

# A paradigm shift in the conceptualization of psychological trauma in the 20th century

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## Abstract

The inclusion of posttraumatic stress disorder (PTSD) in *DSM-III* in 1980 represented a paradigm shift in the conceptualisation of post-trauma illness. Hitherto, a normal psychological reaction to a terrifying event was considered short-term and reversible. Long-term effects, characterized as “traumatic neurosis”, were regarded as abnormal. Enduring symptoms were explained in terms of hereditary predisposition, early maladaptive experiences or a pre-existing psychiatric disorder. The event served merely as a trigger to something that existed or was waiting to emerge. Secondary gain, the benefits often but not solely financial that a person derived as a result of being ill, was considered the principal cause of any observed failure to recover. The recognition of PTSD reflected a diversion from the role of the group, in particular the “herd instinct”, towards a greater appreciation of the individual’s experience. From being the responsibility of the subject, traumatic illness became an external imposition and possibly a universal response to a terrifying and unexpected event. This shift from predisposition to the characteristics of the event itself reduced guilt and blame, while the undermining of secondary gain made it easier to award financial compensation.

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The formal recognition of posttraumatic stress disorder (PTSD) in 1980 represented a paradigm shift in the way that psychiatric trauma was interpreted (APA, 1980). Hitherto, it was argued that if a healthy individual suffered psychological effects as a result of a life-threatening event, these would resolve themselves naturally, like a self-healing wound, with no long-term effects. The discovery of a so-called “delayed stress syndrome” during the Vietnam War appeared to show that healthy soldiers subjected to the trauma of combat could suffer chronic, adverse effects that were not apparent at the time of their exposure (Figley, 1978).

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Before the 1970s, anyone who suffered long-term psychiatric effects after a frightening event was considered constitutionally predisposed to mental illness or subject to a repressed childhood trauma; in either case, responsibility lay with the individual. The event itself served merely as a trigger. With the recognition of PTSD, primary causation transferred to the terrifying experience and any exposed individual was largely absolved from blame or responsibility. The new concept of psychological trauma also saw the retreat into obscurity of “secondary gain”, the attention and benefits that a patient received as a consequence of suffering from a recognized psychiatric disorder.

PTSD reflected a cultural shift from the group towards the subject. Psychological casualties in both World Wars were, in part, considered a failure of training, unit cohesiveness, leadership and morale (Wessely, 2006). PTSD was a product of a society in which the emphasis had moved from the duties of a citizen towards the rights of an individual. The new diagnosis was also designed to fill a gap created by loss of “gross stress reaction”, a disorder introduced by *DSM-I* (American Psychiatric Association (APA, 1952)) but not included in *DSM-II* (1966). Andreasen believed that a period of relative peace between the end of World War Two and the outbreak of the Vietnam conflict sixteen years later had led to a “foolish optimism” that such a category was no longer needed (Andreasen, 1980, p. 1518). In this paper, we explore how mental health professionals continually reinterpreted psychological trauma in response to wars, disasters and cultural undercurrents.

## 1. The conception of psychological trauma in the early 20th century

During the 19th century, with the exception of psycho-analytical literature, the word “trauma” generally referred to an open wound or violent rupture to the surface of the skin; it carried no psychological connotations. If, for example, a soldier broke down on campaign, he was deemed either to have succumbed to major mental illness, such as melancholia or dementia praecox, or to be suffering from the side effects of climate or disease (Jones & Wessely, 2005). The idea that a soldier of previously sound mind could be so emotionally disturbed by combat that he could no longer function was not entertained; that he might suffer long-term psychological consequences of battle was also dismissed. As a result, it was believed by British military physicians that “no war neuroses were observed in the Boer War”, the first “traces” being detected by Royal Army Medical Corps’ doctors sent to observe the Russo-Japanese War of 1905 (Stanford Read, 1920, p. 143).

### 1.1. *Traumatic neurosis*

The hypothesis that a terrifying event might have effects other than the purely physical had been proposed in the aftermath of the Franco-Prussian War (1870–1871). In 1882 wards dedicated to the treatment of men with hysteria were opened at the Salpêtrière. There Jean-Martin Charcot (1825–1893), a neurologist, identified a wide range of common but seemingly inexplicable symptoms including: palpitations, exhaustion, chest pain, dizziness and fainting, headache, back pain, trembling of the hands and neck, difficulty sleeping and mental disorientation. Among his patients were a number of ex-servicemen troubled almost a decade after the conflict had ended. One veteran, who he termed “D-ray”, had fought in the Mexican and Franco-Prussian Wars, and suffered from a range of symptoms including nightmares of his wartime experiences (Micale, 2001). Devising new diagnostic terms, “*névrose traumatique*” and “*hystérie traumatique*” to classify these cases, Charcot concluded that an event, such as a railway

accident or war, could serve as a trigger (“agent provocateur”) in individuals with an inherited disposition or “diathèse”.

