

Shell Shock and Mild Traumatic Brain Injury: A Historical Review

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Mild traumatic brain injury is now claimed to be the signature injury of the Iraq and Afghanistan conflicts. During World War I, shell shock came to occupy a similar position of prominence, and post-concussional syndrome assumed some importance in World War II. In this article, the nature of shell shock, its clinical presentation, the military context, hypotheses of causation, and issues of management are explored to discover whether there are contemporary relevancies to the current issue of mild traumatic brain injury. When shell shock was first postulated, it was assumed to be the product of a head injury or toxic exposure. However, subsequent clinical studies suggested that

this view was too simplistic, and explanations soon oscillated between the strictly organic and the psychological as well as the behavioral. Despite a vigorous debate, physicians failed to identify or confirm characteristic distinctions. The experiences of the armed forces of both the United States and the United Kingdom during World Wars I and II led to two conclusions: that there were dangers in labeling anything as a unique “signature” injury and that disorders that cross any divide between physical and psychological require a nuanced view of their interpretation and treatment. These findings suggest that the hard-won lessons of shell shock continue to have relevance today.

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For many, shell shock was, and indeed remains, the signature injury of World War I, just as traumatic brain injury is claimed in some quarters today to be the characteristic injury of the Iraq and Afghanistan conflicts (1–3). In this article, we explore the symptoms, military context, hypotheses of causation, and issues of management of shell shock, in the expectation that some contemporary parallels will emerge.

In 1915, shell shock was initially conceived as a neurological lesion, a form of *commotio cerebri*, the result of powerful compressive forces (4, 5). However, doubts soon arose about the contribution of direct cerebral trauma to shell shock, and some expressed the view that the symptoms were more psychological than organic in origin, even to the extent of characterizing them as “traumatic neuroses” (6, 7). Some military doctors went so far as to state that the disorder was environmentally or contextually determined and that the way in which health care and compensation were organized served to reinforce both symptoms and disability. A vigorous debate ensued between the various schools of thought that led to a series of novel managerial interventions designed to limit what had become an epidemic of patients and war pension claims.

Military Context

During World War I, British troops found themselves exposed to a range of blast injuries, particularly before the introduction of the steel helmet at the beginning of 1916. Engaged in static trench warfare, frontline soldiers experi-

enced artillery barrage and mortar attacks, together with the threat of devastating mines. It is estimated that 60% of deaths in World War I were caused by shrapnel (8). Lt. Col. John Rhein, consultant in neuropsychiatry to the American Expeditionary Force, reported that 50% to 60% of soldiers with shell shock admitted to his base hospital claimed to have been concussed; for example, “a man states that he had lost consciousness or memory after having been blown over by a shell” (9).

Shell Shock

Head wounds and brain injury following exposure to exploding ordnance were recognized as a significant cause of invalidity in the opening phase of World War I. These casualties offered Gordon Holmes, consultant neurologist to the British Expeditionary Force, an unprecedented opportunity to test the localization of brain function. Cerebral trauma found itself at the cutting edge of military medicine. But what appeared to be a straightforward association between cause (shell explosion) and effect (head wound) soon became clouded and a cause of controversy.

Increasing numbers of soldiers who had been close to a detonation without receiving a head wound presented at casualty clearing stations with puzzling symptoms. They suffered from amnesia, poor concentration, headache, tinnitus, hypersensitivity to noise, dizziness, and tremor but did not recover with hospital treatment (10). Diagnosis became problematic because their clinical presentation was similar in many respects to that of soldiers who had experienced cerebral injury. The term “shell shock” evolved in an

attempt to describe cases that arose in the context of exploding ordnance but where enduring symptoms could not be linked to the presence of an obvious organic lesion. Shell shock entered the medical debate in February 1915 with the publication of a paper on the subject in *Lancet* by Capt. C.S. Myers, a specialist in psychological medicine (6).

In the spring of 1915, after bitter fighting at the second battle of Ypres, the number of shell shock cases increased, but the military made no significant progress in understanding the disorder, and still less in designing an effective management strategy. This was partly a question of priorities. The British Army struggled to open sufficient hospital accommodation for the wounded in France (11). The rapid growth of the British Expeditionary Force and unexpected levels of bacterial infection created pressing medical priorities, forcing shell shock to a lowly position on the military agenda for 1915. As a result, shell shock patients were transferred to base hospitals in France and the U.K. for observation in general wards (12). Without an informed treatment strategy, this puzzling disorder spread throughout the British Army. By the autumn of 1916, with manpower losses following the Somme offensive, the issue of shell shock finally came to the fore. The flow of casualties from the front had to be stemmed and an effective intervention devised to return combat troops to active duty.

