooting event, hypervigilance and easy startle, fear of situations similar to the shooting event, difficulty falling and remaining asleep with multiple awakenings, generalized irritability with occasional explosive anger, decreased concentration, and thoughts of death. He denied feelings of guilt over his survival. He expressed ambivalence about "coming for help" and fear of becoming a patient in the hospital. On exam, he was restless, with labile affect, and was frequently tearful. During all interviews he was remarkably without insight and unable to verbalize his feelings. His diagnosis of post-traumatic stress disorder was confirmed. He was given an additional diagnosis of alexithymia. He was started on fluoxetine (Prozac) 20 mg tablets once a day and clonazepam (Klonopin) 0.5 mg tablets three times a day to improve his anxiety and insomnia. The patient was placed on buspirone (BuSpar) 15mg per day, later increased to 30 mg per day.

He experienced a marked increase in his symptoms during the anniversary week of the shooting. After that, his anxiety decreased slightly and he experienced occasional nights of improved sleep, with fewer awakenings. He complained of mild daytime sedation and the buspirone was discontinued. His PTSD symptoms, in general, remained unchanged. In October 1992, buspirone 5 mg tablets three times a day was reintroduced to improve anxiety, with the goal to discontinue clonazepam and morning drowsiness. He reported gradually decreasing anxiety and improved sleep. However, his anxiety was often markedly worse on weekends and he continued to have two to three awakenings per night.

The patient continued on the above medications with clonazepam reduced to 0.5 mg at bedtime. After 2 months on the medication regimen, the patient reported significantly decreased anxiety, improved feeling of control, and improved sleep quality with fewer awakenings. On exam, his restlessness and lability of affect were improved. His other PTSD symptoms were unchanged and he could not do his work satisfactorily. He was therefore presented to a medical evaluation board (MEB) for separation from the military.

Comment: This soldier had continued to experience difficulties at work and he anticipated difficulties working in a foreign country after impending retirement. The symptoms may have represented an attempt to delay retirement and hold on to his U.S. Army identity. They may also have been an attempt to convey his feelings of disability for compensation purposes.

These case studies suggest that varying degrees of symptoms will follow a traumatic event. Whether they become disabling depends on the use to which they are put. In some circumstances, they can be highly adaptive. The Arabian major, for instance, trapped for 6 more years in a job that he did not enjoy with little chance of promotion, would have gained not only an exit from the army but also additional money for a disability separation. Furthermore, his possible envy and anger toward his upwardly mobile officer son could be justified by making him responsible for the major's current distress. June K., however, does not utilize her symptoms for current conflicts, having other adaptive mechanisms.

1SG MC functioned adequately until the time approached for his retirement from the military with the turmoil and uncertainties of a civilian life in a foreign country. The symptoms kept him his military identity and allowed him to express his distress in an acceptable manner, a common finding in alexithymic persons who cannot express feelings directly but do so with symptoms, usually physical.

One of the main methods of preventing chronic PTSD is by preventing (or properly treating) acute post-traumatic stress symptoms.[42,43] An important element in preventing acute post-traumatic stress (combat breakdown) is the presence of cohesive units or social support *during* the stressful event. Stretch[44] has shown that social support *following* the stressful event is also important in preventing or attenuating symptoms of chronic or delayed PTSD. He found, for example, that soldiers who remained in the U.S. Army (which is socially supportive of the combat role) following combat in Vietnam had significantly less symptomatology than controls who left the military following assignment in Vietnam. It is possible, of course, that self-selection accounted for some differences (with those more prone to PTSD disaffiliating themselves from the military); however, other studies[45] reinforce the importance of social support in the prevention of PTSD.