Pierre Janet (1859–1947), who studied under Charcot, suggested that subconscious fixed ideas (“*idée fixe subconsciente*”), established at earlier periods in the subject’s life, were responsible for neurotic symptoms seen after a traumatic event. The accident itself was not the “cause of the consequent illness, but it was necessary to assign a role to the memories left by the accident, to the ideas and to the concern that the invalid maintained in this connection” (Janet, 1924, p. 39).

In their 1893 paper on “Psychical mechanism of hysterical phenomena”, Joseph Breuer and Sigmund Freud expanded the concept of traumatic neurosis. Having identified fright, heightened by surprise, as the driving force, they too argued that the crucial factor was not the event itself but the “susceptibility of the person affected” (Breuer & Freud, 1893, p. 56). Freud distinguished traumatic neurosis from other forms of neurosis on the basis that its symptoms, including dreams of the frightening event, were not amenable to interpretation. Not being a product of the subject’s imagination, the symptoms had no unconscious meaning. Three outcomes were possible: the disorder resolved spontaneously, became chronic or was transformed into a psychoneurosis (explicable in terms of the subject’s personality and life history). The last was said to happen only if the symptoms were of some advantage to the patient, for example, making a soldier unfit for front-line duties or providing a patient with a claim for financial compensation.

Not only was the event itself relegated to a secondary role, it was suggested that the patient was unaware of the fact that the trauma had produced his symptoms (Borch-Jacobsen, 2000). Janet and Freud argued that treatment required the subject to call to consciousness the repressed traumatic memory and allow the cathartic expression of any associated emotions (Hart, 1927). Abreaction was considered therapeutic regardless of whether the subject understood the significance or any hidden meaning associated with the repressed experience. Once the emotion attached to the memory had been discharged, Freud believed, the patient would be cured.

In the UK, “railway spine”, or functional somatic symptoms experienced by travellers who had been caught in a train crash, was a textbook example of a traumatic neurosis (Harrington, 2003). Some passengers continued to suffer from unexplained pain or disability long after their wounds had healed and many sought recompense from the railway or insurance companies involved (Young, 1995). At first, it was thought that their symptoms were a result of a neurological lesion but research by Henry Page suggested that these claims had no basis in organic pathology. Such observations led to Freud’s concept of secondary or “epinotic” gain to describe any advantage that a patient might secure from his symptoms, the primary gain being a reduction in anxiety following the so-called “flight into illness” (Freud, 1909, pp. 99–100). At first, the gain was conceived as largely monetary, though in time it was elaborated to include suppressed wishes for sympathy, attention or revenge. It had to be an unconscious process because if symptoms were sustained as part of a carefully conceived plan, then the patient was considered a malingerer (Trimble, 1981). Ganser syndrome, characterized by approximate answers, clouding of consciousness and functional somatic symptoms, was first described in 1898 among prisoners. Secondary gain for such individuals, it was hypothesized, might include a reduced sentence or stay of execution.

When World War One began, doctors and administrators did not anticipate an epidemic of traumatic neurosis because battle was an expected outcome for soldiers. It was also believed that those with a predisposition to psychological disorder would not have volunteered or have been rejected during recruitment (Ross, 1941). Furthermore, an infantryman’s training was designed to overcome fear, or at least to provide them with ways of managing it when in combat. The fact

that thousands of soldiers broke down led physicians recruited by the military to re-examine concepts of traumatic neurosis.

## 2. Shell shock

The commitment of mass armies to a prolonged conflict of attrition almost guaranteed a steady stream of psychiatric casualties during World War One. A variety of terms were used by the various combatant nations: “shell shock” in the UK, “choc commotionnel” and “choc traumatique” in France, while German doctors referred to “kriegshysterie”, “granatkontusion” (shell concussion) and “granatexplosionslähmung” (exploding-shell paralysis).