Because shell shock was characterized by a wide range of common symptoms, it was open to multiple etiological explanations. At first, forces of compression and decompression were thought to cause a cerebral lesion, a form of *commotio cerebri* (5). Frederick Mott, then Britain's leading neuropathologist, who was recruited by the War Office to discover the etiology of the disorder, argued that in extreme cases shell shock could be fatal if intense commotion affected "the delicate colloidal structures of the living tissues of the brain and spinal cord," arresting "the functions of the vital centers in the medulla" (13). It was also speculated that the disorder resulted from damage to the CNS from carbon monoxide released by the partial detonation of a shell or mortar (14). In other words, shell shock was formulated as an organic problem even though the pathology remained unclear.

However, research conducted in 1915 and 1916 by Myers, consultant psychologist to the British Expeditionary Force, led to a new hypothesis (15). Based on his own observations, an increasing appreciation of the stress of trench warfare, and the finding that many shell-shocked soldiers had been nowhere near an explosion but had identical symptoms to those who had, Myers suggested a psychological explanation (16). For these cases, the term "emotional," rather than "commotional," shock was proposed. The psychological explanation gained ground over the neurological in part because it offered the British Army an opportunity to return shell-shocked soldiers to active duty. Increasingly short of frontline troops, any initiative that promised to restore soldiers to fitness was attractive. As a result, in November 1916, Arthur Sloggett, Director

General of Army Medical Services, authorized two new classifications: "effects of explosion (wound)" for those who were unable to perform their duties as a soldier as a result of direct contact with "a specific explosion ... without producing a visible wound" and "nervousness" for those whose symptoms were characterized by anxiety (17). In addition, four dedicated units were set up in France close to the front line ("forward psychiatry") for acute cases. Furthermore, specialist base hospitals were established for those already suffering from chronic effects (notably at Maghull, Craiglockhart, and the Maudsley). Considerable resources were diverted toward the investigation and clinical management of this apparently novel disorder.

A further problem encountered by the physicians at these specialist units, whether in France or the U.K., was to establish a definitive link between any explosion and subsequent symptoms. Without an organic lesion, any soldier in a war zone with symptoms of fatigue, memory loss, or dizziness had to be considered a potential shell shock case. Regimental medical officers were required to state on casualty forms whether a serviceman had been close to a detonation or not, but in the heat of battle with other pressing duties, this information was rarely provided (18).

When the United States entered the war in April 1917, U.S. military authorities faced the same steep learning curve. A month later, Maj. Thomas Salmon was ordered to the U.K. and France to study the question of shell shock and make recommendations for U.S. Army policy (19). In essence, he proposed a system of forward psychiatry supported by a large specialist "clearing hospital for mental cases," which led to the creation of Base Hospital No. 117, set up at La Fauche (20). Despite this careful planning, shell shock spread through the American Expeditionary Force and rose to significant levels during the Argonne offensive (21).

Scale of the Disorder

During World War I, 10% of British battle casualties were categorized as some form of shell shock or neurasthenia (22). In October 1917, Salmon reported that shell shock was responsible for one-seventh of all discharges from the British Army, and one-third if wounds were excluded (7). By the end of 1918, the British government had awarded 32,000 war pensions for shell shock, a figure that would rise dramatically once soldiers were discharged from the forces (23). Shell shock had initially caught the popular imagination in part because it was thought to be related to a genuine medical emergency, a head wound or neurological lesion. As Southard observed, the term "compared with the more acutely terrible and life-in-the-balance thing we know as traumatic or surgical shock" (24). In 1917, however, when it had become clear that many cases of shell shock were not directly related to a head injury, military medical authorities attempted to restrict use of the diagnosis. Servicemen invalided from the front were

given a preliminary label of “not yet diagnosed, nervous,” and those who failed to recover but had no visible cerebral injury were then classified as “neurasthenic.” Disputes over the etiology and management of shell shock served to inhibit further the design of an effective protocol. The involvement of the media and politicians, ostensibly to support the claims of individual veterans, added an emotive element that distorted policy and research (25). In November 1917, for example, Myers was denied permission to submit a paper on shell shock to the *British Medical Journal* because orders had been issued to the press bureau that nothing regarding the disorder should be released to newspapers (26).

