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Scientific Review

Post traumatic stress disorder and spinal cord injuries

Paul Kennedy*,[1] and Jane Duff[1]

[1]*National Spinal Injuries Centre, Stoke Mandeville Hospital NHS Trust, Aylesbury, Bucks HP21 8AL, UK*

Post Traumatic Stress Disorder (PTSD) was first recognised by psychiatric international classification systems in 1980 and a wealth of research and treatment literature has developed since. This paper provides a review of PTSD and Spinal Cord Injuries. A brief history of the disorder is provided before descriptions of the defining characteristics, assessment and differential diagnoses. The paper provides an overview of the incidence and prevalence of PTSD and risk factors within the general population, before considering both veteran and non-veteran research within spinal cord injuries. Pharmacological and psychological approaches to the treatment of PTSD are also discussed. The review closes with recommendations for future research into the prevalence and treatment of PTSD in spinal cord injuries.
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Introduction

The syndrome of Post Traumatic Stress Disorder (PTSD) as it is currently known was first described in the Diagnostic and Statistical Manual of Mental Disorders by the American Psychiatric Association in 1980. Whilst this disorder is one of the newest entries to psychiatric nosology, it was first described as traumatic neurosis by Seguin[1] who advocated the term to replace Railway Brain and Railway Spine (both of which came from the considerable number of railway accidents) and compensation neurosis. Freud and Breuer[2] developed the conceptualisation further, which dominated thinking during World War I. Dissatisfaction with the Freudian focus on early developmental experience renewed interest in the condition which went through a variety of terms that included shellshock, gross stress reaction and combat neurosis. Research into the psychological effects of the wars, natural disasters and violence of the 20th century contributed to the recognition of the syndrome as post-traumatic stress disorder in 1980. Following exposure to a traumatic event of exceptional severity which involves actual or threatened harm and is associated with fear, helplessness or horror, some people develop symptoms of intrusive re-experiencing of the trauma, avoidant behaviours and symptoms of increased physiological arousal.

Traumatic responses to the major incidents of the last century have been extensively studied. Swank[3] examined combat exposure in World War II. Kleber *et al*[4] reported that by 1991 over 500 papers had been published on the psychological effects of the US war in Vietnam. The psychological effects of natural disasters have also been examined, from James'[5] observations of the San Francisco earthquake, the Ecuadorian earthquakes of 1987[6] and Hurricane Andrew.[7] De Silva[8] reviews traumatic reactions to other disasters, including violence, accidents, concentration camp experiences and torture.

Whilst the nomenclature is new, there exist early Greek and Egyptian descriptions of the impact of war and many Shakespearean plays refer to acute stress reactions. However, it is Samuel Pepy's diary entries around the time of the Great Fire of London in 1666 that provide what Daly[9] describes as an excellent record of the development of post traumatic stress disorder.

The diagnosis of post-traumatic stress disorder

Post-traumatic stress disorder is classified as an anxiety disorder and is recognised by international psychiatric nosological systems. As mentioned, it was first recognised in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III).[10] Yule *et al*[11] report that following further research, the criteria whereby the disorder was defined was revised in DSM-III-R[12] and again in DSM-IV.[13] The most recent World Health Organisation's Classification of Mental and Behavioural Disorders (ICD-10)[14] recognised two

*Correspondence: P Kennedy

reactions to acute stress; one which was transient and was reported as an acute reaction to stress, and the other which was considered an adjustment reaction which lasted longer. Yule *et al*[11] report that whilst the ICD-10[14] criteria for PTSD are similar to those of DSM-IV,[13] both involve identification of a threatening event which is thought necessary in the onset of the disorder.

However, there are some subtle differences between the diagnostic approach of the two systems. ICD provides an overall approach, whereas DSM provides a more mechanistic set of guidelines. ICD prefers that only one diagnosis be given to the patient, whereas DSM encourages the making of multiple diagnosis and acknowledges the degree of co-morbidity. The biggest single difference lies in the emphasis placed on emotional numbing. ICD sees this as a frequent accompaniment to PTSD, but not being necessary, whereas DSM regards this as one of the criterion characteristics. The two official definitions are provided in Tables 1 and 2 respectively.