At first, doctors in the UK proposed an organic explanation: either a microscopic cerebral haemorrhage caused by the concussive or toxic effects of an exploding shell. When it became clear that many servicemen with the symptoms of shell shock had not been close to an explosion and some not even exposed to combat, other hypotheses were considered. Marr (1919) estimated that a physical lesion was responsible for only 20% of soldiers diagnosed with shell shock, while the majority, he believed, had an “inherent or constitutional disposition to nervous disease” (p. 49).

Because the term shell shock had overtones of a neurological disorder and for a period had entitled sufferers to a wound stripe, military authorities officially discouraged its use. As a result, doctors were forced to consider other labels to describe soldiers who had broken down in battle. Many opted for the term “war neurosis” (MacCurdy, 1918; Mott, 1919) based on the assumption that the conflict had evoked a pre-existing or latent psychological disorder. Elliot Smith and Pear, who had treated shell-shocked soldiers at the Red Cross Military Hospital in Maghull, concluded that “the manifestation of a severe psychical shock must necessarily be determined in a large measure by the nature of the mind upon which the injury falls” and any symptoms would be a function of the “individual patient’s ‘mental make-up’” (Elliot Smith & Pear, 1917, p. 16). Devine (1929) concurred with this explanation because many soldiers diagnosed with shell shock had broken down before they were exposed to actual danger, exhibiting “anticipatory neuroses” (p. 214).

Major Stanford Read, who commanded the British Army’s psychiatric unit at Netley Hospital during World War One, concluded that war neurosis depended on three causal factors: “heredity, early individual experiences, and the precipitating experience”. He was perhaps unusual in suggesting that

amid the horrors of modern warfare the precipitating situation is so fundamental and provocative that heredity and past nervous traces need be less necessary as adjuvant aetiological forces. This is where the psychoneuroses of war differ in origin from those of civil life (Stanford Read, 1920, p. 140).

Although Bartlett (1927) conceded that “every character, even the most stable, will crack and break under certain conditions of prolonged strain”; “some”, he added, “are more likely to be upset than others” (p. 10). Referring to what he called “the psycho-neuroses of warfare”, Bartlett argued that breakdown in combat was not essentially about the war but related to pre-service experiences and innate character. “A very important predisposing condition”, he argued, was “an unusual degree of shyness, or lack of sociability, or secretiveness” (p. 199). Bartlett divided cases into two diagnostic groups: conversion hysteria and anxiety neurosis; the former he thought more common among other ranks, while the latter principally affected officers and “the better class of officers too” (p. 188).

Indeed, war was not considered per se a traumatic event but could serve a therapeutic role. Janet (1924) argued that World War One, which may have done “much harm to many neuropaths, relieved some in an astonishing way and depressed subjects, sufferers from doubt and phobias became heroic soldiers” (p. 219). In Germany similar sentiments were expressed by Otto Binswanger who reported how “young men with weak nerves: anxious, timid, vacillating . . . who exhausted themselves in complaints about their physical and mental pain” were transformed by military service (Kaufmann, 1999, p. 128). There was also a suggestion of an inoculating effect: that having overcome a danger and fears on the battlefield, the individual was then protected against any further exposures.

### 3. Prisoners-of-war and combat wounded

Evidence gathered in Germany about prisoners-of-war appeared to confirm that it was not combat itself but the personality of the soldier that was the determining factor in any form of war neurosis. Reports that they rarely exhibited hysterical symptoms suggested a functional basis for these disorders grounded in wishes and desires (Lerner, 2003). Only soldiers in the front line had need of such symptoms to give them cause for hospitalization and a possible claim for compensation. In 1920, Julius Wagner-Jauregg, the Viennese professor of psychiatry, was accused by veterans of the brutal use of electric-shock therapy to treat of German soldiers invalidated with functional somatic symptoms. He argued in his defence that war neurosis was rarely seen in prisoners-of-war, who had escaped the fighting without resort to such symptoms (Ellenberger, 1970). Wagner-Jauregg said that very few war neuroses originated in the front line and that most cases arose amongst soldiers in base units.