TREATMENT

The treatment of chronic PTSD, like its etiology, involves multiple modalities including emotional conditioning, cognitive restructuring, and pharmacological interventions.[46]

Psychotherapy

Individual therapy is often too intense for both patient and therapist, and group therapy with peers

is usually preferable. While some "debriefing" (abreaction and ventilation) of the original traumatic events must be expected, it is important to prevent these sessions from becoming "stuck in the past," endlessly reiterating old guilts and grudges and trying to outdo others' stories. Here, and in individual therapy if it is undertaken, the focus is on current issues as in Glasser's reality therapy approach.[47] It is usually clear to an objective observer what reinforcements are maintaining the symptoms. To help clarify this area and intervene in diminishing this reinforcement, family and other interested parties may need to be interviewed individually and conjointly. This may also reveal that the patient has minimized significant alcohol or other substance abuse. When substance dependency is clearly established, an intervention and referral for detoxification and rehabilitation in a specialized program is indicated. Self-help groups such as Alcoholics Anonymous (AA), Narcotics Anonymous (NA), and Cocaine Anonymous (CA) should play a prominent part in the treatment.

Deconditioning by teaching the evocation of the relaxation response[48] can help alleviate not only the heightened tension of the hyperadrenergic state but also the tendency to use substances for relaxation. Exercise programs can also be beneficial in this respect.

Pharmacotherapy

Since the early descriptions of barbiturate treatment of combat stress casualties of World War II,[49] a variety of medications have been used to treat acute and chronic PTSD symptoms. The first relatively effective use was reported in an uncontrolled study of five war veterans utilizing phenelzine.[50] Subsequent studies have validated the usefulness of MAOIs and tricyclic antidepressants (TCAs) in the treatment of some PTSD patients, particularly when complemented with psychosocial therapies.[51–54] More recently, the SSRIs may be useful for some of these patients.

Most reports have emphasized the presence of concurrent disorders with PTSD, particularly substance abuse, depressive disorders, and personality disorders.[52,54] These concurrent disorders often require different approaches and may prevent effective pharmacotherapy of PTSD symptoms.

For the different PTSD symptoms themselves, different medications may vary in efficacy. Antidepressants appear to improve intrusive symptoms (such as recurrent intrusive recollections, nightmares, and panic episodes) but may be less effective with avoidant symptoms (such as withdrawal, emotional numbing).[54] Intrusive symptoms as well as hostility and feelings of violence were improved by the tricyclic anticonvulsant carbamazepine (Tegretol) in 70% of Vietnam veteran inpatients in a study by Lipper and others.[55] Again, improvement in the avoidant symptoms was somewhat less. In view of carbamazepine's antikindling effect, this suggests that PTSD symptoms may arise from the effect of trauma in evoking repeated strong emotions, which result in neuronal irritability and inappropriate activation as postulated by Post et al[56] in the pathophysiology of certain mood disorders and borderline personality disorders. Antikindling agents such as valproic acid (Depa-kene) and clonazepam appear useful as alternatives to lithium and carbamazepine.

Other medications that have been helpful with some PTSD patients include the antihypertensive drugs, clonidine and propranolol, both of which inhibit noradrenergic activity though by different mechanisms.[57] While benzodiazepines would seem useful in anxiety symptoms of PTSD, they have not been systematically studied, probably because of their high abuse potential in this population. Preliminary reports of lithium therapy by Davidson et al[54] reveal encouraging results in diminishing explosiveness, irritability, mood swings, and impulsive behavior, as well as in reducing nightmares and improving sleep. Carbamazepine, valproic acid, and clonazepam appear similarly useful in these symptoms.

At this time, careful assessment and treatment of concurrent conditions and clinical trials with a variety of medications along with psychosocial interventions are warranted for most chronic PTSD patients.

PTSD can occur following any severe stressor; however, prisoners of war represent a special case in which the stressors tend to be chronic as well as severe. They merit special consideration for study and proposed interventions since the entire group suffers similar status and deprivation stressors.

PTSD AMONG PRISONERS OF WAR

Prisoners of war (POWs) frequently develop symptoms of chronic PTSD (see Chapter 17, The Prisoner of War). POWs do not necessarily develop psychiatric disorders; however, follow-up studies of POWs during World War II, the Korean conflict, and the Vietnam conflict indicate an increased risk

of such disorders.[58–60] The risk is greatly influenced by the conditions of captivity. Holocaust victims almost universally suffered from PTSD.[61] Soldiers incarcerated by the Japanese during World War II and by the North Koreans (and brainwashed by the Chinese) during the Korean conflict have had increased rates of depressive, anxiety, and psychosomatic disorders, as well as suicide. Conditions of captivity were often excessively harsh with many deaths from malnutrition, infections, and exposure.

