Psychiatric history

Trauma-induced dissociative amnesia in World War I combat soldiers

Onno van der Hart, Paul Brown, Mariëtte Graafland

Objective: This study relates trauma-induced dissociative amnesia reported in World War I (WW I) studies of war trauma to contemporary findings of dissociative amnesia in victims of childhood sexual abuse.
Method: Key diagnostic studies of post-traumatic amnesia in WW I combatants are surveyed. These cover phenomenology and the psychological dynamics of dissociation *vis-à-vis* repression.
Results: Descriptive evidence is cited for war trauma-induced dissociative amnesia.
Conclusion: Posttraumatic amnesia extends beyond the experience of sexual and combat trauma and is a protean symptom, which reflects responses to the gamut of traumatic events.
Key words: childhood sexual abuse, dissociation, post-traumatic amnesia, war trauma.

The psychological impact of trauma in both the military and civilian arenas has been documented for well over 100 years [1], but the validity of the traumatic neuroses and their key symptoms have been continuously questioned. This is particularly true for posttraumatic amnesia and therapeutically recovered traumatic memories. Freud's [2] abandonment of his seduction theory was followed by decades of denial of sexual trauma in the psychoanalytic and broader sociocultural realms [3]. Concomitant negation of posttraumatic symptomatology was noted in regard to the war neuroses, emanating equally from military, medical and social spheres [4]. Thus, Karon and Widener [5] drew attention to professional abandonment of the literature on posttraumatic amnesia in World War II combatants. They considered this to be

Paul Brown, Director (Correspondence)

Pierre Janet Centre, PO Box 42, Kew, Victoria 3101, Australia. Email: <drpaul@mpx.com.au>

Onno van der Hart, Professor; Mariëtte Graafland, Psychologist

Department of Clinical Psychology and Health Psychology, Utrecht University, Utrecht, The Netherlands. Email: <onnovdh@wxs.nl>

due to a collective forgetting, comparable to the repression of soldiers, but instead occurring on account of social prejudices. He further noted that the validity of memories was never challenged at the time since there was ample corroborating evidence.

Recent research confirms the findings of earlier investigators such as Janet [6], validating posttraumatic amnesia of both civilian and military origin. Van der Hart and Nijenhuis [7] cited clinical studies reporting total amnesia for combat trauma, experiences in Nazi concentration camps, torture and robbery. There is also increasing evidence for the existence of amnesia for child sexual abuse. Thus, Scheflen and Brown [8] concluded from their analysis of 25 empirical studies that such amnesia is a robust finding. Since then, new studies, for example those of Elliott [9], have appeared supporting their conclusion.

This paper examines posttraumatic amnesia in World War I (WW I) combatants. The findings are offered as an historical cross-validation of posttraumatic amnesia in all populations, including those subjected to childhood sexual abuse. The Janetian dissociative model of posttraumatic amnesia is pre-

ferred over the Freudian model of unconscious repression. Janet [10] equated the latter with his own narrowing of the field of consciousness. As a form of mental disintegration, dissociation does not require any active unconscious agency. For Janet, the term dissociation denotes both the process and the products of psychological and somatic splitting which result from the impact of trauma emotions. Dissociation is, however, fostered by active efforts to avoid confrontation with the traumatic memory. These efforts range from those which are carried out with conscious intent and which are closest to present day suppression (regarded by Rivers [11] as a form of conscious repression), to those which are carried out in awareness, but without conscious intent: Janet's [6,12] 'phobia of traumatic memory'. To facilitate clarity, repression will refer to the Freudian sense of the term, while when used by WW I authors in the present day sense of suppression, it will be designated 'repression'.

Conceptual and terminological limitations of these WW I studies must be recognised. There was no common language for describing posttraumatic amnesia, and important explanatory concepts such as suppression, repression, and dissociation were used interchangeably (e.g. Rivers [11]). Case reports were often incomplete, failing to provide crucial details regarding memory loss and the grounds for psychological diagnosis. Expression of symptomatology was often delayed, and many of those affected would not have recognised their disorder or sought help. Follow-up data was also lacking. Soldiers were rapidly returned to the front without psychiatric review, limiting the study of recovery, recidivism or development of fresh symptomatology. Finally, the studies quoted are a selected sample principally referring to English and American publications, including a few French and German studies, but excluding works in Russian.

Shell shock and combat neurosis

Combat reactions were noted from antiquity, but military psychiatry only fully emerged during the first two decades of the 20th century [4]. During WW I, soldiers in the trenches were exposed to some of the most extreme battle conditions ever known, in particular from artillery explosives. Shell shock was a controversial term used to refer to the acute psychiatric condition which followed exposure to exploding shells. War neurosis was the more encompassing term. By the Battle of the Somme in July 1916, these

disorders had reached epidemic proportions [13]. Bailey *et al*. [14] noted that combat reactions were at first baffling, and descriptions varied widely. Symptoms included 'staring eyes, violent tremors, a look of terror, and blue, cold extremities. Some were deaf, and some were dumb, others were blind or paralysed'. There were alterations of consciousness ranging from slight dizziness and shakiness to profound stupor, lasting from minutes to hours or more. Brown [15] subsequently distinguished three forms of combat neurosis: hysteria, neurasthenia and transient psychosis. Symptoms of hysteria were the commonest form, and included both somatic dissociations (mutism, blindness, deafness, muscle contracture and anaesthesia) and more purely mental dissociative phenomena. Neurasthenia found expression in anxiety, obsessions, compulsions and depression. The principal feature of the third group was mental instability, with wide ranging affective and psychotic symptoms, and alternating mental states reminiscent of bipolar affective disorder.