During World War One, medical officers argued that shell shock was rarely seen in soldiers with a wound: evacuated from the front line, they “did not need a neurosis” (Thom, 1943; Wiltshire, 1916). Subsequently, Freud offered a more elaborate explanation: a “wound or injury inflicted simultaneously works as a rule *against* the development of a [war] neurosis” (Freud, 1920, p. 281) because simultaneous physical injury binds “the excess of excitation” (Ibid, p. 305). Whereas the person who survives an unexpected and frightening experience unharmed finds it difficult to assimilate the experience. Follow-up evidence from World War One suggested that Freud’s hypothesis was unfounded. Although soldiers recovering from wounds appeared to be free of neurosis, once an injury had healed, psychological disorders often emerged. Millais Culpin, who had served as a military surgeon in France and later trained in psychiatry, observed that this was because such individuals had “a strong predisposition to such disorders, so that subsequent relapse was certain” (Culpin, 1920, p. 31).

This interpretation stands in sharp contrast to current beliefs about prisoners of war who are considered at risk of psychological disorder (Wessely, 2006). Rather than list individual investigations, it is easier to say that we are unaware of any contemporary study, whether from Israel, United States, former Yugoslavia or elsewhere, that does not report psychiatric disorder in returning POWs, though rates are far from being uniformly high. Nice et al. (1996) found less than 5% of tortured POWs met PTSD symptomatic criteria.

How, then, can we explain the findings of the War Congress of the German Psychiatric Association held at Munich in September 1916 when a number of doctors including Robert Gaupp reported that French and British prisoners were virtually immune from psychological disorders (Lerner, 2003)? Retrospective study of UK veterans who had been POWs and been awarded a pension for neurasthenia/shell shock or disordered action of the heart (DAH) showed that they were rarely free from psychological symptoms. Their pension files do not record when

symptoms first arose but demonstrate that imprisonment did not confer long-term protection against psychological disorders. Furthermore, in 1945 a “neuroses centre” for repatriated POWs was opened at the Southern Hospital, Dartford, under the direction of Maxwell Jones. Operating for eleven months, it treated 1200 servicemen released from camps in north-west Europe (Jones, 1952, p. 16). Although no systematic study of outcomes was conducted, Tanner and Jones (1948) found that POWs had greater difficulty adjusting to civilian life than other soldiers, common symptoms being fatigue, loss of energy, anxiety and poor concentration. Rees estimated that 20% experienced “marked difficulty in the process of resocialization and reintegration into life in the army or life at home” (Rees, 1945, p. 102).

#### 4. World War Two and psychological trauma

Meeting in July 1939 in an effort to prevent another epidemic of shell shock, the Horder Committee decided that an official acknowledgement of war neurosis opened a route to discharge from the forces and the prospect of financial compensation. As a result, the British government announced that no pensions would be awarded for psychiatric war injuries (Shephard, 1999). Henceforth, soldiers traumatized by the stress of combat were to be diagnosed as suffering from “exhaustion” and retained within the forces. The term was chosen to imply that this was not a serious medical disorder, but a condition that would recover naturally with rest and respite. Abreaction and the ventilation of emotion were questioned as effective treatments. The control of fear and restoration of physical well-being were emphasized. In practice, this behavioural approach to war neurosis could be sustained only in totalitarian states such as Germany and the Soviet Union. In the UK, public opinion and pressure from doctors, Trade Unions and MPs forced the government to abandon its embargo on war pensions for psychological disorders in June 1941 and 2 years later to transfer the onus of proof from the claimant to the Ministry (Jones & Wessely, 2005).

Sargant and Slater (1944) explained war neurosis by the “constitutional approach”. To the dramatic “stresses and strains” produced by war, they argued “the predisposed personality can react only along limited lines”. Although they acknowledged extreme stress could disturb even the “most secure and stable personality”, most of those who broke down in battle were “the psychopathic, the damaged, defective or constitutionally unstable” (p. 9). Once again, vulnerability factors, rather than actual traumatic events, were viewed as primary in determining psychiatric morbidity.

When preparing for war, the government and its specialist advisers had predicted an epidemic of psychological casualties among civilians exposed to air-raids. When these failed to materialize even after the London Blitz of 1940, psychiatrists concluded that those with a predisposition to neurosis had evacuated the cities before the bombing began (Ross, 1941, p. 1). Other explanations included the strength of the herd instinct amongst people from the same locality gathered together in communal shelters and the fact breakdown did not provide a civilian an escape from danger or a claim for compensation (Kalinowsky, 1951, p. 343).