Soldiers who had surrendered to the Japanese were treated with absolute contempt because they had violated the samurai warrior code of *Bushido,* which required the fighting man to die in combat, commit *hara kiri* (suicide by disembowelment), or request execution by the less favored method of decapitation.[62] Undoubtedly, brain syndromes caused by malnutrition exacerbated the chronic posttraumatic stress disorders arising from captivity.

During the Korean conflict, captive Americans were not only exposed to malnutrition, disease, and harsh camp conditions but also to a calculated psychological offensive aimed at breaking them that came to be called *brainwashing*. Crude coercive measures involving Pavlovian conditioning, both aversive and positive, with sleep deprivation, physical and psychological torture, and rewards for "acceptable" behavior (such as denouncing capitalism and American "imperialism," and admitting to using chemical and biological warfare) were used in conjunction with sophisticated social manipulation. Officers were separated from enlisted ranks to destroy the influence of leadership. Cohesion was destroyed by rewarding selected soldiers for informing on their fellows. Rewards of food, clothing, and medication might mean the difference between survival and death during the cold Korean winters. Information was carefully controlled; only adverse news (such as race riots in the United States) was presented to the POWs.

Critics of the POWs, not taking into account this new form of psychological warfare and hearing American soldiers denounce America or confess to fabricated war crimes, accused them of lacking willpower and indicted American child-rearing practices as producing psychological weaklings. Such criticisms were even extended to those who died of malnutrition, exposure, and illness; they were accused of having "give-up-itis," moral strength so weak that they would die rather than try to live in adverse circumstances.[63]

Based on his interviews with 20 randomly selected repatriates at the end of the Korean conflict and the work of colleagues who interviewed 300 men, Schein,[64] a researcher at Walter Reed Army Institute of Research, described what was probably the most extensive attempt to destroy unit cohesion and realign social viewpoints ever perpetrated against U.S. prisoners of war. The men were segregated according to race and ethnicity. Military rank was disregarded and the Chinese captors randomly selected small unit leaders. Informers were actively solicited and given special treatment, some of it life sustaining, such as adequate food while others were starving. Such favors were sometimes given noncollaborating soldiers to destroy confidence in them. POWs soon felt that they could not trust anyone. Signed confessions of germ warfare or other war crimes by U.S. forces were solicited and shown to POWs to attack the moral position of the United States. No group association was allowed other than Chinese-orchestrated self-criticism or propaganda lectures. Mail was censored so that only bad news was transmitted. Similarly, all news was from communist sources—press, radio, magazines, and movies. Race riots and criminal acts in the United States were highly publicized.

The postcaptivity evaluations revealed that the communists had little success in changing beliefs and attitudes; however, in producing collaboration they had been much more successful with about 10% to 15% of the men chronically collaborating in giving pro-communist lectures, broadcasting propaganda, giving false confessions, informing on fellow POWs, and so forth. Some attempted to obstruct the communists and they were generally transferred elsewhere. A few developed severe apathy, quit eating, and died. The most common response was neither collaboration nor obstruction of the communists but what the men called "playing it cool"; that is, physical and emotional withdrawal from the whole environment, developing an attitude of watching and waiting rather than hoping and planning.

Schein concluded, "Ultimately that which sustains humans is their personality integration born out of secure and stable group identifications."[64(p30)] Long-term follow-up has revealed that while many POWs improved, symptoms were often life-long.[65–67]

Following the Korean conflict, there was a great deal of media attention focused on the behavior of Korean-era POWs and measures that might be taken for the physical and psychological survival of the POW. In 1955, President Eisenhower issued the Code of Conduct[68] (Figure 16-1) that reaffirmed the basic tenets of resisting the enemy as much as possible and attempting to escape when feasible. It was and is believed that these guidelines actually pro-

THE CODE OF CONDUCT

Article 1

I am an American fighting man. I serve in the forces which guard my country and our way of life. I am prepared to give my life in their defense.

Article 2

I will never surrender of my own free will. If in command, I will never surrender my men while they still have the means to resist.

