

Posttraumatic Stress Disorder: A Persistent Diagnostic Challenge

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Posttraumatic stress disorder (PTSD) is unique in the nosology of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) because it is one of the only categories that clearly implies a causal agent (ie, trauma). Early reports from the Fort Hood shootings in Texas suggested that the alleged shooter was suffering from an expansion of reacting to

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trauma. Some mental health professionals and the media attempted to connect his killing of fellow soldiers to “pre-PTSD”¹ syndrome because the alleged shooter was about to deploy to the Middle East.

Therefore, careful consideration of an individual’s reaction to trauma and adversity continues to be affected by the media, sensationalism, and lack of critical thinking.

This article supports Treisman and McHugh, who suggest that further critical thinking is needed about this category.² The article concludes

with a review of a study, which used a before-and-after comparison of subjects’ reactions to a traumatic experience, highlighting premorbid vulnerabilities to trauma.

THE CONCEPTION OF PTSD Preconception Stance

Humans have always been at war. They have dealt with the stress of witnessing other humans perpetrate violence and killing, and reactions to such traumatic events have been described for thousands of years. More than 2,500 years ago, during the Bat-



Figure 1. Greco-Persian wars. During the Battle of Marathon (circa 500 BC.), an Athenian warrior saw a fellow soldier die. The warrior then became blind from emotional stress.

tle of Marathon, an Athenian warrior saw a fellow soldier die. The warrior then became blind from emotional stress^{3,4} (see Figure 1).

The concept of combat stress has long been documented in our nation's history. During the Civil War, when fighters had stress-induced chest pain, dyspnea, fatigue, and palpitations, along with gastrointestinal symptoms and insomnia; this was called "soldier's heart."⁵

In World War I, it was called "shell shock."⁶ In World War II, it was "battle fatigue" or "war neurosis."⁷ McCurdy, in an article about war neuroses,⁸ and Swank, in a related article about "combat exhaustion,"⁹ wrote about military psychiatry and combat in World War I and World War II, respectively. They describe the factors that shape the expression and course of psychological breakdown.

These include the cohesion and morale of the combat team (in other words, "group identity"); pre-war

vulnerabilities of temperament and personality in the troops; leadership's understanding of psychological reactions to combat and their skill in managing these reactions; and the psychiatric services available behind the lines. They recap that these factors have crucial influence over the outcome of combat stress.

In 1966, Glass and Berucci composed a report for the office of the Surgeon General on the relationship between prolonged combat and psychological breakdown in World War II. It was probably the first report to use good data to confirm the association.¹⁰

Charles Wilson, who was later honored with the title "Lord Moran," was Churchill's personal physician during World War II. In his book, *The Anatomy of Courage*, he writes that "Men wear out in war like clothes." This means that soldiers have a kind of "bank of courage," which, like any bank account, could be depleted, in this case by prolonged war conditions.¹¹

The modern concept of war-related trauma had political roots in the Vietnam War. In *The Politics of Readjustment: Vietnam Veterans Since the War*, Scott writes how military psychiatrists before the Vietnam War era entertained two co-existing hypotheses.¹² One was that only soldiers who were marginally adjusted before combat fell victim to PTSD; the other was that PTSD afflicted well-adjusted troops that were subjected to too much stress.

Kolb wrote *Modern Clinical Psychiatry*, which became a well-established and widely used textbook, if not the standard textbook, in the 1970s. In it, he writes about the standard approach to "Gross Stress Reactions," later known as PTSD. The term "posttraumatic" was actually used to refer to the psychiatric effects of head injuries. The standard then was to identify aspects of the situation or the event, the personality of the individual, and the social role and responsibilities they had which could explain the individual's vulnerability and resilience to a stressful event.^{2,13}

Therefore, before the conception of PTSD, two opposing views on war-related trauma existed. One was that posttraumatic stress is not a distinct mental disorder but an intense and prolonged expression of the normal reaction to trauma. The other was that PTSD is a discrete mental disorder derived from identifiable traumatic experiences. The latter view won, and in this theory, the etiology was "trauma memories," which have been "dissociated" or "repressed" and are acting and will continue to act dis-

TABLE.

Five Intrinsic Problems with the Concept of PTSD

Emotions of Adjustment	Posttraumatic stress appears to be more of a natural emotion rather than a separate entity or malady.
Disease Sponsorship	Several failed attempts have been made to promote PTSD as a disease category.
Faulty Numbers	Errors in diagnostic methods and epidemiological data in PTSD are paramount.
Reliability of Recall	There is the mystery of “dissociation” and unreliability of memory.
Politics and Medicine	PTSD can be regarded as the best example of sociopolitical influence on a medical profession.

ruptively, generating symptoms of depression and anxiety, along with an array of behavioral problems.²

Conception Stance

The modern evolution of PTSD occurred in a politically charged era of antiwar sentiment during the Vietnam War. Such antiwar feelings were strongly embraced by many mental health professionals.

In the 1970s, the “post-Vietnam syndrome” became an expansive term encompassing many different expressions of mental disorder and distress. The syndrome’s central assumption carried out by veterans’ self-help groups and their psychiatric supporters was that problematic temperaments, personalities, and behaviors were not shaping a subject’s response to trauma but were instead products of it.¹⁴

Yet there was no evidence suggesting that Vietnam veterans were so maladjusted to their situation that they were suffering from such a syndrome.

So what led to the conception of PTSD? In the 1960s, there was discontent over the American military. There were strong antiwar opinions in society, soldiers were being drafted,

and there were no open arms when soldiers returned home.² During World Wars I and II, proudly wearing the uniform in your community was met with appreciation; during Vietnam, it was met with derision.

Also during the 1970s, the American Psychiatric Association (APA) was drastically transforming psychiatric diagnostic methods

During World Wars I and II, proudly wearing the uniform in your community was met with appreciation; during Vietnam, it was met with derision.

and nomenclature, with radical revisions to the DSM-II. Authors and editors of the DSM-III were called for testimony from “experts.” These experts worked effectively on the editorial committees of the APA to insert PTSD into the manual with its encompassing acute, chronic, refractory, or delayed forms.²

With the publication of the DSM-III in 1980, PTSD became an offi-

cial and standardized classification scheme in American psychiatry.