Based on evidence gathered from aircrew and elite army units, British doctors came to believe by 1943 that all servicemen, however carefully selected, well-trained or led, had a breaking point. Statistical studies conducted in the aftermath of the war confirmed these clinical observations. A study by Swank and Marchand (1946) of US infantry in north-west Europe found that after 60 days of continuous combat, 98% of surviving soldiers were likely to have become a psychiatric casualty of some kind, whether combat exhaustion, acute anxiety state or depression. In the remaining 2%, who were capable of enduring a sustained period of combat, they encountered a predisposition toward “aggressive psychopathic personality”. While it was now accepted that

all could breakdown, orthodoxy continued to dictate that only those with a constitutional vulnerability would not recover quite naturally once removed from danger.

## 5. Post-1945

The Korean War (1950–1953) led to no major innovation in the conception or treatment of psychiatric battle casualties, which continued to be regarded as varieties of “war neurosis”. *DSM-I*, published in 1952, contained the new category “gross stress reaction”, though no operational definition was provided. It described the extreme behavioural responses of normal individuals to exceptional stressors such as war or natural catastrophes. Although the main causal factor was an overwhelming environmental stress, similar to “criterion A” in PTSD, the effects were described as transient. The prognosis for gross stress reaction was considered good.

A chapter by Kardiner (1959) in Arieti’s *American Handbook of Psychiatry*, entitled “Traumatic Neuroses of War” saw no new insights. Kardiner interpreted breakdown in terms of intra-psychoic conflict and reached the startling conclusion that “traumatic neurosis is a disease very closely related to schizophrenia” (Kardiner, 1959, p. 256). In the UK, Henderson & Gillespie’s *Textbook of Psychiatry* made little reference to psychiatric trauma but identified hereditary and early life experiences as of importance: “if any doubts remained after the First World War as to the share of pre-existing personality in the causation of war neuroses . . . they were removed in the Second one” (Henderson & Batchelor, 1962, p. 466). The authors did concede that “stable individuals” exposed to circumstances of overwhelming fear could develop a traumatic neurosis, though these “usually diminish and disappear”. The symptoms could, however, be revived by “analogous experiences”. Henderson and Gillespie quoted the case of a pilot on a country walk who found

his heart pounding suddenly although he felt no fear; he then noticed a smell of burning and realized that it reminded him of a plane crash in which he had been involved and some of his comrades had been burned (p. 475).

Re-experiencing, sometimes in the form of flashbacks, became a key element in the new diagnosis of PTSD, though in the example reported by Henderson & Gillespie the phenomenon was explicable rather than involuntary.

Published in 1966, while the Vietnam War (1961–1975) was in progress, *DSM-II* introduced the term “transient situational disturbance”. This included all acute reactions (even brief psychotic episodes) to stressful exposures. The emphasis remained with the individual rather than the event and received wisdom suggested that it would be short-lived:

If the patient has good adaptive capacity his symptoms usually recede as the stress diminishes. If, however, the symptoms persist after the stress is removed, the diagnosis of another mental disorder is indicated (APA, 1966, p. 48).

## 6. PTSD defined

Codified in *DSM-III* (1980), PTSD was originally termed “post-Vietnam syndrome” or “delayed-stress syndrome”, having first been identified in veterans who had returned to the US. The treatment of acute combat fatigue had apparently been well managed by military psychiatrists attached to combat divisions. However, servicemen who had returned to civilian life presented with what appeared to be a range of delayed or chronic symptoms. Mental-health

professionals, who were politically opposed to the Vietnam War, took up their case. Figley (2002), himself a veteran and anti-war protestor, completed a doctorate on PTSD as part of his aim to demonstrate that “the toll of war went far beyond the battlefield” (p. 19). Robert J. Lifton, a prominent anti-war campaigner, was a key member of the sub-commission for reactive disorders that proposed the formal recognition of PTSD by the American Psychiatric Association. In part, validation of the disorder’s existence was a further way of undermining the US government’s pursuit of the war. If it could be shown that the conflict caused long-term and widespread psychological injury to US servicemen, then this was further reason to call the campaign to a close. Hence, along with “rentenkampfneurosen” (pension struggle neurosis defined in pre-1914 Germany), PTSD was one of the few politically driven psychiatric diagnoses.

PTSD entered the psychiatric canon obliquely and the careful epidemiological or nosological research required to support the diagnosis came later (Scott, 1990). Observers located its origins less in the jungles of Vietnam and more in the socio-political climate of America in the Vietnam era (Marlowe, 2000). The intense and critical attention given to the conflict was novel, contrasting with the careful censorship employed during World War Two to maintain morale amongst civilians. As a result, veterans, apparently subjected to stresses never felt by returning servicemen from other wars, were said to have become profoundly alienated.