Posttraumatic amnesia

Posttraumatic amnesia frequently accompanied the war neuroses [11,16–28]. It was associated with dissociative symptomatology with loss of sensory and perceptuomotor functions. However, memories were not completely eliminated. They underlay symptomatic states, and could be recovered in treatment [11,15,19,20,22,24,26]. Both central and peripheral details of the traumatic experience were lost. Rivers [11] noted that soldiers manifested memory gaps from the moment preceding the shock to the point of hospital presentation, often weeks later. Nevertheless, they appeared to manifest normal consciousness during the intervening period, and were capable of distinguishing themselves in further military action. Posttraumatic amnesia was often accompanied by memory loss for non-traumatic personal information. For the sake of simplicity it can be divided into temporally localised combat forms, and generalised amnesias with loss of autobiographical information [29]. Localised amnesia is the inability to recall events related to a circumscribed period [30]. The following case described by Thom and Fenton [28] illustrates this symptom:

> A patient without past or family history of psychiatric disorder was picked up by military police suffering from a 6-month period of amnesia. Hypnosis at the military base hospital led to rapid recovery of

his memories. He had been separated from his company, and while crossing a field in search of his comrades a shell struck an unknown ally who was approaching 'and knocked his head off and buried itself in the ground about two feet from me'. The ordeal continued with shelling, but the next thing he remembered was being picked up by the police (p. 441).

Generalised amnesia refers to the subject's inability to recall important aspects of their entire life [30]. Memory loss could be both anterograde and retrograde. Many subjects were unable to recall their name, regiment, family background or marital status. In short, they had lost their identity [15,23,26]. Myers [25] described a case of total amnesia, following shell explosion:

> A soldier was assessed three days after having been admitted into a field ambulance. He was unable to give his name, regiment, or number, and he could not be identified. He could remember being found on the outskirts of a village, but his military history and all events in his past including his childhood were a complete blank (p. 65).

Malingering

Little WW I literature exists on malingering. Pure counterfeiting of psychological symptoms was considered rare by Myers [25] and Eder [18]. According to the former, some cases arose from a combination of true clinical hysteria, and autosuggestion, itself a minor trauma-related suggestibility effect. However, Miller [24] found that complete memory loss or somnambulism, which were, according to him, rare in genuine amnestic subjects, were not uncommonly simulated by malingerers. However, he gave no indices for prevalence.

Manuals were prepared to assist in the detection of malingering [31]. Hypnosis was recommended to differentiate malingering from true hysterical amnesia. Bassett Jones and Llewelling [32] differentiated between functional and organic upper arm monoplegia, and malingered cases. When the arm was raised and then released in hysteria, there was a moment of hesitation and then the arm fell slowly. This was in contrast to the deadness of the fall characteristic of true neurological injury. Malingerers were said to allow their arm to drop like a stone. Whether physicians were specifically warned against malingering for posttraumatic amnesia remains obscure.

Combat neurosis and causation

Combat neurosis was at first linked with premorbid psychopathology. Wolfsohn [33], in his study of 100 cases of war neurosis, found a family history of alcoholism, epilepsy, or insanity in 74%, and a prior 'neuropathic constitution' in 72%. Much lower incidences were found in other studies. Only 33% of Eder's series [18] of 100 cases evinced a previous personal or family history, and Bowman [34] felt 'that the preponderance of heredity for the mass of neuroses and psychoses is but a trifle more than healthy individuals'.

None of these findings were specifically related to posttraumatic amnesia. Most amnesic subjects had served at least 6 months before being exposed to the final pathogenic traumatic event [15]. They had been repeatedly exposed to combat stress under the most extreme conditions [18–21,26]

Organic versus non-organic aetiology

The relative neurological and psychological contributions to posttraumatic amnesia were hotly debated. Some favoured chemical causation, others advocated concussion.

Chemical theories were not readily supported [26,34]. In two soldiers exposed to noxious gasses, microscopic and vascular cerebral changes found at death were absent from the cerebrospinal fluid at 48 h [35]. Supporting this finding, hospital lumbar puncture was almost always normal [26,31]. Nevertheless, Mott and Myers did not exclude the possibility of as yet undetected submicroscopic changes.