CURRENT DEFINITION AND DSM EVOLVEMENT

In 1980, DSM-III was published and had major revisions, including explicit diagnostic criteria, a multi-axial system, and a descriptive approach that attempted to be neutral with respect to theories of etiology.^{15,16} Yet this is when PTSD, a condition that requires an etiologic factor (ie, trauma), was inserted into the DSM. DSM-IV and its text revised edition followed in 1994 and 2000, respectively. DSM-V is due in 2012.¹⁷

The DSM-IV diagnostic criteria for PTSD require that the patient must be exposed to trauma (criterion A), must re-experience the trauma (criterion B), and must persistently avoid stimuli associated with the trauma and experience a numbing of general responsiveness (criterion C). All are followed by persistent symptoms of arousal (criterion D). These symptoms should last for more than 1 month and cause clinically significant distress or impairment in functioning.¹⁸

Qualifiers in the DSM are acute (duration less than 3 months); chron-

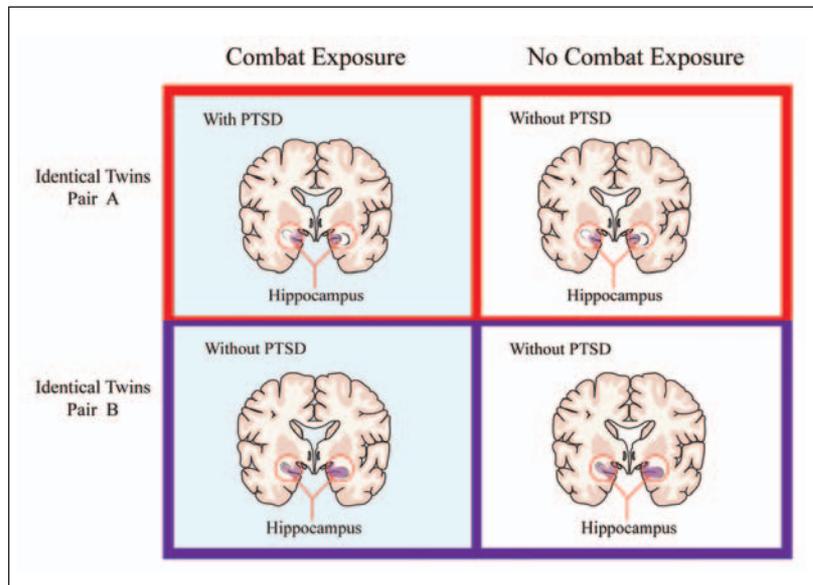


Figure 2. Hippocampal volume differences, combat exposure, and development of PTSD in monozygotic twins. Identical twins pair A have smaller hippocampal volume regardless of combat exposure and PTSD. Identical twins pair B have larger hippocampal volume and no PTSD, regardless of combat exposure, suggesting that hippocampal volume is a product of genetic and familial factors, and not a result of trauma and PTSD.²⁹

ic (duration equal to or longer than 3 months); and with delayed onset (if onset of symptoms is at least 6 months after the stressor). Some also refer to cases that are refractory,^{19,20} complicated, subdromal,²¹ and even “masked” forms of PTSD. Recent literature also cites a new entity called “secondary trauma,” which affects close associates of those suffering from PTSD.^{22,23} This perhaps implies that the condition can be contagious.

There are at least five intrinsic problems with the concept of PTSD that pose a persistent diagnostic challenge. These are:

- Posttraumatic stress appears to be more of a natural emotional response rather than a separate entity or malady;
- Several attempts to promote PTSD as a disease category have failed;
- Several notable studies illustrate errors in diagnostic methods and

data collection, leading to some faulty epidemiological numbers;

- Memories regarding traumatic events are far from reliable. The mystery of “dissociation” and the unreliability of recall will be examined; and
- PTSD demonstrates the best example of sociopolitical influence on a medical profession (see Table, page 509).

A DIAGNOSTIC CHALLENGE

PTSD is a persistent diagnostic challenge in psychiatry. Its concept has posed significant problems not just in psychiatry and medicine but in every aspect of society, influencing legal, ethical, cultural, and financial arenas. The five problems with the diagnosis of PTSD mentioned above will be considered, as will the fact that the concept itself has created misdirection in

our field by moving us away from a better understanding of human response to trauma. This misguided venture has affected our clinical approach, scientific research, and ultimately our service to our patients and society at large.

Emotions of Adjustment

Posttraumatic stress appears to be more of a natural emotional response rather than a separate entity or malady. Perhaps stress after a trauma is better regarded as the emotions of adjustment rather than as a disease or a disorder.

Long before PTSD, physicians and therapists had seen how frightening events that threaten or cause injury can also produce a stereotypical response and psychological state in patients. The view was that these remarkably “similar set of symptoms across cases were states of mind following traumatic stresses — whether sudden and acute, or protracted and chronic — were emotional reactions with fairly predictable courses.”²

In his book, *Try to Remember*, McHugh writes about the move to PTSD and asserts that it is not a separate “entity” or “malady” but a “natural emotional response provoked by frightening events and varying in its intensity from patient to patient ...”¹⁴ In other words, these are emotions of adjustment common in us all. Just as the loss of a loved one causes grief, and relocation causes homesickness; a threat — a traumatic event — causes posttraumatic stress. If we do not consider homesickness and grief as distinct maladies, diseases, or disorders requiring specialized treatments, then why do we consider PTSD as such?

Disease Sponsorship

Many attempts have been made to promote PTSD as a disease category. However, the results have been contradictory and often erroneous. The most focused efforts have been made on hippocampal volume and the hypothalamic-pituitary-adrenal (HPA) axis.