Davidson and Foa[15] reported that until recently it was not unusual to encounter some skepticism as to the validity of the condition. Some claimed that PTSD was mainly a form of malingering or the expression of another psychopathological disorder. They report that since its introduction in 1980, it has become the subject of extensive literature carrying risks of chronicity, morbidity, mortality and increased physical and psychiatric disturbances. The body of information about the subject has grown rapidly.[16] Yehuda and McFarlane[17] report that in the last decade of the 20th century the biological concomitants of PTSD have provided objective validation that it is more than a political or social conceptualisation of human suffering. In their extensive review of the psychobiology of PTSD, they and their co-writers document the neuroendocrinology, psychophysiology, neurochemistry and neuroanatomy of traumatic stress reactions.

Differential diagnosis

Jones and Barlow[18] conclude that PTSD is clearly differentiated from other anxieties (with a possible exception of some simple phobias) as the anxiety is centred around cues associated with the original trauma and these cues serve as the triggers for alarms. There is also more dissociation present in severe cases of PTSD than anxiety disorders. Acute Stress Reaction (ASR) is an ICD 10 diagnosis, has a similar etiology to PTSD but is differentiated on the basis of temporal resolution, as the minimum duration for ASR is at least 2 days, but nor more than 1 week.

DSM-IV[13] also recognised and introduced the diagnosis of Acute Stress Disorder (ASD). Although similar to PTSD in relation to the symptoms of re-experiencing, avoidance and arousal, two differences distinguish it from PTSD: the onset of symptoms 2–4 weeks post trauma, and dissociative symptomatology.[19] Harvey and Bryant[20] concluded that a full diagnosis of ASD is highly predictive for the development of chronic PTSD. In ASD, dissociative symptoms (such as numbing, depersonalisation and amnesia) are essential diagnostic criteria, but in PTSD they are not. Adjustment disorders are a state of subjective distress and emotional disturbance, usually interfering with social functioning and performance and occur in response to a significant life change (such as a spinal cord injury). They usually last for 1 month and symptoms rarely exceed 6 months. Persons with chronic PTSD are more likely than those with acute PTSD to have co-morbid psychiatric disorders rather than other medical conditions.

Comorbidity

People who experience PTSD symptoms often meet the criteria for at least one other psychiatric diagnosis. Brady[21] reported that 80% of individuals with PTSD met the criteria for at least one other psychiatric diagnosis, with the most common being depression.[22,23] Simon[24] suggested that other co-morbid associations, including dissociative disorder, anxiety, panic disorder, drug abuse/dependence and neuroticism, have been found to be associated with vulnerability to chronic PTSD.

Assessment

Yule *et al*[11] argue that there is no substitute for an in-depth clinical interview when obtaining information both for the purposes of making a diagnosis and planning treatment. This clinical interview should be carried out by a well-trained mental health professional. In addition to diagnostic interviews, there are a number of semi-structured interviews that assess PTSD symptoms. Watson[25] reviews the psychometric measurement techniques of PTSD. The Clinician Administered PTSD Scale[26] is reported by Yule *et al*[11] to be the most useful. The Structured Clinical Interview for DSM (SCID) developed by Spitzer *et al*[27] is also frequently used. Other measures that were specifically developed to identify the main features of PTSD include the Impact of Event Scale developed by Horowitz *et al*.[28] This scale identifies the two main features of PTSD, ie intrusion and avoidance and is the measure used by the authors in assessing PTSD symptoms with a spinal cord injured population. It is the single most widely used instrument for assessing the psychological consequences of traumatic events.[29]

Keane[29] recommends the following five steps when assessing PTSD. The first involves the conduct of a standard comprehensive detailed clinical examination that also includes information on the traumatic event which should be assessed in detail. The second relates to the use of the structured clinical interview and explores the possible ICD/DSM diagnoses that may be applicable. Thirdly, the use of general personality questionnaires to provide information on general