The contribution of concussion to posttraumatic amnesia by inducing unconsciousness [11,19, 21,22,28] proved harder to dismiss, finding expression in the term concussion amnesia [21]. This covered both anterograde and retrograde deficits. Concussion could be followed by periods of unconsciousness, fluctuating consciousness or delirium. During the latter, soldiers often imagined themselves fighting without fear. There was also considerable fatigue, and an amotivational state called aboulia. With recovery from concussion, traumatic memories were gradually retrieved [20,21]. However, many amnestic soldiers did not suffer from head injury or concussion [11,20,24], and there was consistent failure to confirm physical causation. Even with severe head injury, evidence accrued for the predominantly functional origin of posttraumatic amnesia

[11,17,20,22–25,27,36]. Moreover, in most cases, the rate-limiting factor in the recovery of memory was psychological rather than physical. Persistence of amnesia rather reflected phobic resistance to facing painful memories [15,24,26].

Myers [26] attributed posttraumatic amnesia to massive emotional shock, equivalent to present day DSM-IV acute stress disorder [30]. He stated:

> Typically the immediate result of trauma is a certain loss of consciousness. But this may vary from a slight, momentary, almost imperceptible dizziness or clouding to profound and lasting unconsciousness. When the shock is slight, the patient may be able to 'pull himself together'. When the shock is severe, it may be followed by an unrestrainable excitement, depression, fugal automatism or stupor, on recovery from the graver forms of which the patient can recall none of the aspects performed by him during that condition. But in the states of lighter stupor and in the excitement, depression and automatism just mentioned, the attention would appear to be concentrated on some narrow field, doubtless generally on the scene which produced this condition (p. 66).

Combining neurological with functional causation, Thom and Fenton [28] speculated whether transient psychophysiological concomitants of shell shock (cessation of respiration and heartbeat, paralysis of body movements, glandular hyperactivity, etc.) were sufficient to produce unconsciousness, and thereby posttraumatic amnesia. They compared these findings with feinting following civilian accident trauma in which unconsciousness could also be followed by amnesia.

Amnestic soldiers were frequently hypnotisable, and vivid traumatic memories could be readily retrieved using this approach [11,15,17, 18,20–25,28,36,37]. The memories of a man who saw a soldier decapitated by a shell were recovered in this way within 2 h [28]. When memories were restored, concomitant functional symptoms (e.g. paralysis) tended to disappear [11,16,17,21,22,24,36]. However, subjects were prone to symptom substitution [15], and risked the development of chronic functional symptomatology. Furthermore, in most cases of posttraumatic amnesia, particularly those suffering postconcussional amnesia, there was a residual amnestic period extending from the traumatic experience to hospitalisation. This period was resistant to hypnosis and other treatment methods [11,20,21,23,24,26,28], leaving open the question of physical versus psychogenic causation.

In conclusion, organic and functional factors were not seen as mutually exclusive, and somatically orientated psychiatrists increasingly acknowledged psychological contributions [11,15,18–21,23].

Dissociation

Following WW I, two rival functional explanations for posttraumatic amnesia obtained: the theory of dissociation and that of repression and suppression. These approaches and their respective terminologies were often used interchangeably, with symptomatic and explanatory distinctions frequently blurred.

The dissociative view was founded on Janet's work [15,17,19,22,23,25,26,28,36–39]. Janet conceived of posttraumatic hysteria and war neuroses in general as dissociative disorders [40]. In these, traumatic memories and related personality functions are dissociated from ordinary consciousness, subconsciously persisting as fixed ideas. They are masked by posttraumatic amnesia, isolated from conscious awareness and voluntary control, only to be triggered under state-specific conditions to give rise to the 'accidental' symptoms of hysteria or the re-experiencing phenomena [7,41].

Freud's concepts of repression and suppression, were also applied to posttraumatic amnesia [20,24,42]. According to this view, traumatic memories are consciously suppressed or unconsciously repressed, on the one hand nullifying the impact of traumatic affects, but on the other giving rise to the symptoms of hysteria. Some workers combined both Freudian and Janetian models, assigning (Freudian) repression an initiating [17,23,26,37] and/or maintaining [23] role in the (Janetian) dissociative process.

The following WW I findings are not only consistent with the Janetian model of posttraumatic amnesia but also with contemporary dissociative approaches. Unconscious repression does not appear to account for memory loss. Military psychiatrists were able to make the same symptomatic distinctions between different hysterical dissociative phenomena as had Janet in traumatised subjects in civilian populations. The latter [43] distinguished protean hysterical somatoform phenomena ('stigmata') accompanied by posttraumatic amnesia from reactivated traumatic memories disguised in posttraumatic re-experiencing ('accidental') phenomena. In this regard, Myers [26] and McDougall [22] similarly described the reemergence of symptomatic equivalents of traumatic memories in fugues, somnambu-

lisms, and dreams. Myers [26] regarded these as manifestations of the 'emotional' personality, alternating with the 'apparently normal' personality. When in the former state, the subject relived combat trauma. Fighting scenes were vividly re-experienced during which the soldier exhibited pseudo-fighting movements. At such times, stigmata typifying the 'apparently normal' personality (e.g. paralysis or contracture) were usually held in abeyance. Upon 'waking', however, the 'apparently normal' personality showed no fear, had no recollection of the alternate state, and immediately resumed expression of the stigmata. The following is McDougall's [23] description of re-experiencing phenomena:

> A robust soldier appeared to quickly recover from shell shock. He was about to return to the field, when his copatients reported that he was sleepwalking. Several times nightly he arose, walked over to the sergeant's bedside, and stood there until led back to his bed. Under hypnosis he described his accident. A shell exploded, killing and wounding several comrades. When rushing off to report this incident to his sergeant a second shell exploded, dazing him, and leading to his hospital admission. In somnambulism he was reliving this scene, the memory of which was dissociated (p. 259).