In a study of hippocampal volume, Brenner and colleagues used magnetic resonance imaging-based measurements and showed the “shrinkage” of the hippocampus in combat-related PTSD patients.²⁴ A few other studies on hippocampal volume have shown this supposed decrease in size but none with a well-designed control group.^{25-28.}

Several years after Brenner’s initial research, Gilbertson made a key discovery. In a well-designed study using a Vietnam Era Twin Registry, he found that identical twins of combat veterans with PTSD, who were not themselves exposed to combat, had hippocampal volumes comparable to their brothers but significantly smaller than those of combat veterans without PTSD and their non-combat exposed twins (see Figure 2, page 510). These data indicate that hippocampi in those with PTSD have a pre-existing, familial vulnerability factor and that hippocampal volumes are not a product of trauma exposure per se.²⁹

Significant knowledge has been gained on how the HPA axis works. The evidence in support of increased levels of cortisol and corticotropin-releasing factor during physiological and psychological stress, as studied in major depression, is well supported³⁰⁻³⁴ (see

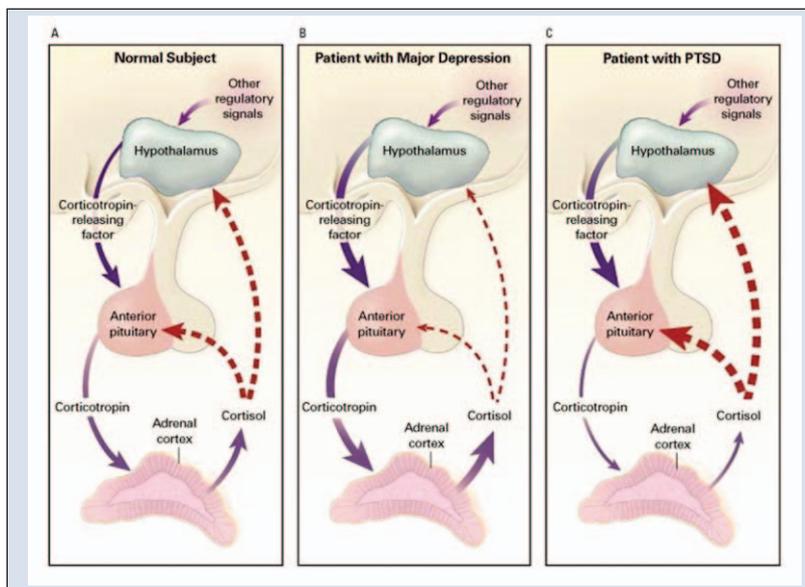


Figure 3. Response to stress in a normal subject (panel A), a patient with major depressive disorder (Panel B) and a patient with PTSD (panel C). Post-traumatic stress disorder. During normal physiologic state (panel A) of the hypothalamic-pituitary-adrenal (HPA) axis, corticotropin-releasing factor (CRF) from the hypothalamus stimulates the production of corticotropin from the pituitary which in turn stimulates the production of cortisol from the adrenal cortex. The negative-feedback system works by cortisol inhibiting the release of corticotropin and CRF. During Major Depression (panel B), increased levels of cortisol, corticotropin, and CRF are thought to be due to decreased sensitivity of the negative-feedback system. In contrast, patients with PTSD (panel C), low cortisol levels and high CRF levels are said to be due to increased sensitivity of the negative-feedback system of the HPA axis. Reprinted from Yehuda R. *N Engl J Med.* 2002;346(2):108-114. With permission.

Figure 3). Several studies suggest that patients with PTSD have low levels of cortisol due to increased sensitivity of the negative feedback system of the HPA (the opposite of major depression).³⁵

Many of these studies have very few participants (one cited had nine participants),³⁶ and many have no controls to poorly designed matched groups.³⁷⁻⁴¹ In reality, studies of cortisol levels in PTSD have produced mixed results.⁴²⁻⁴⁵ In a well-designed, 10-year study of a cohort community of 292 participants (69 of whom met PTSD criteria), Young and colleagues found no difference in cortisol levels in those with or without PTSD.⁴⁶

Numerous other studies attempting to show change in physiologic function, such as heart rate and skin conduction, have been inconclusive, showing nonspecific findings.⁴⁷⁻⁵⁰ These responses are present in many other examples of human emotional response.

So why have attempts to place PTSD in a disease category failed? Perhaps it is because PTSD is not a disease, or a mental disorder, but rather a mental state that is noticed because of its intensity and duration.

Conceivably, the results have been inconclusive and nonspecific because people can have this state of mind without any traumatic event. Many symptoms of PTSD, such as difficulty falling asleep,

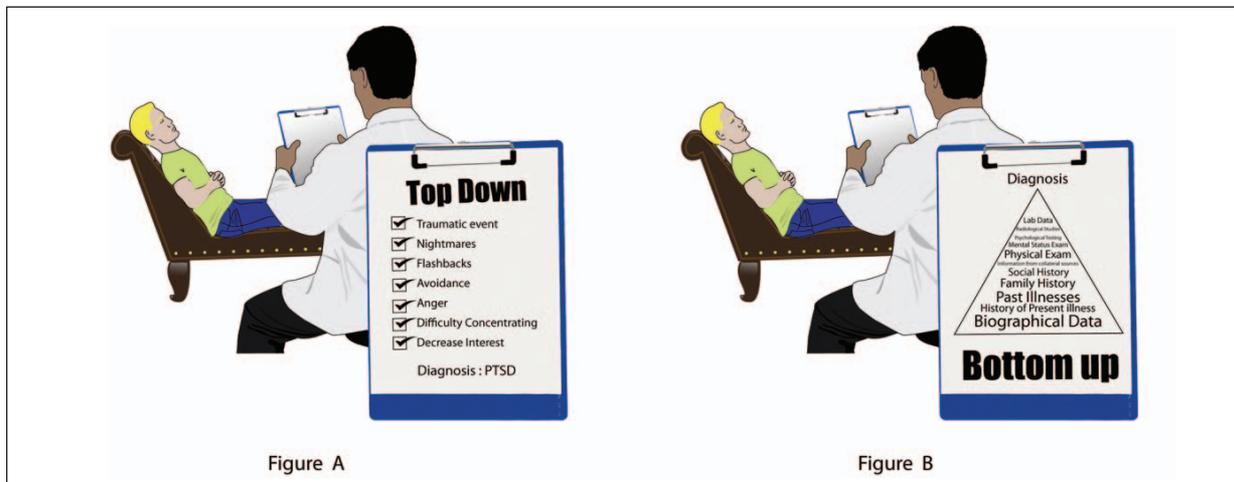


Figure 4. Psychiatric diagnostic interview and approach to patient evaluation and assessment. A. "Top-down" approach of symptom check-list leading to a pointed diagnosis. B. "Bottom-up" approach of patients leading to a more comprehensive diagnosis.