## 7. Secondary gain

Secondary gain was a major concern of both clinicians and government planners largely because it was seen as playing a major part in preventing patients from getting well and thereby increasing the cost of disability pensions and other forms of financial compensation (Ross, 1966). In July 1939, when setting parameters for psychiatric casualties from the impending conflict, the Horder Committee decided that no awards should be made for psychoneurosis because “the pension itself may become such a preoccupation that it lessens the natural incentive to recovery” (Kalinowsky, 1951, p. 340). It was popularly believed that the refusal from 1926 to make any payments to German veterans for psychological disorders had led to the virtual disappearance of “shell-shock cases with shaking, paralysis, mutism, Ganser syndrome, and so on” (Kalinowsky, 1951, p. 341). In the UK, a distinction was drawn between “true” and “false” war neuroses. The former were said to occur in those with minimal predisposition and disappeared rapidly once the soldier was removed from danger. “False” neuroses arose in men with a constitutional weakness and were only thought likely to resolve in a favourable environment; failure to find employment and the payment of a pension provided fertile ground for the continued existence of these “false” presentations (Shephard, 2000, p. 151).

This issue dominated policy towards psychological trauma throughout World War Two. In 1941, for example, Ross argued that one of the “great causes of prolongation” of psychoneurosis was “the gain to be derived from the illness” (Ross, 1941, p. 17). After “hunting or skiing accidents”, Ross believed, traumatic neuroses were non-existent but they arose

very markedly after motor accidents and after accidents arising out of a workman’s occupation, i.e. we have them after an accident where someone else is responsible and will have to pay, especially if that payment will be made not by an individual but by a company ... This factor of gain through illness may be seen also in soldiers (pp. 26–27).

So long as a veteran or worker was receiving financial compensation for an industrial injury, he had little motivation to recover, particularly if that involved a return to an unpleasant or hazardous occupation.

In the period from the end of World War Two until the widespread acceptance of PTSD, secondary gain held an important place in psychiatric thinking about trauma. The *American Handbook of Psychiatry*, edited by Arieti (1966), for example, contained an entire chapter by Donald Ross entitled “Neuroses following trauma and their relations to compensation”. Henderson and Batchelor (1962) argued that the prognosis for traumatic neuroses was noticeably better when there was no prospect of financial reward. As late as 1983, the *Oxford Textbook of Psychiatry* had a section on “compensation neurosis”, which declared that “a single final settlement of the claim is often followed by an improvement in symptoms or disability” (Gelder, Gath, & Mayou, 1983, p. 358).

Not only had secondary gain been granted a causal role in traumatic neurosis, Miller (1961) argued that functional symptoms often disappeared once compensation had been awarded. A number of studies (Mendelson, 1982; Tarsh & Royston, 1985) undermined this suggestion and by the late 1980s, it was established that payment of a pension or a lump sum exerted little material effect on symptoms or function. More recently, a retrospective analysis of UK pension files for World Wars One and Two has suggested that long-term financial aid can “inhibit the natural process of recovery and consolidate distressing symptoms” (Jones, Palmer, & Wessely, 2002, p. 378).

At first, the concept of secondary gain appeared to have little relevance to PTSD. The trauma itself, rather than any subsequent management, was crucial in determining outcomes. Furthermore, implicit in the acceptance of PTSD was the idea that individuals without a predisposition to psychological disorder could suffer long-term psychological effects. Although secondary gain drifted into the background after the rise of PTSD it did not entirely disappear. A meta-analysis by Fishbain, Rosomoff, Cutler, and Rosomoff (1995) showed that 38 studies published between 1952 and 1994, employed the concept of secondary gain to analyze illness behaviour. In recent years, increased emphasis has been placed on the influence of compensation in the realisation that not just those with a vulnerability to psychological disorders are susceptible to financial pressures. Indeed, the issue of secondary gain found its way into DSM-IV guidelines for PTSD, which cautioned clinicians to be alert to malingering “in those situations in which financial remuneration, benefit eligibility, and forensic determinations play a role” (DSM-IV; APA, 1994, 467). Rosen argued that a systematic failure to rule out feigned cases, those motivated by pecuniary advantage, may have led to inflated rates of PTSD (Rosen, 2006). If PTSD is a culturally-conditioned response to adversity (Young, 1995), rather than a universal trauma reaction, then issues of compensation and other forms of reward will undoubtedly play a part in symptom formation and their duration.