According to McDougall, a symptomatic transition could be readily recognised from partial to complex reenactment, from fits through somnambulism to fugue. Symptoms were underlied throughout by extensive posttraumatic amnesia. The following case, from McDougall [23], illustrates the transition from somnambulism back through a hysterical fit:

> A young soldier suffered day and nighttime 'attacks' while recovering from a foot wound. In these he dramatically re-lived the repulsion of an enemy attack in the trenches. At the height of the attack he shouted to his comrades from his machine gun emplacement. As his excitement gradually subsided these re-enactments were replaced by spasmodic movements, which in turn subsided leaving him sleeping quietly. There was no subsequent recall (p. 260).

Dissociation was sometimes so complete, that manifestations of trauma did not obtrude into consciousness and recognition was considerably delayed [25,26]. Traumatic memories remained latent and inactive, and amnestic subjects appeared to function normally [23]. In cases described by Kardiner [20] and Miller [24], amnestic soldiers only developed symptoms after a prolonged period of normal func-

tioning. The following example is drawn from Miller [24]:

> A sergeant survived the war without a scratch, and following demobilization, bought a taxi-cab. A slight accident brought about a complete collapse, with fugues, phobias, and an hysterical gait. He had been reluctant to speak of the war, and at assessment, there was complete amnesia for his combat experience. As treatment progressed his symptoms indicated some specific incident leading to his amnesia (p. 39).

Hypnosis, itself believed to psychologically induce dissociation, was understood to be capable of accessing dissociated experiences of combat trauma. It, therefore, lent support to psychogenic theories of posttraumatic dissociation and dissociative amnesia. Memories were vividly relived under hypnosis (e.g. as if the hypnotic subject was again fighting in the trenches [15,19,20,22,23,25,26,36,44]). Traumatic experience appeared to be faithfully reproduced rather than imaginatively reconstructed. A case by Brown [15] illustrates this:

> He talks as he talked at the time he received the shock. He really does live through the experience at the awful time. Sometimes he speaks as if in a dialogue, punctuated with intervals of silence corresponding to the remarks of his interlocutor, like a person speaking at the telephone. At other times he indulges in imprecations and soliloquy. In some cases he is able to reply to my questions and give an account of his experiences. In others he cannot do so, but continues to writhe and talk as if he were again under the influence of terrifying emotion (p. 93).

Consistent with Janet's experimental studies, Brown [15] considered high hypnotisability as evidence for the dissociative basis of posttraumatic amnesia. The corollary was similarly supported: subjects became less hypnotisable as memory was recovered. Further evidence for posttraumatic dissociation was provided by personality reintegration accompanying hypnotic recovery of traumatic memories [15,20,23,24]. Myers [26] called this the transformation of the 'apparently normal' to the 'normal' personality. According to him, although amnestic soldiers simulated normality, their appearance and behaviour altered dramatically after their memories had been recovered. It was as if their actual personality had returned. This is illustrated by Myers [26] in the following case:

A soldier was found on a road a few miles behind the front. At base he manifested combat shock with psychosomatic collapse, and amnesia for his personal and military history. Under hypnosis he was able to give his name, number, and regiment. He described heavy shelling, with attacks of dizziness and wandering into parts of the trenches where he should not be. He subsequently retained his recovered memories, and his general physical condition was rapidly restored. 'Delighted with his recovery, he returned after three weeks rest to duty at the front where he continued in good health'(p. 45).

McDougall [22,23] and Brown [15,17] did not regard dissociation as entirely psychogenic. Rather, they advocated a complex biopsychosocial model in which psychological processes were also mediated by organic factors, including undetected submicroscopic changes in the nervous system. McDougall [23] further proposed that affected cortical 'dispositions' were disconnected at higher cortical levels from all others. According to Brown [15], dissociation of memories was due to activity in the central nervous system, whereas dissociation of other personality functions was due to the sympathetic nervous system. Symbolisation and psychological overlay were considered to act upon this organic substratum. In conformity with Janet's [43] recognition of the ideational elaboration of symptoms, Brown [15] and Myers [26] felt that the majority of hysterical symptoms were 'grafted' onto wounds and injuries to the affected body parts. Brown [15, p.99] further regarded dissociated sensory and motor functions as representing the physical counterpart of fear experienced during the traumatic event. His case below illustrates his views:

A soldier was dragged from a shell hole by his arm, causing a modest traction injury to his brachial plexus. A paralysis developed with anaesthesia, vasomotor disturbances, and exaggerated tendon reflexes. There was marked functional overlay. This was confirmed while re-living his painful combat experiences under hypnosis when he was freely able to move his hand (p. 99).

Myers [26] and Miller [24] expanded on the symbolic relationship between functional deficits and traumatic memories. In a difficult case, the latter described the relief of aphonia following the revival of war memories:

Then it came at the critical moment of a most tragic episode. The man himself explained that this inabil-

ity to speak had been an expression of his reluctance to allow any emotion in his voice (p. 43).