Table 1 DSM-IV criteria for post-traumatic stress disorder

A. The person has been exposed to a traumatic event in which both the following were present:
   (1) The person experienced, witnessed or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others.
   (2) The person's response involved fear, helplessness or horror. *Note:* In children, this may be expressed instead by disorganised or agitated behaviour.
B. The traumatic event is persistently re-experienced in one (or more) of the following ways:
   (1) Recurrent and intrusive distressing recollections of the event, including images, thoughts or perceptions. *Note:* In young children, repetitive play may occur in which themes or aspects of the trauma are expressed.
   (2) Recurrent distressing dreams of the event. *Note:* In children, there may be frightening dreams without recognisable content.
   (3) Acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations and dissociative flash back episodes, including those that occur on awakening or when intoxicated). *Note:* In young children, trauma-specific re-enactment may occur.
   (4) Intense psychological distress at exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.
   (5) Physiological reactivity on exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.
C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:
   (1) Efforts to avoid thoughts, feelings or conversations associated with the trauma.
   (2) Efforts to avoid activities, places or people that arouse recollections of the trauma.
   (3) Inability to recall an important aspect of the trauma.
   (4) Markedly diminished interest or participation in significant activities.
   (5) Feeling of detachment or estrangement from others.
   (6) Restricted range of affect (eg unable to have loving feelings).
   (7) Sense of foreshortened future (eg does not expect to have a career, marriage, children or a normal life span).
D. Persistent symptoms of increased arousal (not present before the trauma) as indicated by two (or more) of the following:
   (1) Difficulty falling or staying asleep.
   (2) Irritability or outbursts of anger.
   (3) Difficulty concentrating.
   (4) Hypervigilance.
   (5) Exaggerated startle response.
E. Duration of the disturbance (symptons in criteria B, C and D) is more than 1 month.
F. The disturbance causes clinically significant distress or impairment in social, occupational or other important areas of functioning.

Specify if:
Acute: if duration of symptoms is less than 3 months.
Chronic: if duration of symptoms is 3 months or more.

Specify if:
With delayed onset: if onset of symptoms is at least 6 months after the stressor.

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Table 2 ICD-10 criteria for post-traumatic stress disorder

This disorder should not generally be diagnosed unless there is evidence that it arose within 6 months of a traumatic event of exceptional severity. A 'probable' diagnosis might still be possible if the delay between the event and the onset was longer than 6 months, provided that the clinical manifestations are typical and no alternative identification of the disorder (eg as an anxiety or obsessive-compulsive disorder or depressive episode) is plausible. In addition to evidence of trauma, there must be a repetitive, intrusive recollection or re-enactment of the event in memories, daytime imagery or dreams. Conspicuous emotional detachment, numbing of feeling and avoidance of stimuli that might arouse recollection of the trauma are often present but are not essential for the diagnosis. The autonomic disturbances, mood disorder and behavioural abnormalities all contribute to the diagnosis but are not of prime importance.

The late chronic sequelae of devastating stress, ie those manifest decades after the stress experience, should be classified under F62.0.

Includes: traumatic neurosis.

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functioning, fourthly the administration of specific tests to directly measure PTSD and its associated clinical features such as those already referred to earlier. Finally, the inclusion of measures of social role functioning to determine the extent of social and vocational impairment.

Other measures include the Keane PTSD Scale of the Minnesota Multi-Phasic Personality Inventory (MMPI),[30] the Penn Inventory for PTSD[31] and Saunders *et al*[32] derivation from the Symptom Check List-90 (SCL-90).[33]

The evaluation of malingering in post traumatic stress disorder

A diagnosis of PTSD is based almost entirely on the patient's report of subjective symptoms. It is beyond our brief to speculate as to the motivation for malingering, but Keiser[34] suggests financial compensation, sympathy and social support. It is thought that pure malingering is uncommon in PTSD cases, but exaggeration of symptoms is more likely. Resnick[35] proposes that special interview techniques and psychological testing should be considered when clinicians suspect malingering. The MMPI[36] is the most validated psychological test to ascertain malingering and mental illness and Chaney *et al*[37] found that the MMPI can be helpful in distinguishing claimants with true PTSD from those with functional disorders.