difficulty concentrating, diminished interest or participation in significant activities (avoidance), restricted range of affect, and irritability, are nonspecific to begin with. Many psychiatric disorders produce symptoms of this sort. We must stay clear of "the logical fallacy that *post hoc ergo propter hoc*" — just because one event follows another, it is not necessarily caused by it."²

Faulty Numbers

If stressful trauma is expanded to events and experiences listed in DSM-IV,⁵¹ then epidemiologists can show that 90% of us experience something of the sort in our lifetime.² DSM-IV's definition of criterion A, "exposure to trauma," is "a personal experience of an event that involves actual or threatened death or serious injury or other threat to one's physical integrity; or witnessing an event that involves death, injury, or a threat to the physical integrity of another person' or learning about violent death, seri-

ous harm or threat of death or injury experienced by a family member or other close associates."¹⁸ The inclusive nature of this criterion is impressive. From this, one can accurately say no life is trauma-free.

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The faulty numbers can be traced back to how the Veterans' Affairs Administration (VA) started gathering data on PTSD in the Vietnam War. In the book, *Stolen Valor*, by Burkett and Whitley, Burkett, himself a Vietnam veteran, discovered in his research how frequently men claiming PTSD from Vietnam had not only never seen combat, but they had never served in the military at all.⁵²

Another study also discovered that some of the PTSD cases being treated at the VA medical centers had never been to Vietnam, and some had never been in military service. Frueh and colleagues reviewed medical and military records on 100 patients in a VA medical center and saw that 94% were diagnosed with PTSD, but only 41% had evidence of combat exposure.⁵³ It is interesting that the information to confirm combat exposure was easily accessible but never sought by anyone in the VA medical center to corroborate the histories they were being given.

In 1983, Congress commissioned a specific and comprehensive study of the mental health status and general life adjustments of Vietnam Veterans. In 1988, the National Vietnam Veterans Readjustment Study (NV-VRS) reported that 1 million of the 3.14 million who served in Vietnam still had PTSD; that is, 31% of all men who served in that conflict had PTSD, even though only 15% had been assigned to combat units.^{54,55}

In other words, about 300,000 veterans had been assigned to combat units, yet 1 million had PTSD. A plausible explanation of the high prevalence could be that some men assigned to noncombat duty may have been exposed to PTSD-inducing stressors without being exposed to combat, but the high number of cases is still implausible and raises doubts about the epidemiological data. This, in turn, leads to questions about the assessment methods used to reach such high numbers.

Why so many PTSD cases? In an examination of methods for diagnosis employed in VA medical centers, case identification came to rest on symptom searches and questionnaires that, in a checklist fashion, probed for the DSM's criteria symptoms for PTSD diagnosis.⁵⁶ We assume that through years of training, a psychiatric interview is more than a mere checklist of symptoms in order to "meet criteria" for a disorder.⁵⁷

A psychiatric diagnostic interview needs to take a "bottom-up"^{2,13} approach that takes into account the patient's full biographical data. It examines the history of present illness, past illnesses, family and social history, and includes a physical exam and mental status exam. It also includes information from collateral sources to assess previous psychological problems and temperament and often involves psychological testing.^{2,13,58} The "top-down" symptom check-list approach has many flaws, not the least of which is the propensity for a high number of false-positives, as many psychiatric diagnoses in the DSM either share or have very similar symptoms (see Figure 4, page 512).

SIDEBAR.

Key Points in PTSD Treatment¹⁴

1. Symptoms should be seen for what they are, which are normal human reactions varying in severity, depending on the individual and the context of the traumatic event.
2. Patients should be treated by supportive management and coherent symptoms relief, all with the aim of facilitating recovery that will eventually emerge.
3. These conditions should be differentiated from and not confused with other emotional or behavioral conditions that can imitate them or emerge from them (eg, major depressive disorder, personality disorder, long-term behavioral problems; all of which require specific treatment programs of their own).

From "Try to Remember" by McHugh

Unreliability of Recall

Unreliability of recall is a common clinical finding. We know that a patient's recall of his/her life during a major depressive episode is often unrealistically negative and excessively guilt-ridden. Once treated, these memories are drastically different and more positive.¹⁴

Southwick and colleagues studied the consistency of memory for combat trauma in 59 veterans in the first Persian Gulf conflict. The veterans were given a 19-item questionnaire about combat 1 month and then 2 years after their return.⁵⁹ Results showed that nearly 90% changed their responses, with later memories displaying a significant increase in intensity and severity. With their new, and perhaps "recovered" memories, the subjects reported that PTSD symptoms increased significantly.¹⁴ The results of this study raise questions about the accuracy of recall for traumatic events and show that traumatic memories are neither fixed or lasting.

Fairbanks' work, along with a number of similar studies, used Minnesota Multiphasic Personality

Inventory (MMPI) testing, which showed extreme elevations on the validity scales in a "faking-bad" direction in Vietnam veterans with PTSD.⁶⁰ "Faking-bad" is a validity scale in the MMPI-2 designed to detect overreporting or exaggerating the prevalence or severity of psychological symptoms.⁶¹ Frueh suggests that "self-reports should be corroborated, whenever possible, by independent data sources."⁶² It is important to note that it is not the trauma that alters what is remembered and believed; rather it is the intensity of emotions and often the suggestions of others that alter what is remembered.¹⁴

Politics and Medicine

The mixing of politics with medicine always makes for bad medicine. The development of PTSD is a case of such a mixture.⁶³ Treisman and McHugh state that "PTSD is an example of how 'cultural attitudes' and 'power politics' came to dominate the profession's assumptions about meaning, memory, and mind."² Add sociocultural and political factors and we have the formula: Vietnam war + antiwar

psychiatrists + distressed America veterans = PTSD (a special condition, caused by wartime experiences, leading to a range of emotional illnesses that last for years, or decades, entirely because of “traumatic memories” that disrupt all of psychological life.)