Despite such evidence for dissociation underlying posttraumatic amnesia, however, others implicated repression and suppression.

Repression and suppression

Rivers [11,45] linked dissociation and posttraumatic amnesia with hysteria, and 'repression', suppression and obsessive recall with neurasthenia. He regarded 'repression' as a process of active, conscious exclusion of traumatic memories from awareness, and suppression as the successful consequence of this 'repression'. He wrote:

It is natural to thrust aside painful memories just as it is natural to avoid dangerous or horrible scenes in actuality, and this natural tendency to banish the distressing is especially pronounced in those whose powers of resistance have been lowered by the long-continued strains of trench life, the shock of explosion, or other catastrophes of warfare. This natural tendency to repress being in my experience almost universally fostered by their relatives and friends, as well as by their medical advisers (p. 173).

Typical of neurasthenia was continuous, direct and indirect obtrusion of traumatic memories (e.g. in ruminations and anxiety dreams). Hysteria was instead characterised by posttraumatic amnesia in the waking state, memories emerging in somnambulistic crises (flashbacks and re-enactments) and under hypnosis. In contrast to dissociated memories in hysteria, 'repressed' memories in neurasthenia were regarded as more readily available to recall. Rivers [11,45]) gave the following example of 'repression':

An officer was buried following a shell explosion. His symptoms suggested cerebral concussion. He continued to fight, despite headache and vomiting, until encountering the mutilated body of a fellow officer. His days were spent in anxious anticipation of the evenings which were haunted by this vision, and he feared going to sleep and having nightmares. Although encouraged to try and keep thoughts of war from his mind, he was unable to do so (p. 174).

Role of repression in dissociation

Brown [15,38] considered dissociation to be due to the ineffectiveness of repression (in the sense of Freud and Breuer [46]). He stated [38]:

Without accepting Freud's sexual theory of the origin of the psychoneuroses, I do accept his theory of the cause of mental dissociation; that it is the result of the mental conflict that involves repression of emotional states. In most cases of nervous shock caused by shell explosion, a state of intense fear is aroused in the patient's mind, which from its very magnitude, produces loss of self-control or apparent loss of consciousness. There is no real loss of consciousness, but the attempted repression and control of the fearful emotion at its inception brings about a splitting of the mind, which appears later as an amnesia of great or less extent, often involving other losses of functions (p. 198).

McDougall [22] and Myers [36] rejected Brown's view. Instead they considered dissociation to either follow trauma directly, or to result from prolonged 'repression' (present day suppression), themselves due to prolonged exposure to danger. Most dissociative phenomena, however, including posttraumatic amnesia, were considered the direct emotional consequence of acute war trauma. This accorded with Janet's view [6,47]. In this, 'vehement' trauma emotions weaken mental capacities by impairing the subject's ability to integrate mental contents into personal consciousness. Myers [26,36] stated in this regard:

In cases of shell shock, conflict and attempted repression do not necessarily precede dissociation. In most amnestic cases the essential pathological process consists in a dissociation affecting the entire personality owing to 'sudden overwhelming emotional shock' by exploding shells. The soldier is no longer 'himself', and with return of the 'apparently normal' personality, after profound stupor or confusion, repression occurs. The memory of the actual scene hardly escapes from its repression. It is merely dissociated and may repeatedly disturb the waking and sleeping life of the patient in many well known ways' [26, p.67, 35, p.22].

Thus, Myers suggested that 'repression' plays a part in maintaining the dissociative process. Consistent with this, McDougall [23] wrote: 'The repressing forces do not maintain the dissociation in any direct or positive manner, but rather they do prevent the abolition of the dissociative barriers by working against, and thus rendering less efficient, all conation directed to overcome barriers and recover the lost memories' (p. 247). In the same vein, Janet's [6] 'phobia of the traumatic memory' might be

regarded as an avoidant behaviour preventing the abolition of such dissociative barriers.

Such findings might explain the strong inclination during the amnestic period, both during the waking state and under hypnosis, to act as if memories are actively inhibited [20,23,25]. In this regard, Myers [25] wrote:

Whenever some patients were endeavored to think of their forgotten memories, pains frequently caused the patient to wake from hypnosis. Headaches became severe. When, if mute, they endeavored to talk, they complained generally of a pain in the throat. (…) When at length his reluctance was overcome the attitude of the patient often changed from depression to excitement. His pulse and respiration increased in frequency, he sweated profusely, and not infrequently showed clear evidence of living through the scenes which were vividly coming to his mind (p. 68).

According to Kardiner [20], resistance to entering hypnosis and verbalising amnestic material was due to discomfort anticipated when reliving traumatic memories. This is another example of Janet's [6] 'phobia for the traumatic memory'. Janet did not equate this emotional disturbance with unconscious repression. Rather, he saw it as a fearful sign of dissociation, reflected in the inability or even reluctance to assimilate the most intrusive and painful aspects of traumatic experiences. McDougall [23] described a case in which an active, conscious effort of 'repression' (present-day suppression) was more manifest:

A Canadian soldier with amnesia for both recent military events and for his personal history reported that he could not bear to look at war-pictures in the newspapers. Under hypnosis, he retrieved fragments of his combat experience and of his prior family life. He saw pictures of two men lying wounded and blood stained in a trench, but treatment did not advance any further. The therapist explained that horror prevented him from remembering, to which he replied: 'Can't I go on all right without remembering those things?' With urging he reported further details of the bombardment and the wounding of his chums in the trenches. He was also able to give details of his home life in Canada (pp. 246–246).