In addition, Resnick[35] believes that the following criteria may raise questions concerning reliability of the clinical interview. These include: inconsistency of symptom presentation; evasiveness; over-idealisation of functioning before the trauma; anti-social personality traits; unvarying repetitive dreams; discrepant capacity for work and recreation; prior 'incapacitating' injuries; and poor employment record. This is a sensitive area which underlines the need for comprehensive and thorough approaches to history taking and assessment.

Epidemiology: incidence and prevalence

Since 1980 a number of research studies have sought to estimate the prevalence of PTSD within the general population and clinical groups. However, such estimations are limited by variation in the criterion for diagnosis and the sampling bias of many studies. Nevertheless, in a large review of epidemiological studies, Breslau[38] suggests that the conditional risk, ie the probability of PTSD among those exposed to trauma, is between 10% to 15%. However, the conditional risk varies by type of trauma and, of course, its frequency. One of the main conclusions of the epidemiological studies is that road traffic accidents, whilst not the most frequent event or most traumatising, have the most adverse combination of frequency and impact. Indeed, Norris[39] suggested that it was 'the single most significant event' (p. 416) in her epidemiological study. Norris[39] reported a lifetime frequency of 23% (ie the proportion of the population who will experience a RTA) with a PTSD rate of 12%, and comments that this translates to 28 seriously distressed persons per 1000 adults in the United States.

Given that approximately 50% of people who sustain a spinal cord injury do so through road traffic accidents[40] and the occurrence of PTSD from non-traumatic aetiologies, such as surgery,[41–43] the incidence of PTSD following traumatic and non-traumatic spinal cord injury requires further attention.

What is clear from the epidemiological research is that while many people experience traumatic events, exposure to an event is not sufficient for subsequent development of PTSD. Research with clinical populations following road traffic accidents have recorded incidences for PTSD of 0% to 100%. However, in a review of the area, Blaszczynski *et al*[44] suggest an average rate of about 40%, with 80% of symptoms emerging within 2 months of the trauma. Mayou *et al*[45] examined the incidence of PTSD following whiplash injury. Acute and moderately severe distress was common, 25% fulfilled the ICD-10 criteria for acute stress syndrome and 10% the criteria for PTSD, and the incidence was greatest in motorcyclists. Ehlers, *et al*[46] in a study of consecutive attenders at an Accident and Emergency department following a road traffic accident, reported a rate of 23.1% at 3 months and 16.5% at 1 year post injury using DSM-IV criteria. More importantly, they found that participants who met the criteria at 3 months had a 50.3% chance of still experiencing PTSD at 1 year. There is some evidence that sustaining a physical injury increases the risk of developing symptoms of PTSD.[47,48]

Few studies have investigated the timeline of PTSD and the factors that contribute to chronicity. Much of this research has concerned war veterans. Rates of PTSD in World War II POWs were reported to be 48% to 60% immediately after the war and from 29% to 48% 40 years later.[49–51] In one of the benchmark studies of trauma, Kulka *et al*[23] as reported in Breslau,[38] reported that 31% of their sample had combat related PTSD and half of these cases continued to meet the diagnostic criteria 20 years later. Breslau and Davis[52] considered the timeline in an epidemiological study. They reported that more than three quarters of those who met the criteria for PTSD continued to do so 6 months following the event, and 57% continued to meet the criteria at 1 year. Crucially, they found that spontaneous remission tapered off beyond 1 year. In addition, they found that those with chronic PTSD were at greater risk of developing other disorders, such as a major depressive episode or anxiety, as discussed earlier. Brelau[38] suggested that symptoms of PTSD last longer in women than men, and longest when the trauma is directly experienced. Greene[53] concludes that there is clear evidence that PTSD is a long lasting disorder, with up to a half of those who develop the disorder having it decades later without treatment.

A consistent finding of the general population studies is that the conditional risk for women is approximately twice that of men.[39,54,55] However, this finding may be affected by differences in reporting and pre-existing mental health. Breslau *et al*[56] in an attempt to illuminate these differences, found

similarities in the lifetime prevalence of traumatic events between men and women, but that risk of PTSD increased markedly if an individual was exposed to trauma in childhood. In their study, a greater proportion of women reported sexual or physical abuse in childhood, whilst men reported a greater proportion of exposure to serious accident and injury. The latter trauma did not lead to PTSD in either group.