TREATMENT APPROACH TO PATIENTS

Numerous studies have highlighted misguided assumptions about the approach to, and treatment of, patients with posttraumatic stress. Fontana and Rosenheck studied the effectiveness of inpatient treatment of PTSD. The study took place in the National Center for PTSD in West Haven, Conn. It included 4 months of intensive hospital treatment, including group, individual, behavioral, and family therapy, in addition to vocational guidance. The results showed that shortly after discharge, patients reported improvement, but 18 months later, they not only had no improvement but were also reporting a worsening of psychiatric symptoms, more family problems, and difficulty in personal relationships.⁶⁴

Another study by McNally and colleagues showed that debriefing has no effect on the overall rate of PTSD, and in some individuals, it may increase the rate of morbidity.⁶⁵ Perhaps the rush of “disaster counselors” to accident and injury sites to prevent “traumatic memories” and PTSD needs to be reconsidered.²

The importance of resiliency as a human trait, and as the most common response to trauma, has recently been brought to light again.⁶⁶⁻⁷⁰ In the epidemiology of Sept. 11, 2001, the resilience of the citizens

of New York City to the horrific terrorist attacks was applauded, although it surprised many contemporary “traumatologists,” who predicted an outbreak of PTSD in New York City and beyond.^{71,72}

The Army has a new approach to the nomenclature for traumat-



... the standard to treatment should be neutral in judgment, stating “it should neither deny the hero his laurel, nor shame the casualty with the white feather.”

ic exposure in its soldiers. The term now encouraged is “combat stress.”⁷³ Army leadership is beginning to realize that labeling the soldiers’ reaction to combat as a “disorder” does not serve its soldiers, commanders, or its mission.

Wessley, in an article about combat motivation and breakdown, underscores that the standard to treatment should be neutral in judgment, stating “it should neither deny the

hero his laurel, nor shame the casualty with the white feather.”⁷⁴

Solomon and colleagues, in an article describing the Koach Project (a form of cognitive behavioral therapy provided by the Department of Israel Defense Forces Medical Corps for veterans with posttraumatic stress), expressed that by provoking the patients to concentrate too much on their psychological wounds from combat, the therapists distracted them – and everyone else – from the patients’ “here-and-now” causal issues, all to the detriment of recovery and rehabilitation.⁷⁵ Perhaps, instead of concentrating and focusing on psychological “wounds,” treatment should focus on problem-solving and the here-and-now problems of adjustment.¹⁴

In treating patients with stress after traumatic events, it is important to note that recovery is variable and may depend on several factors, including the intensity of the provocative event, the person’s response, and the community’s reaction (for example, the reception of WWII veterans compared with that of Vietnam veterans). Ordinarily, symptoms settle with time, and patients adjust in some way to new circumstances. Just as grieving person responds, a patient who has faced a trauma benefits from sympathetic listening, reassurance that recovery will follow, and that symptoms gradually fade away, although it may take weeks to months. The reality is that some residual effects — such as a sense of loss, occasional bad dreams, and avoidance of places or other cues about the event that was experienced — may last for years or longer, but these reactions are all normal.

In many cases, the patient experiences difficulties beyond the normal emotions of adjustment to trauma. These are circumstances where the problems of adjustment have led to comorbid conditions that resemble psychiatric disease states, such as major depression, or panic disorder; physical manifestation of stress, such as coronary artery disease or peptic ulcer disease; or behavioral problems, such as excessive alcohol consumption, aggression, or suicide. All of these are recognized diagnoses that have established treatment programs of their own and should be treated as such.

Compensation is an important factor that has played a significant role in the chronic nature of PTSD and in the treatment-resistance of many cases. Huddleston and Hunt published *Accidents, Neuroses, and Compensation* in 1932. They wrote about compensation for PTSD, or “neuroses,” as it was generally called then. This is an interesting historical perspective on the issue of compensation. They write that in cases of treatment resistance, “[it] is no favor to war veterans, this perpetuation of their neuroses by continued subsidy for their psychogenic illness. It is far from curative as a beer diet in obesity. Relief via facilitated employment, even a service pension as such, is better medicine.”⁷⁶

In another historically interesting book, *War Stress and Neurotic Illness*, by Kardiner and Spiegel (Spiegel himself was a World War II veteran and a major in the Army) had the following view on cases of treatment resistance: “Very often I

was confronted in the treatment of these cases with the most profound resistance, based entirely upon the idea that if the patient got well, he would lose his compensation.” The authors go on to declare that “only those cases which fail to respond to treatment within 6 months should remain to be dealt with as problems of readjustment.”

In the end, they offer a solution, asserting that: “compensation should probably be reserved for only those cases proven to be incurable after treatment for 2 or 3 years under custodial care.”⁷⁷ One can only assume how this will affect the increasing number of PTSD cases. The Sidebar (see page 513) is from *Try to Remember*, McHugh’s pragmatic three-fold suggestion for helping parents deal with posttraumatic stress.¹⁴

PREMORBID VULNERABILITIES TO TRAUMA

David A. Alexander and Andrew Wells, two Scottish psychiatrists, did a before-and-after comparison study of police officers’ reactions to body handling after a major disaster.⁷⁸ In 1988, the *Piper Alpha*, an oil rig off the coast of Aberdeen, Scotland, exploded and left 167 men dead. Three months later, 105 bodies were still missing, entombed in the accommodation module of the platform lying at the bottom of the North Sea. It took several months to retrieve this enormous structure from the seafloor. Ultimately, 73 bodies were successfully recovered. Alexander and Wells studied the traumatic experiences and emotional reactions of 51 police officers whose

responsibilities included the stripping, washing, and photographing of recovered bodies. The officers also aided the team of pathologists in the postmortem reports.