There were two prominent antecedents of 'repression' in this case: horror of the trenches culminating in the death of a buddy, and domestic anxieties.

In summary, Brown based his view of posttraumatic dissociation on the Freudian conception with

inadequate repression the prime cause. Myers and McDougall instead assigned 'repression' a maintenance role preventing dissolution of dissociation rather than initiating it. As with Janet, they ascribed dissociation to 'overwhelming shock' caused by the emotional impact for example of exploding shells.

Discussion

By the end of WW I a considerable body of diagnostic data on combat trauma and posttraumatic amnesia had accrued. This data was repeatedly confirmed and then neglected. Ingraham and Manning [48] wrote: 'The problem of battle stress has been "solved" too many times this century, only to reappear where and when least expected' (p. 60).

The historical data not only corroborates modern studies of military trauma (e.g. [49]) but also illuminates and confirms the impact of trauma in civilian settings. Again quoting Ingraham and Manning [48]: 'Symptoms in many cases resembled those seen in civilian practice, but coloured to be sure, in distinctive ways by the precipitating events of war' (p. 28).

Both military and civilian trauma manifest comparable syndromes of posttraumatic stress, characterised by either partial or complete amnesia, or by hypermnesia. Thus, the historical findings are an important source of data informing the current debate on whether amnesia for childhood sexual trauma can occur, and whether traumatic memories can be lost and either spontaneously or therapeutically recovered.

The most important findings of the present study are the validation of posttraumatic amnesia and recovered memories, and the preliminary elucidation of their psychogenic basis. Two lines of evidence spelled the demise of the initial neuropathological model. As Ingraham and Manning [48, p. 29] stated: 'First, patients with head wounds and central nervous system damage seldom exhibited the symptoms; the disorder was almost invariable the malady of the unwounded. Second, prisoners of war evidenced no symptoms despite enduring the same shelling as their captors. The explanation, therefore had to be mainly psychological, not neuropathological'.

It rapidly became clear that peri-traumatic dissociation led to acute memory loss for part or even the entire traumatic experience. Memories were not abolished but could be recovered involuntarily, either in hypermnestic re-experiencing phenomena such as flashbacks, or under hypnosis. These historical findings challenge those of contemporary critics of functional posttraumatic amnesia (e.g. [50]), who argue

for intentional rather than involuntary suppression, and for loss of peripheral rather than central details of trauma.

In World War I studies of traumatic memories, however, memories were recovered, were supported by very high external validity. Such findings provide historical support for the validity and reliability of recovered memories of present day trauma, be it combat trauma or child sexual abuse. This is not to imply that restored memories cannot be ascribed to suggestion, but rather to counter any blanket assertion that traumatic memory is generally fabricated, or lacking in external validation.

Functional amnesia in WW I soldiers was best explained by the Janetian dissociation model. Posttraumatic amnesia comprised memory loss, not only for combat trauma, but also for more extensive autobiographical data. Functional amnesia today classified in DSM-IV [30] as dissociative amnesia, while loss of identity, often associated with wandering far afield, as dissociative fugue.

Traumatic memories of amnestic WW I combatants were vividly and uncontrollably relived in re-experiencing phenomena, including somnambulistic crises, hysterical fits and fugue states. They could also be accessed under hypnosis. Such findings corroborate those of contemporary theorists and their antecedents, particularly Pierre Janet. All assert that dissociated traumatic memories are re-experienced and reproduced as if reliving actual events, with all the cognitive, emotional, somatosensory and behavioural concomitants, rather than being derived from narrative reconstructions [51–55]. These findings counter those modern critiques which fail to differentiate traumatic from narrative memory.

The dimensions of dissociation of traumatic memories, including predisposition, psychogenesis and the relationship to the development of acute and chronic posttraumatic states are only beginning to be elucidated. According to Gabriel [4, p.4]: 'History teaches that no matter how well trained the soldier is, technically proficient his leaders are, men in battle will succumb to the stresses and strains inflicted upon them by their horribly destructive environment. Indeed, military history amply demonstrates that no one is immune to battle stress'. However, during WW I it was mostly assumed that, given the functional nature of shell shock, dissociation was the direct consequence of constitutional makeup [48, p.29]. Subjects were regarded as manifesting congenital psychological weakness. This was contrary to the teaching of Janet [6,56], who espoused a

biopsychosocial view well in advance of the contemporary version proposed by Engel [57]. Janet recognised the contribution of both biological and psychosocial factors to the dissociative weakening of ego integrative functions which results from acute traumatic stress [58–60]. However, it was then unclear why, under identical conditions, some individuals do not develop these posttraumatic disorders.