Postconcussional Syndrome

To avoid another epidemic of shell shock once World War II was under way, British authorities banned the term (27). Despite this precaution, soldiers exposed to blast injury continued to present with a range of common symptoms of distress. In 1939, Schaller coined the term “posttrauma concussion state” to describe ongoing “disturbance of consciousness with no immediate or obvious pathologic change in the brain” (28). Although he proposed a range of symptoms or behavioral characteristics to differentiate this disorder from “posttraumatic psychoneurotic state,” Schaller could find none that were pathognomonic. Indeed, the two disorders shared a number of characteristics, including headache and dizziness. Mira (29) had argued from cases seen in Barcelona, Spain, after air raids during the Spanish Civil War that the presence of amnesia was a powerful indicator of a cerebral injury. Yet Culpin (30) argued, based on his experience of treating shell shock patients during World War I, that for many amnesia was the product of an unconscious process designed to block unpleasant memories, which could be recovered by hypnosis or suggestion. Indeed, Hadfield (31) reported the case of an air-raid warden who had been concussed during the London Blitz. In the immediate aftermath he suffered amnesia, wandering in a fugue state for 4 days. He subsequently experienced 18 months of headaches, insomnia, and severe neck pain but as a result of psychotherapy was able to recall in detail the experience of being blown in the air and the districts he had walked through.

By 1941, the term “postconcussion syndrome” had caught hold. The disorder was characterized by headache, dizziness, fatigue, tinnitus, memory impairment, poor concentration, and nervousness—symptoms that Wittenbrook (32) argued could not alone reliably differentiate it from “postconcussion neurosis.” Fulton concluded that “the problem of distinguishing such cases from organic concussion resulting from blast is delicate and often difficult” (33). Disagreement about etiology followed tracks laid down during World War I. Penfield (34), for example, thought that subdural adhesions could be found in patients who suffered from posttraumatic headache, and he

quoted Russel’s uncompromising nonorganic explanation: “Whereas in the last war the soldier who cannot ‘stand the gaff’ considered himself a victim of ‘shell shock’ ... in this war he has learned that the complaint of headache following a blow on the head is apt to serve as entitlement to invalidism and discharge.”

Individual symptoms, suggested Denny-Brown (35), did not hold the key, but their timing and number could distinguish between severe head injury and postconcussional syndrome. In the former, he argued, symptoms were immediate and severe with a trend to progressive recovery, while in the latter there was often a delay in onset and a tendency to get worse rather than better. This observation had also been made by Schwab and Fenton in the aftermath of World War I: “instead of passing away in a few days, as they normally do, [symptoms] begin after a comparatively free interval, become apparent again with a definite degree of persistence and exaggeration” (36). However, the hypothesis was not supported by a number of clinical investigations. A follow-up study of 1,020 military personnel with closed head injury led Symonds to the conclusion that “the practice of dividing the postconcussional cases into two groups, labeling the one organic and the other functional or neurotic” was “unprofitable and misleading” (37). Indeed, Lewis in 1942 and Guttman in 1946 (38, 39) underlined the similarities in the presentations of head-injured and non-head-injured soldiers seen in army psychiatric units. The two groups of soldiers seemed equivalent in terms of family and personal histories of psychological disorder and even range of symptoms. Furthermore, a study by Barrow and Rhoads of 200 U.S. Army personnel exposed to high-explosive blasts identified significant psychological effects in those who survived without apparent physical injury: “these patients were listless and apathetic and they seemed overcome with fatigue and lassitude” (40). Although most recovered quickly from this state, others continued to report symptoms for which no organic basis could be found.

With this knowledge in mind, British military doctors largely abandoned any attempt to divide servicemen with postconcussional syndrome into different categories based on whether or not they had a defined head wound. Specialist neuropsychiatric units, such as Mill Hill and Northfield, adopted a pragmatic approach designed to avoid invalidity and promote competence (41). They retained soldiers with shell shock in the armed forces and offered occupational therapy and vocational training based on aptitude tests. Given the debilitating effect of the shell shock label, the key, it was thought, was to return service personnel to purposeful activity without paying too much attention to causation.