Article 3

If I am captured, I will continue to resist by all means available. I will make every effort to escape and aid others to escape. I will accept neither parole nor special favors from the enemy.

Article 4

If I become a prisoner of war, I will keep faith with my fellow prisoners. I will give no information or take part in any action which might be harmful to my comrades. If I am senior, I will take command. If not, I will obey the lawful orders of those appointed over me and will back them up in every way.

Article 5

When questioned, should I become a prisoner of war, I am required to give name, rank, service number, and date of birth. I will evade answering further questions to the utmost of my ability. I will make no oral or written statements disloyal to my country and its allies or harmful to their cause.

Article 6

I will never forget that I am an American fighting man, responsible for my actions, and dedicated to the principles which made my country free. I will trust in my God and in the United States of America.

Fig 16-1. Code of Conduct for Members of the Armed Forces of the United States. After the Korean conflict, it was realized that coercive "brainwashing" could cause even the most patriotic soldier to be induced to make statements denouncing his country. The code was originally issued by Executive Order 10631 on 17 August 1955 by President Dwight D. Eisenhower and was amended by Executive Order 12017 on 3 November 1977 by President Jimmy Carter. Each soldier is given a copy. Data source: Department of the Army, *Code of Conduct/Survival, Evasion, Resistance, and Escape (SERE) Training*. Washington, DC: DA, 10 December 1985. Army Regulation 350-30.

tect the soldier from undue guilt associated with giving in under a less stringent code. More recently, forced "confessions" and denunciations of America are ignored because they are obtained by coercion.

Most of the American POWs of the Vietnam conflict were aviation officers shot down over North Vietnam. They had succeeded in the rigorous selection and training process for aviation pilots. Not surprisingly, these highly intelligent men invented ingenious methods of resisting the enemy, communicating among themselves even though usually placed in solitary confinement, and strengthening their mental defenses. They often practiced regular physical exercise, meditation, and "mental exercises" such as remembering books, mentally building a home, and writing journals though deprived of writing materials. Follow-up studies conducted after their release revealed that they were healthier than matched controls in all physiological systems other than dental and mental health. They tended to suffer more psychological problems than the controls but the differences were slight.

The relatively small number of POWs who had been captured in South Vietnam consisted primarily of ground troops whose experiences were similar to those captured by the Japanese and North Koreans. Their postcaptivity adjustment resembled that of the World War II and Korean conflict POWs (ie, increased morbidity).

An understanding of and techniques for handling captivity stress have been developed[69–72] based on the experiences of POWs from World War II, the Korean and Vietnam conflicts as well as other groups

(USS *Pueblo* crew members captured by North Korea and the U.S. Embassy personnel taken hostage in Iran). These can be considered in three phases: (1) precaptivity training; (2) captivity adaptation; and (3) postcaptivity recovery.

Precaptivity Training

Precaptivity training is needed for anyone likely to become captive, including military personnel, embassy staff, and others. This should involve learning the expected responses to phases of captivity and methods of adapting optimally to them, and realistic role-playing for purposes of familiarization and desensitization. Generally such training will have survival value. While 30% of American prisoners of the Japanese and North Koreans died in captivity, only 15% of American POWs in North Vietnam died. Although demographic differences (higher education, older, and mostly officers in the Vietnam POW population) and a less hostile climate were predominantly responsible for fewer deaths, soldiers who became POWs in Vietnam had been given captivity training.[71]

Captivity Adaptation

Captivity adaptation generally occurs in certain stages, which have been described by Rahe and Genender[72] as follows:

Stage 1: Startle/Panic—First Seconds to Minutes

Captivity typically occurs as an abrupt transition from normal daily activities to a situation of forceful, often brutal subjugation, a situation that cannot be assimilated quickly. Captors are excitable and have an increased likelihood of killing the captives at this point, producing paralyzing fear, stunned dissociation, or panic flights in captives. Feelings of defenselessness and confusion usually follow actual capture. Successful coping involves rapidly controlling these emotions. This may be facilitated by conscious attempts to count the captors, to memorize their features, and to focus on details of the situation.