## 8. Group versus individual

In the first half of the 20th century, theories of breakdown were framed in terms of the group rather than the individual. Soldiers were analysed not as single entities but as part of a hierarchical and structured organization. Hence, when the 1922 Southborough Committee attempted to prevent future episodes of shell shock, it made recommendations that referred to units rather than individuals. Training was to be designed to consolidate “the sense of collective responsibility and efficiency by securing the prompt and automatic obedience of orders”, while “military and medical witnesses were unanimous in insisting that good morale . . . is the first essential factor in diminishing the incidence of mental disorders” (Southborough, 1922, pp. 150–151).

Of crucial importance in understanding the role of the group was the primacy of ideas related to the “herd instinct”. Described in 1908, but given impetus by World War One, Trotter (1919)

argued that an individual should not be conceived in isolation as an autonomous being but as a gregarious animal whose “cardinal mental characteristic . . . is his sensitiveness to his fellow members of the herd” (p. 148). External threats, such as war, he believed, gave an intense stimulus to the herd instinct, while the “virtues of the warrior”, courage, endurance and enterprise, were grounded in the “homogeneity of the herd” (p. 150). It followed, therefore, that those who broke down were in some fashion abnormal and some argued that a “weak herd instinct” could be detected in many soldiers who were diagnosed with shell shock (Culpin, 1920, p. 32). Writing of his experiences during World War Two, Hunter observed that the military psychiatrist’s patient “is the army rather than the individual . . . To consider the individual soldier as an isolate, entirely detached from his group, is to pave the way for erroneous theories and unwise decisions” (Hunter, 1946, pp. 127–28). Until the Vietnam War, war neurosis was often conceived as a failure of attachment and identification with the group.

These observations were given statistical and empirical credibility with the publication of the landmark study of US servicemen who had fought in World War Two, usually referred to as the “American Soldier” (Stouffer et al., 1949), and also by the accessible post-battle analyses of S.L.A. Marshall (1947). Scholars and those who organized the education of future generations of officers were also acutely aware of the analysis conducted by Shils and Janowitz (1948) of the fighting qualities of the Wehrmacht. All of these works had at their core the finding that men were motivated in combat by small group ties and loyalties. Conversely, breakdown occurred when that cohesion failed and soldiers reverted to being individuals as opposed to members of the primary group. As one US psychiatrist wrote at the end of World War Two “the main characteristic of the soldier with a combat-induced neurosis is that he has become a frightened, lonely helpless person whose interpersonal relationships have been disrupted . . . the soldier must function as part of a group and his resistance to the traumata of combat will vary directly with his ability to integrate himself within the group” (Weinstein, 1947, p. 307). The contrast between pre-1980 concepts of breakdown in war as a failure of group dynamics and the post-1980 view that it should be understood in terms of the individual and their specific exposure is notable (Wessely, 2006).

## 9. Discussion

Since its formal recognition in 1980, PTSD has become a high-profile and politically sensitive psychiatric disorder (Vedantam, 2005). However, its international acceptance was not rapid or without controversy. It was slow to catch on in the UK where the disorder was initially considered specific to the US and Vietnam veterans. A study of *Post-traumatic neurosis* by Trimble (1981) never once mentioned the Vietnam War or PTSD. Similarly, the *Oxford Textbook of Psychiatry*, first published in 1983 and revised in 1986, made only the briefest of reference to a “post traumatic syndrome”. PTSD appears only in a table listing *DSM-III* labels and is not mentioned in the text at all (Gelder et al., 1983, p. 135). The authors, like most British psychiatrists, regarded it as a US phenomenon related to the Vietnam War (Mayou, personal communication).

Although acceptance of PTSD represented a dramatic break with earlier thinking about the psychological effects of a terrifying event, there had been a period when elements of the new definition were explored. Towards the end of World War One, some doctors conceded that everyone had a breaking point, though this discovery was lost in the findings of the Southborough Report, which concluded that breakdown in robust soldiers was wholly preventable by training, leadership, unit cohesion and morale. Not until 1943 was it accepted by military authorities that these protective forces were of limited capacity. However, it continued to be believed that those

individuals not predisposed to mental illness would recover quickly if taken to a place of relative calm. Interventions, such as debriefing designed to prevent the development of chronic disorders, had no obvious role as they could only interfere with natural processes of recovery in the healthy and would do little to address more deep-seated psychopathology in the constitutionally vulnerable.

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