Predisaster

This study provided an extraordinary opportunity due to a remarkable piece of epidemiological luck. Shortly before the disaster, Alexander had assessed many of the officers in an occupational health study using standardized measures: the Hospital Anxiety and Depression (HAD) scale and the Eyesneck Personality Questionnaire (EPQ). The HAD scale is a 14-item scale that assesses how the subject felt in the previous week. It is reliable and valid in assessing the severity of anxiety and depression in nonpsychiatric populations. EPQ measures indices of personality including: Neuroticism – N (emotionality); Extraversion – E (risk-taking); Toughmindedness – P (psychoticism/aggression); and Dissimulation – L (concealing truth/manipulation).

Postdisaster

Three months after the exercise, the officers completed HAD again. They also completed a revised Impact of Event Scale (IES), which is a 15-item scale of subjective distress after a traumatic event. It has two highly reliable subscales: intrusive imagery and avoidance tendencies. Finally, 12 months after the exercise, police records were used to obtain the amount of sick leave for each officer in an attempt to assess delayed reactions in the officers.

Results

Due to availability of predisaster data, it was possible to generate a control group of officers who were not involved in disaster duties. They were matched for age, sex, rank, marital status, and band scores on the HAD scale. The comparison of before-and-after anxiety and depression scores failed to reveal any increase in psychiatric morbidity and, indeed, showed a significant decline in anxiety scores. Moreover, the number of sick days 12 months after the exercise averaged 2.79 for those officers in the exercise, compared with 3.98 in the control group.

IES revealed that the officers involved in the disaster were not untouched by their experiences because posttraumatic reactions, such as intrusive images, were reported. However, without there being a raised level of psychiatric illness or sick leave, results tend to confirm the important notion that one needs to distinguish between distress and "caseness."⁷⁹ The findings also support the fact that there is not a simple one-to-one relationship between distress and psychiatric illness.⁸⁰

With the EPQ, the neuroticism (N) scale showed a correlation with the scores in the HAD scale in both groups, regardless of exposure to trauma. This shows the relevance of basic personality traits to the development of adverse psychological reactions to trauma. Another study by McFarlane found that neuroticism, as measured by EPQ, was a better predictor of posttraumatic morbidity than the degree of exposure to trauma.⁸¹ This counters the notion

that posttraumatic stress is a dose-response phenomenon and brings to light the importance of premorbid vulnerability to trauma.

The fortunate availability of data obtained before the *Piper Alpha* disaster revealed the risk of misinterpreting posttraumatic scores on measures of morbidity. In the absence of such data, it would have appeared as though a considerable proportion of officers were suffering from clinically significant levels of anxiety and psychological stress due to the disaster work and they would have been labeled as having PTSD.

CONCLUSION

The purpose of this article is not to discount the psychological stress of the aftermath of traumatic events but to highlight some significant problems with the current concept of posttraumatic stress, the diagnosis of PTSD, and how this concept has created misdirection in medicine and the care of our patients by moving us away from a better understanding of human response to trauma. This misguided venture has affected our clinical approach, scientific research, and, ultimately, service to our patients and society at large.

Posttraumatic stress should be regarded as normal human response and emotions of adjustment. When the course of readjustment is complicated, it should be differentiated from comorbid psychiatric conditions, such as major depressive disorders, generalized anxiety disorders, personality disorders, and behavioral problems (eg, substance dependence), because each has an established and specific treatment program of its own. More-

over, patients and society will be better served by removing disability compensation for PTSD and focusing on vocational training and support with the aim for recovery and the goal for better quality of life.

REFERENCES

1. Gjelten T, Zwerdling D, Inskip S. Officials begin putting shooting pieces together. *Morning Edition*. Washington, DC: National Public Radio; 2009.
2. McHugh PR, Treisman G: PTSD: A problematic diagnostic category. *J Anxiety Disord*. 2007;21(2):211-222.
3. Swartz MH. *Textbook of Physical Diagnosis: History and Examination*. Philadelphia, PA: WB Saunders; 2005.
4. Souza PD. *The Greek and Persian Wars, 499-386 BC (Essential Histories)*. New York, NY: Routledge, 2003.
5. Linderman G. *Embattled Courage: The Experience of Combat in the American Civil War*. Florence, MA: Free Press, 1989.
6. Leese P. *Shell Shock: Traumatic Neurosis and the British Soldiers of the First World War*. New York, NY: Palgrave Macmillan; 2002.
7. *Military Psychiatry: Preparing in Peace for War*. Washington, DC: Office of the Surgeon General; 1994.
8. MacCurdy JT. *War Neuroses*. Cambridge, UK: Cambridge University Press; 1918.
9. Swank RL. Combat exhaustion: a descriptive and statistical analysis of causes, symptoms, and signs. *J Nerv Ment Dis*. 1949;109(6):475-508.
10. Glass AJ, Bernucci RJ. *Neuropsychiatry in World War II*. Washington, DC: Office of the Surgeon General, Department of the Army; 1966.
11. Moran L. *The Anatomy of Courage*. Carroll and Graf, eds. 2007 (first published in 1945).
12. Scott W. *The Politics of Readjustment: Vietnam Veterans Since the War (Social Problems and Social Issues)*. Piscataway, NY: Aldine Transactions; 1993.
13. Kolb LC. *Modern Clinical Psychiatry*. Philadelphia, PA: WB Saunders Company; 1977.
14. McHugh PR. *Try to Remember*. New York, NY: Dana Press; 2008.
15. Ghaemi SN. Nosologomania: DSM and Karl Jaspers' critique of Kraepelin. *Philos Ethics Humanit Med*. 2009;4:10.