Stage 2: Disbelief—First Minutes to Hours

Denial in the form of thinking, "This can't be happening" or "I'm dreaming," may occur. Captives often believe that they will be rescued quickly and are disappointed when this does not occur. Captors engage in various dehumanizing activities such as stripping clothing and personal items, binding, blindfolding, beating, and photographing prisoners for propaganda purposes. Similarly, "confessions" may be extorted by torture for propaganda value. Captives usually cope best by turning their attention inward—thinking of loved ones, home, and freedom—because psychological dissociation from the painful situation is adaptive in this setting.

Stage 3: Hypervigilance—First Hours to Days

The emergence of increased alertness to environmental cues can be useful—attempting to keep track of time, mileage, turns of the vehicle; however, guards are usually highly attentive to possible escape attempts at this time. Generally some form of interrogation will begin with emphasis on intelligence gathering. Hypervigilance can be useful in helping the captive withhold desired intelligence, in orienting to a 24-hour cycle, in assessing the captors, and in possibly eliciting the sympathy of guards who may assist the captive in obtaining reading and writing materials and other basic amenities.

Stage 4: Resistance/Compliance—First Days to Weeks

As the captors attempt to coerce the captives into cooperating, the resistance/compliance stage begins. Interrogations change from intelligence gathering to exploitation with coercive demands for "confessions" of "crimes" or enforced public appearances, often in degrading conditions. Given sufficient physical and psychological torture, virtually anyone can be forced to cooperate with his captors. The degree of cooperation depends on the severity of torture inflicted and the captive's commitment to resist.

The techniques used by captors derive from those used by the Czarist Russian and Stalinist secret police and the state police of Nazi Germany, with refinements added by the Communist Chinese and North Koreans. They include intimidating arrest; imprisonments of indeterminate length; physical, social, and nutritional deprivation; disturbances of body rhythms; physical and sensory isolation; stressful (often brutal) interrogations; unpredictable responses from guards and inquisitors; prolonged fear of death; and attempts to "reeducate" the captive. Settings are usually cramped, filthy, pest-ridden, uncomfortably cold or hot, with poor lighting and ventilation. Communication with fellow captives, or even guards, is prohibited.

Coping in these circumstances involves attempts to keep physically fit, to give the captors just enough information (preferably hard to validate and requiring lengthy time) to prevent severe torture, and attempts to communicate with fellow captives. Religious faith, prayer, meditation, and thoughts of loved ones also play an important role in coping.

Stage 5: Depression—First Weeks to Months

As the extent of his losses (freedom, family, friends, fortune, and possibly future) becomes apparent, the captive may become depressed. The captive may show the classical signs and symptoms of depression, including anorexia, retarded speech and movements, insomnia, fatigue, guilt, self-condemnation, and suicidal thoughts or attempts. Coping is greatly aided by a strong support group, hence the need to establish communication. The captive can use his own intelligence to fight boredom, composing stories and poems, mentally constructing buildings, solving mathematical problems, etc. Captor behavior at this time is primarily custodial.

Stage 6: Gradual Acceptance—First Months to Years

The captive realizes that his captivity may be prolonged and that he must make more productive use of his time if he is to survive. Custodial behavior by captors is usually maintained although "re-education" efforts may continue. Coping behavior by captives involves living from day to day, taking each day as it comes, and attempting to maintain physical and mental stamina. Group support, if available, is extremely sustaining. Best is group creative work such as crafts, sports, and possibly escape planning.

Postcaptivity Recovery

Rahe and Genender[72] have described six stages of recovery from captivity as follows:

Stage 1. Brief Euphoria—First Seconds to Minutes

The period of elevated spirits of a released captive is usually short-lived. The captive is often mistrustful that the return from captivity may be another false hope. Celebrations may fall flat.

Stage 2: Hyperarousal—First Minutes to Hours

In contrast to the understimulation of the later stages of captivity, the released captive is overwhelmed by stimuli and mentally slowed, causing a "punch-drunk" appearance. Often sleep-deprived on their transition to freedom, the former captives may be confused and exhausted.