Postwar investigations by Lishman (42) showed that postconcussional syndrome was characterized by subjective symptoms not directly accessible to observers. In addition, in a retrospective study of 670 World War II servicemen with head injuries, Lishman (43) showed that 144

(21.5%) had marked psychiatric disability on follow-up 1 to 5 years later. Enduring symptoms included headache, dizziness, fatigue, and sensitivity to noise. Assessed by a range of criteria (depth of penetration of injury, amount of brain tissue destroyed, or length of posttraumatic amnesia), 71 (10.6%) subjects consistently emerged as having the milder injuries. Lishman estimated that physical injury contributed little more than 7% of total disability and suggested that the emotional impact of the traumatic experience could precipitate psychiatric symptoms in those who are psychologically vulnerable.

In 1981, Trimble concluded that postconcussional syndrome was far from being a clear-cut diagnosis: "there is considerable psychiatric morbidity following head injury.... Neurotic symptoms are not only the prerogative of the mildly injured" (44).

Shell Shock: The Dilemma

Not only did shell shock affect service personnel in theater, it was also an enduring concern for returned veterans and had the potential to be a common disorder. Shell shock was largely free from stigma when used in the early phase of World War I because it was perceived as a wound, or a neurological lesion. Raynor, a divisional psychiatrist serving with the American Expeditionary Force, recalled "with what tenacity men clung to a diagnosis of 'shell shock'... something which was generally recognized as incapacitating and warranted treatment in a hospital" (45). Only in 1917, when the military authorities deliberately discouraged use of the term and suggested an association with malingering, did it become a controversial diagnosis.

Conclusions

In revisiting the debates on shell shock and postconcussional syndrome of the two World Wars, we had a contemporary purpose—to introduce some historical context to the current debate on mild traumatic brain injury, a context that has been conspicuous by its absence. While not seeking to prejudge the status of mild traumatic brain injury, we note that the U.S. military currently committed to serious fighting in Iraq and Afghanistan faces a situation similar to that of the British Army engaged in the Somme offensive of July 1916. Both campaigns have developed into wars of attrition in which head wounds and concussion are common battle injuries. The high casualties of the Somme battle brought the issue of shell shock to the fore when, as traumatic brain injury has done today, it caught the popular imagination and the attention of the media. The British Army struggled to define shell shock without a clear understanding of what it constituted and failed to produce a coherent management plan. The postwar ramifications were enormously expensive, with escalating war pension claims and a series of costly initiatives designed

to treat chronic cases. So troublesome had been the disorder that the term "shell shock" was proscribed on the outbreak of World War II and draconian policies introduced to try to prevent its reappearance.

During World War I, some doctors eventually accepted that the symptoms of both physical and emotional injury overlap and that it was difficult to distinguish between the effects of a mild head injury and an exceptionally stressful experience. Of course the shell shock generation of doctors did not have the benefit of investigative techniques now available, but as a study of U.K. troops returning from Iraq shows (N.T. Fear et al., unpublished 2007 data), there is an association between mild traumatic brain injury and posttraumatic stress disorder, with service personnel having a range of symptoms that often meet the criteria for both diagnoses. In states of uncertainty, it may be that contemporary service personnel prefer to be labeled as suffering from mild traumatic brain injury than any psychological disorder, just as shell shock in its initial quasi-neurological formulation was very popular. It may be that such labels reduce stigma and encourage help seeking, a major issue for the present generation of service personnel (46). But, on the other hand, it may divert attention from more easily treatable disorders, such as depression and posttraumatic stress disorder. Labels themselves affect prognosis. For example, a study of postconcussional syndrome by Whittaker et al. (47) suggested that subjects who believe that their symptoms have lasting and deleterious effects are at higher risk of experiencing an enduring disorder of this kind. In other words, strongly held negative beliefs play a part in maintaining symptoms and functioning—exactly the reasoning that led the British Army to ban the use of the term "shell shock" in 1917.

The generations of the two World Wars believed that future research would help in distinguishing between the physical and psychological causes of ill health in soldiers exposed to blasts. The 1922 *Report of the War Office Committee of Enquiry Into "Shell-Shock"* recommended that evidence be sought to limit the term to those cases in which a "causal connection" existed between "the effects of the explosive force and the symptoms resulting from the shock to the nervous system" (48). So far that hope has yet to be realized, although recent advances in neuroimaging may improve its prospects. Even then, it will remain the case that the symptoms many soldiers suffer are themselves both common and nonspecific. Furthermore, a clear-cut distinction between physical and psychological injury is unlikely to be realized, not least because the two coexist.

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