The paucity of follow-up of WW I cases means that less light can be shed on the relationship between combat trauma and subsequent development of traumatic stress disorders, and on the factor of chronicity. Thus, it would be of interest to know whether the contemporary finding relating peri-traumatic dissociation to the subsequent development of posttraumatic stress disorder [61,62] holds for the WW I sample. Unfortunately, the present study was unable to provide evidence for this since systematic follow-up of the reported cases is generally lacking in the literature.

Conclusion

Posttraumatic amnesia and concomitant somatosensory deficits in traumatized WW I soldiers had, at least in part, a functional origin. Memories were not abolished, but could be recovered therapeutically (e.g. under hypnosis). As memories were restored, somatosensory functions were also recovered. Memory loss was both anterograde and retrograde, pertaining to both traumatic and non-traumatic autobiographical data. Considerable evidence has accrued from these WWI studies, and from others to date, for a dynamic dissociative basis for posttraumatic amnesia in combat soldiers, and for traumatised individuals in general. The evidence also shows that memories, often externally validated, can be recovered in treatment. This evidence lends support to the notion of a common experiential and psychopathological mechanism for psychological trauma, extending to civilian populations and to childhood sexual abuse.

Many questions remain to be answered, not the least the psychobiological basis of dissociation, factors which predispose and those which protect from dissociation, and the specific factors which determine the diverse forms of posttraumatic psychological disorder.

Acknowledgement

References

1. Trimble MR. *Post-traumatic stress: from railway spine to whiplash.* Chichester: Wiley, 1981.
2. Freud S. (1897). Letter in: *The origins of psychoanalysis. Letters to Wilhelm Fliess, drafts and notes: 1887–1902.* London: Imago, 1954.
3. Masson JM. *The assault on truth: Freud's suppression of the seduction theory.* New York: Farrar, Straus and Giroux, 1984.
4. Gabriel RA. *No more heroes: madness and psychiatry in war.* New York: Hill and Wang, 1987.
5. Karon BP, Widener AJ. Repressed memories and World War II. lest we forget. *Professional Psychology: Research and Practice* 1997; 28:338–340.
6. Janet P. L'Amnésie et la dissociation des souvenirs par l'émotion. *Journal de Psychologie* 1904; 4:417–453.
7. Van der Hart O, Nijenhuis E. Amnesia for traumatic experiences. *Hypnos* 1995; 22:73–86.
8. Schefflen AW, Brown D. Repressed memory or dissociative amnesia: what the science says. *Journal of Psychiatry and Law* 1996; 24:143–188.
9. Elliott DM. Traumatic events: prevalence and delayed recall in the general population. *Journal of Consulting and Clinical Psychology* 1997; 65:811–820.
10. Janet P. Psychoanalysis. Report to the Section of Psychiatry, XVIIth International Congress of Medicine, London, 1913. London: Oxford University Press/Hodder and Stoughton, 1913.
11. Rivers WHR. *Instinct and the unconscious: a contribution to a biological theory of the psycho-neuroses.* Cambridge: Cambridge University Press, 1920.
12. Van der Hart O, Steele K, Boon S, Brown P. The treatment of traumatic memories: synthesis, realization and integration. *Dissociation* 1993; 6:162–180.
13. Rows RG. Neurasthenia and war neuroses. In: MacPherson WG, ed. *History of the Great War, based on official documents: medical services, diseases of the War, ii.* London, 1923.
14. Bailey P, Williams FE, Koroma P. *The medical department of the US army in the World War,* Vol. 10, *Neuropsychiatry.* Washington, DC: US Government Printing Office, 1929.
15. Brown W. *Psychology and Psychotherapy.* 3rd ed. London: Edward Arnold, 1934.
16. Brown W. Hypnosis, suggestion, and dissociation. *Lancet* 1919; 195:734–736.
17. Brown W. The revival of emotional memories and its therapeutic value. I. *British Journal of Medical Psychology* 1920–1921; 1:16–19.
18. Eder MD. *War-shock: the psychoneuroses in war: psychology and treatment.* London: William Heinemann, 1917.
19. Hart B. *Psychopathology: its development and its place in medicine.* New York: Macmillan, 1927.
20. Kardiner A. *The traumatic neurosis of war.* New York: P. Hoeber, 1941.
21. McCurdy JT. *War neuroses.* Cambridge: Cambridge University Press, 1918.
22. McDougall W. The revival of emotional memories and its therapeutic value III. *British Journal of Medical Psychology* 1920–1921; 1:23–29.