Postcaptivity management optimally includes a period of from several days to a week of "decompression." The ex-captive needs protection from the very intrusive media and even from relatives, because he may make remarks that he will later regret. For example, persons still influenced by the "Stockholm Syndrome" (identification with the captors) upon release have made statements favoring the aims of their captors and detrimental to the national interests of their country.

During this decompression time, thorough physical examination and correction of medical problems (eg, infections, infestations, dental care) can be accomplished. Rest and restoration of physiological deficits (sleep, food, fluids) are important just as with combat stress cases. In addition to physical restoration, the principles of proximity and immediacy indicate rapid return to the precaptivity milieu after decompression and positive expectancy. Psychiatric examination must be carefully conducted to avoid an expectation of mental illness, which can lead to a chronic "compensation neurosis." A positive expectation that the ex-captive will soon return to work is important. The psychiatric examination should be both diagnostic and therapeutic, allowing ventilation while providing reassurance.

Stage 3: Compliance/Resistance—First Hours to Days

Captives initially on release are likely to comply with most requests, having been conditioned to do so by their captors. As they regain feelings of individual power and capability, captives will begin to resist activities that appear to have little relevance to their own needs, for example, appointments, psychological testing, and intelligence debriefing. Treatment plans should take into account this emerging independence by allowing free time, the wearing of clothing other than hospital pajamas, time for group meetings with fellow captives, etc. Group cohesion can be fostered by having ex-captives eat together and engage in group "rap sessions." As with Marshall's[73] technique of debriefing troops after a battle, the reconstruction of events and correction of misperceptions can be highly therapeutic. Finally, follow-up reunions may be helpful.

Stage 4: Denial—First Days to Weeks

After an initial candidness about their problems adjusting to freedom, captives are likely to enter a phase of denying that their captivity has produced significant emotional or physical problems. While these statements should not be challenged directly, they should not be taken at face value. Arrangements for ongoing follow-up evaluations and treatment should be made, often through liaison with family members.

Stage 5: Restitution—First Weeks to Months

Attempts at restitution may take a variety of forms, such as gross obesity from overeating, problems with emotional control, and isolation to avoid overstimulation. Employers may attempt restitution by granting long vacations from work when, in actuality, return to a work routine generally helps the ex-captive reestablish feelings of self-worth. The family may attempt restitution by providing all the love and attention they could not show during the captivity. Captives may have difficulty interpreting nonverbal behaviors and the affective content of language. Families should be told to expect disconcerting responses, including crude table manners, and bathroom and sleeping habits acquired in captivity. They should also be told to expect and even encourage the ex-captive to recount his captivity experience because it seems to serve a therapeutic abreactive purpose.

Stage 6: Gradual Readjustment—First Months to Years

Follow-up studies of American POWs from Korea and from World War II Japanese prisons, in both circumstances experiences being extremely severe, revealed increased rates of infectious, cardiovascular, degenerative, and psychiatric disorders and accidental deaths compared to control subjects over the next 25 to 30 years.[72] Depending on the severity of circumstances and the individual's coping skills, some psychological scarring and premature physical disability is likely for the duration of the ex-POW's life. Ongoing follow-up for significantly traumatized individuals may prevent or attenuate disability, but some degree of post-traumatic stress symptoms is inevitable.

SUMMARY AND CONCLUSION

Post-traumatic stress disorder has come to epitomize the blaming and legalistic tendency in modern American society. It is given currency to explain the most outrageous behavior from Vietnam veterans' trafficking in cocaine and narcotics and robbing banks to a sexually promiscuous woman's attributing her behavior to trauma on a San Francisco cable car. Despite these unlikely extensions of the PTSD concept, a core of solid data exists suggesting psychic trauma as underlying much of nonbiologically generated mental illness. Such apparently disparate conditions as multiple personality disorders, panic disorders, and psychogenic depressions may result from early physical and sexual abuse. Psychic trauma occurring in older persons may lead to the development of a constellation of symptoms and behaviors that are termed PTSD. It is often unclear as to who is most responsible for these symptoms, the traumatized person or the original stressor. Attorneys make a living persuading juries and judges one way or the other.

The traumatized person's best hope is to accept responsibility for his symptoms and to develop coping methods to neutralize them. This should be the aim of psychiatric treatment.

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