The incidence of PTSD in children exposed to trauma is far from conclusive, with prevalence figures ranging from 0% to 100%.[57] However, Perry and Azad[58] in a review of the literature, suggested an incidence rate of 30%, therefore approximately 1.5 million children (if >5 million children are exposed to traumatic events) will meet diagnostic criteria for PTSD. In addition, they suggest that a further 30% experience clinically significant symptoms, but do not meet the full criteria. Stallard *et al*[59] examined the incidence of PTSD in children following road traffic accidents with a control group who sustained sports injuries. Both groups were admitted through an Accident and Emergency department. Thirty-four per cent of children involved in a road traffic accident met the criteria for PTSD, compared to only 3% of the children who sustained a sports injury.

Very little research has compared the relative incidence of PTSD across the age range. However, Thompson *et al*[60] in research of survivors of Hurricane Hugo, suggested that the middle-aged, rather than young or old, were at the most risk of PTSD. They review four theories of coping capacity and conclude that it is the imbalance between the emotional, financial and societal support received and provided by the middle-aged that influence the increase risk. Weintraub and Ruskin[61] in a review of PTSD in the elderly, comment that old age alone does not place a person at increased risk, but emphasise that clinicians should be aware of underreporting and not rule out a diagnosis because of previous good psychosocial functioning.

Although a number of general risk factors which predispose individuals to developing PTSD have been identified (such as gender and previous mental health problems), these only account for a small proportion of the risk. More significant influences on the development of PTSD concern an individual's interpretation of the event, its personal significance and the perception of threat. (See[38,44,62] for good reviews of risk factors for PTSD).

Spinal cord injury

PTSD following spinal cord injury has received remarkably little research attention. Johnson[63] appears to provide the first published report, citing a 15-patient study by McFall, Umlauf, Roszell and Malas, which found little evidence of PTSD. However, Radnitz and colleagues at the Veterans Affairs Medical Centers have found evidence of PTSD.

Following spinal cord injury, Radnitz *et al*[64] found that 11% of their sample met the diagnosis for current PTSD and 29% the criteria for lifetime PTSD. In addition, a number of the sample demonstrated symptoms of PTSD, but did not meet the full criteria (28% for current PTSD and 41% for lifetime PTSD). It is important to note that the injury demographics of their sample is similar to that of the general spinal cord injured population, with only a small proportion sustaining an injury through combat related violence (8%). However, the impact of prior trauma on the development of PTSD within the veteran population requires careful consideration.

Radnitz *et al*[65] examined factors predictive of PTSD following spinal cord injury. They found that the recency of trauma was not a significant predictor of either severity or diagnosis, and suggested that symptoms of PTSD may not emerge until several years after the event. Differences were found between level of injury, with veterans with paraplegia demonstrating significantly greatly symptomatology than those with quadriplegia. Radnitz *et al*[66] examined level of injury in greater detail, comparing veterans with paraplegia and quadriplegia with non-injured veteran controls. They found similar levels of current PTSD for veterans with paraplegia and non-injured veteran controls (22% and 21% respectively), only 2% of the quadriplegic sample meeting the diagnosis. However, the incidence of lifetime PTSD in those with paraplegia was higher than the non-injured controls (44% compared to 26%), and was again lowest in those with quadriplegia (13%). Binks *et al*[67] considered whether a particular level of paraplegia distinguished the development of PTSD and found that injuries above T1–T3 were less likely to be diagnosed with current PTSD than those with injuries below this area. The explanation of Radnitz *et al*[66] and Binks *et al*[67] for these differences concerns the psychophysiological components of PTSD. They suggest that the nerve fibres responsible for sympathetic arousal may be impaired in higher injuries with the memory of emotional events being modulated through peripheral nervous system activity.[68]

Kennedy and Evans[69] found that 20% of the sample of traumatic spinal cord injuries scored above the clinical cut-off on the Impact of Event Scale and furthermore identified that there is no difference in the prevalence in the sample between those who had a post-traumatic amnesia and those who had not. Duff[70] found a similar level of PTSD following spinal cord injury. Repeated measurement revealed relationships between PTSD symptomatology, mood and coping over time.