23. McDougall W. *An outline of abnormal psychology*. London: Methuen, 1926.
24. Miller E. *The neuroses in war.* London: Macmillan, 1940.
25. Myers CS. Contributions to the study of war shock: an account of certain cases treated by hypnosis. *Lancet* 1916; 192:65–68.
26. Myers CS. *Shell shock in France 1914–18.* Cambridge: Cambridge University Press, 1940.
27. Roussy G, Lhermitte J. *Les psychonévroses de guerre.* Paris: Masson, 1917.
28. Thom DA, Fenton MD. Amnesia in war cases. *American Journal of Insanity* 1920; 70:437–448.
29. Southard E. *Shell-shock and other neuropsychiatric problems presented in five hundred and eighty-nine case histories from the war literature 1914–18.* Boston: Leonard, 1919.
30. *Diagnostic and statistical manual of mental disorders.* 4th ed. Washington, DC: American Psychiatric Association, 1994.
31. Young A. *The harmony of illusions: inventing post trau-matic stress disorder.* Princeton: Princeton University Press, 1995.
32. Bassett Jones A, Llewelling J. *Malingering or the simula-tion of disease*. London: Heinemann, 1917.
33. Wolfsohn JM. War psychoneuroses. *Lancet* 1919; 2:177–180.
34. Bowman CM. Report of the examination of the Regiment, USA, for nervous and mental disease. *American Journal of Insanity* 1919; 74:555–567.
35. Mott FW. The effects of high explosives upon the central nervous system. *Lancet* 1916; 192:316–320.
36. Myers CS. The revival of emotional memories and its thera-peutic value II. *British Journal of Medical Psychology* 1920–1921; 1:20–22.
37. Brown W. The revival of emotional memories and its thera-peutic value IV. *British Journal of Medical Psychology* 1920–1921; 1:30–33.
38. Brown W. The treatment of cases of shell shock in an advanced neurological center. *Lancet* 1918; 17 August:197–200.
39. Jung CG. The question of the therapeutic value of 'abreac-tion'. *British Journal of Medical Psychology* 1921–1922; 2:13–22.
40. Janet P. *The major symptoms of hysteria.* New York: Macmillan, 1907.
41. Kihlstrom JF, Schacter DL. Functional disorders of autobio-graphical memory. In: Baddely A, Wilson B, Watts F, eds. *Handbook of memory disorders.* Chichester: Wiley, 1993.
42. Culpin M. *Recent advances in the study of the psychoneu-roses.* Philadelphia: Blackiston, 1931.
43. Janet P. *Etat mentale des hystériques.* Paris: Rueff, 1892/1894. (Trans: *The mental state of hystericals.* New York: Putnams, 1901. Reprinted: Washington, DC: University Publications of America, 1977.)

44. Simmel E. *Kriegsneurosen und 'Psychises trauma'.* Munich: Nemnich, 1918.
45. Rivers WHR. The repression of war experiences. *Lancet* 1918; 194:173–177.
46. Breuer J, Freud S. Studies on hysteria. In: Strachey J, ed., trans. *The standard edition of the complete works of Sigmund Freud,* Vol. 2. (1893–1895). London: Hogarth, 1955.
47. Janet P. *Les médications psychologiques.* 3 vols. Paris: Felix Alcan, 1919. English edition: *Psychological healing.* 2 vols. New York: Macmillan, 1925.
48. Ingraham L, Manning F. American military psychiatry. In: Gabriel RA, ed. *Military psychiatry: a comparative perspec-tive.* New York: Greenwood Press, 1986:25–66.
49. Belenky GL. Military psychiatry in the Israeli Defense Force. In: Gabriel RA, ed. *Military psychiatry: a compara-tive perspective.* New York: Greenwood Press, 1986:147–179.
50. Loftus EF. The reality of repressed memories. *American Psychologist* 1993; 48:518–537.
51. Brett EA, Ostroff V. Imagery and post-traumatic stress dis-order. *American Journal of Psychiatry* 1985; 142:417–424.
52. Janet P. *L'evolution de la mémoire.* Paris: Chahine, 1928.
53. Van der Hart O, ed. *Trauma, dissociatie en hypnose.* 3rd ed. Lisse: Swets and Zeitlinger, 1995.
54. Van der Kolk BA, Van der Hart O. The intrusive past: the flexibility of the memory and the engraving of trauma. *American Imago* 1991; 48:425–454.
55. Van der Kolk BA, Fisler R. Dissociation and the fragmen-tary nature of traumatic memories: overview and exploratory study. *Journal of Traumatic Stress* 1995; 8:505–526.
56. Janet P. Problèmes psychologiques de l'émotion. *Revue Neurologique* 1909; 17:1551–1687.
57. Engel GL. The need for a new medical model: a challenge for biological science. *Science* 1977; 196:129–136.
58. Van der Kolk BA, Van der Hart O. Pierre Janet and the breakdown of adaptation in psychological trauma. *American Journal of Psychiatry* 1989; 146:1530–1540.
59. Brown P. Pierre Janet: alienist reintegrated. *Current Opinion in Psychiatry* 1991; 4:389–395.
60. Brown P, Macmillan M, Meares R, Van der Hart O. Janet and Freud: revealing the roots of dynamic psychiatry. *Australian and New Zealand Journal of Psychiatry* 1996; 30:480–491.
61. Marmar CR, Weiss DS, Schlenger WE *et al.* Peritraumatic dissociation and posttraumatic stress in male Vietnam theater veterans. *American Journal of Psychiatry* 1994; 151:902–907.
62. Van der Kolk BA, Van der Hart O, Marmar CR. Dissociation and information processing in posttraumatic stress disorder. In: Van der Kolk BA, McFarlane AC, Weisaeth L. *Traumatic stress.* New York: Guilford, 1996:303–327.