16. Frances A, Pincus HA, Widiger TA, Davis WW, First MB. DSM-IV: work in progress. *Am J Psychiatry*. 1990;147(11):1439-1148.
17. *DSM-V: The Future Manual*. Washington, DC: American Psychiatric Publishing; 2009.
18. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders* [text revision], 4th ed. Washington, DC: American Psychiatric Publishing; 2000.
19. Albuher RC, Liberzon I. Psychopharmacological treatment in PTSD: a critical review. *J Psychiatr Res*. 2002;36(6):355-367.
20. Kinrys G, Wygant LE, Pardo TB, Melo M. Levetiracetam for treatment-refractory posttraumatic stress disorder. *J Clin Psychiatry*. 2006;67(2):211-214.
21. Wallen K, Chaboyer W, Thalib L, Creedy DK. Symptoms of acute posttraumatic stress disorder after intensive care. *Am J Crit Care*. 2008;17(6):534-543.
22. John P, Wilson HG. Managing secondary PTSD among personnel deployed in post-conflict countries. *Disaster Prevention and Management*. 2004;13(3):199-207.
23. McGirk T. Hasan's therapy: Could "secondary trauma" have driven him to shooting? *Time*. Nov. 7, 2009.
24. Bremner JD, Randall P, Scott TM, et al. MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder. *Am J Psychiatry*. 1995;152(7):973-981.
25. Felmingham K, Williams LM, Whitford TJ, et al. Duration of posttraumatic stress disorder predicts hippocampal grey matter loss. *Neuroreport*. 2009;20(16):1402-1406.
26. Bonne O, Vythilingam M, Inagaki M, et al. Reduced posterior hippocampal volume in posttraumatic stress disorder. *J Clin Psychiatry*. 2008;69(7):1087-1091.
27. Schuff N, Neylan TC, Fox-Bosetti S, et al. Abnormal N-acetylaspartate in hippocampus and anterior cingulate in posttraumatic stress disorder. *Psychiatry Res*. 2008;162(2):147-157.
28. Rogers MA, Yamasue H, Abe O, et al. Smaller amygdala volume and reduced anterior cingulate gray matter density associated with history of post-traumatic stress disorder. *Psychiatry Res*. 2009;174(3):210-216.
29. Gilbertson MW, Shenton ME, Ciszewski A, et al. Smaller hippocampal volume predicts pathologic vulnerability to psychological trauma. *Nat Neurosci*. 2002;5(11):1242-1247.
30. Modell S, Yassouridis A, Huber J, Holsboer F. Corticosteroid receptor function is decreased in depressed patients. *Neuroendocrinology*. 1997;65(3):216-222.
31. Ising M, Horstmann S, Kloiber S, et al. Combined dexamethasone/corticotropin releasing hormone test predicts treatment response in major depression — a potential biomarker? *Biol Psychiatry*. 2007;62(1):47-54.
32. Sachar EJ. Corticosteroids in depressive illness. II. A longitudinal psychoneuroendocrine study. *Arch Gen Psychiatry*. 1967;17(5):554-567.
33. Carroll BJ, Feinberg M, Greden JF, et al. A specific laboratory test for the diagnosis of melancholia. Standardization, validation, and clinical utility. *Arch Gen Psychiatry*. 1981;38(1):15-22.
34. Nemeroff CB, Widerlov E, Bissette G, et al. Elevated concentrations of CSF corticotropin-releasing factor-like immunoreactivity in depressed patients. *Science*. 1984;226(4680):1342-1344.
35. Yehuda R. Post-traumatic stress disorder. *N Engl J Med*. 2002;346(2):108-114.
36. Anisman H, Griffiths J, Matheson K, Ravindran AV, Merali Z. Post-traumatic stress symptoms and salivary cortisol levels. *Am J Psychiatry*. 2001;158(9):1509-1511.
37. Yehuda R. Advances in understanding neuroendocrine alterations in PTSD and their therapeutic implications. *Ann N Y Acad Sci*. 2006;1071:137-166.
38. Bremner JD, Licinio J, Darnell A, et al. Elevated CSF corticotropin-releasing factor concentrations in posttraumatic stress disorder. *Am J Psychiatry*. 1997;154(5):624-629.
39. Baker DG, West SA, Nicholson WE, et al. Serial CSF corticotropin-releasing hormone levels and adrenocortical activity in combat veterans with posttraumatic stress disorder. *Am J Psychiatry*. 1999;156(4):585-588.
40. Mason JW, Giller EL, Kosten TR, Ostroff RB, Podd L. Urinary free-cortisol levels in posttraumatic stress disorder patients. *J Nerv Ment Dis*. 1986;174(3):145-149.
41. Yehuda R. Current status of cortisol findings in post-traumatic stress disorder. *Psychiatr Clin North Am*. 2002;25(2):341-368.
42. Pitman RK, Orr SP. Twenty-four hour urinary cortisol and catecholamine excretion in combat-related posttraumatic stress disorder. *Biol Psychiatry*. 1990;27(2):245-247.
43. Mason JW, Wang S, Yehuda R, et al. Marked lability in urinary cortisol levels in subgroups of combat veterans with posttraumatic stress disorder during an intensive exposure treatment program. *Psychosom Med*. 2002;64(2):238-246.
44. Lemieux AM, Coe CL. Abuse-related posttraumatic stress disorder: evidence for chronic neuroendocrine activation in women. *Psychosom Med*. 1995;57(2):105-115.
45. Rasmusson AM, Lipschitz DS, Wang S, et al. Increased pituitary and adrenal reactivity in premenopausal women with posttraumatic stress disorder. *Biol Psychiatry*. 2001;50(12):965-977.
46. Young EA, Breslau N. Cortisol and catecholamines in posttraumatic stress disorder: an epidemiologic community study. *Arch Gen Psychiatry*. 2004;61(4):394-401.
47. Lang PJ, Davis M, Ohman A. Fear and anxiety: animal models and human cognitive psychophysiology. *J Affect Disord*. 2000;61(3):137-159.
48. Liberzon I, Abelson JL, Flagel SB, Raz J, Young EA. Neuroendocrine and psychophysiological responses in PTSD: a symptom provocation study. *Neuropsychopharmacology*. 1999;21(1):40-50.
49. Litz BT, Orsillo SM, Kaloupek D, Weathers F. Emotional processing in posttraumatic stress disorder. *J Abnorm Psychol*. 2000;109(1):26-39.
50. Blechert J, Michael T, Grossman P, Lajtmann M, Wilhelm FH. Autonomic and respiratory characteristics of posttraumatic stress disorder and panic disorder. *Psychosom Med*. 2007;69(9):935-943.
51. Breslau N, Kessler RC. The stressor criterion in DSM-IV posttraumatic stress disorder: an empirical investigation. *Biol Psychiatry*. 2001;50(9):699-704.
52. Burkett BG, Whitley G. *Stolen Valor: How the Vietnam Generation was Robbed of Its Heroes and Its History*. Dallas, TX: Verity Press; 1998.
53. Frueh BC, Elhai JD, Grubaugh AL, et al. Documented combat exposure of US veterans seeking treatment for combat-related post-traumatic stress disorder. *Br J Psychiatry*. 2005;186:467-472.
54. McNally RJ. Can we solve the mysteries of the National Vietnam Veterans Readjustment Study? *J Anxiety Disord*. 2007;21(2):192-200.
55. Shephard B. *A War of Nerves: Soldiers and Psychiatrists, 1914-1994*. London, UK: Baillelief, Tindall and Cassell; 2000.