In perhaps the most extensive study to date with spinal cord injury, Znoj and Lude[71] examined the contribution of distress (using the Impact of Event Scale), emotional regulation and perceived disability with 264 persons with spinal cord injury. They found that the incidence of PTSD type symptoms was similar to that found by previous studies[69] and

lower than levels found following trauma such as rape.[72]

Recently published research has considered the role of violence in the development of PTSD following spinal cord injury. Adkins *et al*[73] comment on the dramatic rise in violence related injuries in the USA, the incidence more than doubling between 1973 and 1994. They did not find significant differences between those that sustained a spinal cord injury through RTA or gunshot, but did find that those with most prior exposure to violence and crime had greater symptoms of both depression and PTSD.

In relation to children who sustain a spinal cord injury, 33% of Boyer *et al*[74] sample exhibited symptoms that had a diagnostic criteria for PTSD, with an additional 19% meeting two of the diagnostic criteria. Children injured through gunshot and violence were significantly more likely to exhibit symptoms than those injured through road traffic accidents. Contrary to Radnitz *et al*,[66] Boyer *et al*[74] found no difference between level of injury and PTSD diagnosis, but tetraplegic patients were significantly more likely to have higher levels of avoidance. Recent research has also shown that parents of children who sustain spinal cord injury can also develop PTSD[75] and the impact that PTSD can have upon children's level of functional independence.[76]

Between 40% and 50% of those who sustain a spinal cord injury also sustain a closed head injury.[77,78] Radnitz *et al*[65] found that sustaining a concomitant head injury correlated with PTSD severity. They suggested that the occurrence of head injury during trauma was a risk factor for increased PTSD symptomatology but emphasise the need for differential diagnosis between PTSD and postconcussive syndrome. There is much debate whether PTSD can occur in closed head injury. Sbordone and Liter[79] found no evidence of PTSD in their sample of 70 participants and suggested that head injury is mutually incompatible with PTSD because of lack of recall for the event. However, other studies have documented PTSD. Bryant and Harvey[80] found that 13.9% of their sample of adults who had sustained a mild traumatic brain injury met the criteria for Acute Stress Disorder, with 23.8% meeting the criteria at 6 months. They cite a number of other studies and estimate the frequency of PTSD to be between 17% and 33% following mild traumatic brain injury.

Chemtob *et al*[81] found that combat veterans with a diagnosis of PTSD were significantly more likely (1.67 times) to report a history of head injury, half of whom had lost consciousness. Those who sustained a head injury also had more severe symptoms of PTSD. However, the study employed a self-report measure of head injury and was unable to comment whether the head injury and PTSD occurred at the same time. Bryant and Harvey[82] found that post-concussive symptoms were more evident in mild traumatic brain injured patients with PTSD than those without PTSD. Twenty per cent of the mild traumatic brain injured patients were diagnosed with PTSD and 25% of non-traumatic brain injured patients. They found no correlation between the length of post traumatic amnesia and PTSD in the brain injured group and suggest that the degree of PTSD contributed to persistent post-concussional syndrome and compounds the neurological effects of mild traumatic brain injury. Sbordone,[83] in a review of the acute stress disorder literature and in an attempt to close the debate, suggests that brain injured patients can develop PTSD following exposure to events, but that the events need either to have occurred prior to the onset of retrograde amnesia or after cessation of anterograde amnesia (PTA). Sbordone[83] comments that a too liberal definition of mild closed head injury is used in many studies and recommends instead the use of DSM-IV diagnostic criteria for post-concussional disorder. Radnitz *et al*[65] also comment that there are difficulties in diagnosing PTSD following head injury because the cutoffs utilised by many measures do not correspond to the differential in symptom sequelae of a head injury.

The treatment of PTSD

It is beyond the scope of this article to provide a full review of the pharmacological and psychological treatment approaches for PTSD. However, a brief overview of the evidence base and research for the management of PTSD is provided.

Pharmacological treatment

There is a paucity of randomised clinical drug trials for the treatment of PTSD compared to other conditions, with only nine trials being published, the first of which was in 1988. Most trials have considered the use of antidepressants (tricyclic antidepressants, monoamine oxidase inhibitors (MAOIs), selective serotonin reuptake inhibitors (SSRIs) and benzodiazepine) *versus* placebo, and have been mainly used on military populations. In reviewing the three randomised trials which have had clinically significant effect sizes, Friedman[84] suggests that although SSRIs have emerged as the first choice for treatment, the most efficacious approach may be a combination of SSRIs and MAOIs. However, the prescription of MAOIs tends to be limited by the strict adherence requirements concerning diet, the use of alcohol and illicit substances. McIvor and Turner[85] state that the trials so far indicate symptom reduction to occur only when medication is used for longer than eight weeks. Use of SSRIs and MAOIs in spinal cord injury is rare because of autonomic instability and polypharmacy, particularly in the early stages. Any decision to use the above would require careful assessment and an individually orientated approach.

Friedman[84] provides a good review of the literature and treatment recommendations, but comments that most of the drug trials do not exceed the threshold for

clinically noticeable difference in symptomatology. This is contrasted sharply with the effect size published by randomised exposure or cognitive-behaviour therapy treatments. McIvor and Turner,[85] in their review of the area, comment that psychological treatment trials have indicated a greater level of symptom reduction than drug treatment to date, but suggest that pharmacotherapy may be particularly useful in the presence of a comorbid condition such as depression.

Psychological treatment

Blake and Sonnenburg,[86] in their review of psychological literature for the treatment of PTSD, document eleven published approaches from a variety of theoretical backgrounds. For the purpose of this review, the outcome literature for psychodynamic psychotherapy, hypnotherapy and cognitive behavioural therapy is considered. For a more detailed review of psychological treatment approaches see Foa and Meadows.[87]

The evidence base for psychodynamic psychotherapy and hypnotherapy is mainly confined to case studies. Both have shown success in treating PTSD but there is a paucity of rigorous published clinical outcome trials. Hypnotherapy has been used to treat combat stress reactions[88] (cited in Sbordone[83]), but has limitations because some patients are resistant to hypnosis for fear of losing control, and others may respond by developing dissociative states[89] (cited in Sbordone[83]). Brom *et al*[90] conducted one of the few controlled trials comparing hypnosis, psychodynamic psychotherapy and desensitisation approaches with waiting list controls. Participants who developed PTSD following bereavement were randomly assigned to the treatment modalities. The research used standardised measures but not blind evaluation of the treatments. Participants' symptoms of PTSD in all three of the treatment approaches showed greater improvement than those on the waiting list. Although there were no significant differences across the treatment modalities, desensitisation yielded the greatest mean symptom reduction.

Cognitive Behavioural Therapy (CBT) has yielded the greatest number and most rigorously controlled studies in this area. CBT is an umbrella term which encompasses several specific forms of therapy which differ in the emphasis each places upon the cognitive and behavioural components of treatment. The common aspects are behavioural change through exposure to the fear, cognitive restructuring techniques and anxiety management strategies. Behavioural and cognitive change is important because exposure to trauma typically leads to changes in thought processes and content and the symptoms of PTSD have a number of cognitive components.[86]

Cognitive behavioural strategies typically involve altering the way an individual perceives and understands situations and events. Exposure promotes symptom reduction through recollection of the trauma in an objectively safe environment so that an individual learns to cope with recalling the trauma without re-experiencing it. Therapy also involves an individual monitoring their thinking patterns and learning to recognise when their thinking is unrealistic and challenge these patterns. More appropriate, realistic and adaptive cognitions are then developed. In particular for PTSD, this might include restructuring an individual's appraisals of the event, their memory and recollections.

Although different strands of cognitive therapy have been developed, such as Cognitive Processing Therapy,[19] in practice these involve a blend of restructuring the meaning of the event and accompanying thought processes. Stress Innoculation Training[92] is a further common approach which is primarily designed to help an individual manage the anxiety symptoms of PTSD. When implementing this approach, care needs to be taken to ensure that the exposure component is sufficient to enable habituation to occur.