56. Young A. *The Harmony of Illusions: Inventing Post-traumatic Stress Disorder*. Princeton, NJ: Princeton University Press; 1995.
57. Treisman G. The DSM: has the tail begun wagging the dog? *Am Psychiatry News*. 2008;1-7.
58. Tavakoli HR. A closer evaluation of current methods in psychiatric assessments: a challenge for the biopsychosocial model. *Psychiatry (Edgemont)*. 2009;6(2):25-30.
59. Southwick SM, Morgan CA, Nicolaou AL, Charney DS. Consistency of memory for combat-related traumatic events in veterans of Operation Desert Storm. *Am J Psychiatry*. 1997;154(2):173-177.
60. Fairbank JA, Keane TM, Malloy PF. Some preliminary data on the psychological characteristics of Vietnam veterans with posttraumatic stress disorders. *J Consult Clin Psychol*. 1983;51(6):912-919.
61. Graham JR. *MMPI-2: Assessing Personality and Psychopathology*. Oxford, UK: Oxford University Press; 2000.
62. Frueh BC, Hamner MB, Cahill SP, Gold PB, Hamlin KL. Apparent symptom overreporting in combat veterans evaluated for PTSD. *Clin Psychol Rev*. 2000;20(7):853-885.
63. Stein DJ, Seedat S, Iversen A, Wessely S. Post-traumatic stress disorder: medicine and politics. *Lancet*. 2007;369(9556):139-144.
64. Fontana A, Rosenheck R. Effectiveness and cost of the inpatient treatment of posttraumatic stress disorder: comparison of three models of treatment. *Am J Psychiatry*. 1997;154(6):758-765.
65. McNally RJ, Bryant RA, Ehlers A. Does early psychological intervention promote recovery from posttraumatic stress? *Psychological Science in the Public Interest*. 2003;4(2):45-79.
66. Mancini AD, Bonanno GA. Resilience in the face of potential trauma: clinical practices and illustrations. *J Clin Psychol*. 2006;62(8):971-985.
67. Bonanno GA. Resilience in the face of potential trauma. *Current Directions in Psychological Science*. 2005;14(3):135-138.
68. Bonanno GA, Mancini AD. The human capacity to thrive in the face of potential trauma. *Pediatrics*. 2008;121(2):369-375.
69. Hoge EA, Austin ED, Pollack MH. Resilience: research evidence and conceptual considerations for posttraumatic stress disorder. *Depress Anxiety*. 2007;24(2):139-152.
70. Bonanno GA. Loss, trauma, and human resilience: have we underestimated the human capacity to thrive after extremely aversive events? *Am Psychol*. 2004;59(1):20-28.
71. Bonanno GA, Galea S, Bucchiarelli A, Vlahov D. Psychological resilience after disaster: New York City in the aftermath of the September 11th terrorist attack. *Psychol Sci*. 2006;17(3):181-186.
72. Breslau N, McNally RJ. The epidemiology of 9/11: technological advances and conceptual conundrums. In: *9/11: Mental Health in the Wake of Terrorist Attacks*. Neria Y, Gross R, Marshall R, eds. Cambridge, UK: Cambridge University Press; 2006.
73. Wiltout K. Military updating tactics on trauma. *The Virginian Pilot*. Nov. 8, 2009:1.
74. Wessely S. Twentieth-century theories on combat motivation and breakdown. *Journal of Contemporary History*. 2006;41(2):268-286.
75. Solomon Z, Bleich A, Shoham S, Nardi C. The "Koach" project for treatment of combat-related PTSD: Rationale, aims, and methodology. *Journal of Traumatic Stress*. 1992;5(2):175-193.
76. Huddleson JH, Hunt JR. *Accidents, Neuroses, and Compensation*. Baltimore, MD: Williams & Wilkins; 1932:178-197.
77. Kardiner A, Spiegel H. *War Stress and Neurotic Illness*. New York, NY: Paul B. Hoeber; 1947:405-413.
78. Alexander DA, Wells A. Reactions of police officers to body-handling after a major disaster. A before-and-after comparison. *Br J Psychiatry*. 1991;159:547-555.
79. Ingham J. Neurosis: disease or distress. In: *What is a Case? The Problem of Definition in Psychiatric Community Surveys*. Wing JK, Bebbington P, Robins LN, eds. London, UK: Grant McIntyre; 1981.
80. McFarlane AC. Long-term psychiatric morbidity after a natural disaster. Implications for disaster planners and emergency services. *Med J Aust*. 1986;145(11-12):561-563.
81. McFarlane AC. The aetiology of post-traumatic morbidity: predisposing, precipitating and perpetuating factors. *Br J Psychiatry*. 1989;154:221-228.