The efficacy of CBT interventions has been demonstrated by a large number of single case studies, uncontrolled trials, and more latterly, randomised trials with waiting list controls. Foa *et al*[93] conducted one of the most rigorous studies in this area, comparing imaginal exposure, SIT, supportive counselling and waiting list controls. All treatment conditions demonstrated significant reduction in symptomatology post treatment and at follow-up. Prolonged exposure and SIT were significantly superior to supportive counselling. Immediately post-treatment, SIT was superior to prolonged exposure, but this effect was reversed at follow-up with those who participated in exposure faring significantly better than those who utilised SIT techniques.

Marks *et al*[94] conducted a randomised trial examining lone cognitive restructuring and exposure treatment with a mixed cognitive restructuring/exposure condition and a placebo control. The results showed that all three treatment conditions were superior to the placebo control. Of particular note is that the combined treatment condition did not yield greater benefit than the stand-alone approaches. Tarrier and Humphreys,[95] in a randomised trial comparing cognitive therapy and imaginal exposure, found that both approaches displayed equally good results until those who failed to respond to treatment were excluded. Following this exclusion, patients who received imaginal exposure demonstrated significantly greater reduction in symptomatology than those who received cognitive therapy. One of the difficulties in generalising these results to a spinal cord injured population concerns participant sampling, as most acquired PTSD following sexual assault or combat. Bryant *et al*[96] examined the efficacy of CBT *versus* supportive counselling for acute stress disorder following road traffic and industrial accidents. Treatment in each condition was commenced within 2 weeks of the trauma. Following the brief CBT

programme, only 17% of the sample received a diagnosis of PTSD at 6 month follow up, compared to 67% of the supportive counselling sample.

One of the most recent psychotherapeutic approaches to be developed is Eye Movement Desensitization and Reprocessing (EMDR), originating in 1989.[97] It combines desensitisation and cognitive appraisal with saccadic eye movements and requires the patient to focus on a disturbing memory or image (including the related emotions, cognitions and physiological changes) while tracking the therapist's finger across their visual field. The treatment has evoked considerable controversy since its inception, and although case studies have reported positive findings, Foa and Meadows[87] comment that EMDR, like psychodynamic psychotherapy and hypnotherapy, lacks adequate controls, standardised measurement and blind evaluations. However, Shapiro[98] in a review of EMDR, details four controlled studies on the effectiveness of the approach post-treatment and cites evidence that 84% to 100% of the single trauma subjects are no longer diagnosed with PTSD.

From the research to date it is apparent that the greatest number and most rigorous trials have considered the efficacy of cognitive behavioural approaches. However, although the evidence base for CBT as a whole for the treatment of PTSD has been established, much of the research comparing different approaches has been inconclusive about which aspects of the treatment contribute to the change. Ehlers and Clark[99] provide an overview of the cognitive techniques they consider contribute to change, but have yet to establish the evidence base for the observed differences. Clark[100] reported effect sizes of 2.6 following treatment with CBT for PTSD. In addition, when considering the efficacy of the above studies it is important to note that to date there has been no published research concerning the treatment of PTSD following spinal cord injury, and only a few studies have considered the treatment of PTSD in populations who were exposed to trauma in circumstances akin to those in which a spinal cord injury may be sustained.

Conclusions

Research suggests that the prevalence of PTSD post spinal cord injury ranges from 10% to 40%. What is not clear is whether the PTSD is as a consequence of the trauma experienced which resulted in the spinal cord injury, or the spinal cord injury itself, or both. Future research needs to explore ways of clarifying these issues. It is likely that a more longitudinal investigation of PTSD type symptoms in the months and years following injury would help identify more accurately the actual incidence and prevalence of the disorder in the spinal cord injured population. Reseachers need to fully describe the stressor and explore ways of identifying appraisal processes. It is also important to fully assess the context, both in terms of the individual, their family and caregivers. Another avenue of future research would also include examination of the psychophysiological factors associated with spinal cord injury and PTSD. There is also a need to recognise the issue of comorbidity, not only in terms of prevalence of anxiety and depression, but also associated with the processes of adaptation and adjustment to a spinal cord injury, the coping propensities and the degree of social support.

A range of therapeutic interventions for the treatment of PTSD has been discussed. However, this review has identified a gap in research on the treatment of PTSD in a spinal cord injured population.

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