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## Posttraumatic Stress Disorder after Stroke: A Review of Quantitative Studies

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### 1. Introduction

Stroke is a sudden and devastating illness that occurs when the blood supply to the brain is cut off due to a blood clot or when blood vessels supplying the brain burst, thereby damaging or destroying brain cells. Stroke is the third most common cause of death after heart disease and all cancers in both the UK and the US. In the UK approximately 100,000 people have a stroke each year, with stroke causing over 50,000 deaths. Stroke predominantly affects older people, with almost 80% of first-time strokes occurring in people aged 65 years or older. Stroke is a leading cause of severe, long-term disability. Stroke survivors may experience a range of ongoing problems including weakness or paralysis, problems with balance and coordination, speech and language impairments (e.g., aphasia), cognitive and psychological problems, and emotional lability. As well as the direct care costs, which have been estimated to be £2.8 billion per annum, stroke accounts for £1.8 billion per annum in lost productivity and disability and £2.4 billion per annum in informal care costs in the UK. In the US, the direct and indirect costs of stroke for 2010 have been estimated to be \$73.3 billion (American Stroke Association, 2011; Stroke Association, 2011). Elevated levels of psychological distress have been documented after stroke, although research to date has focused almost exclusively on depression and general anxiety symptoms (e.g., Fure et al., 2006; Hackett et al., 2005; Leppavuo et al., 2003). However, stroke has many of the characteristics of events likely to trigger post-traumatic stress disorder (PTSD) symptoms in that it is unexpected, uncontrollable and potentially life-threatening (Field et al., 2008). Stroke is a “frightening experience” with the symptoms (e.g., weakness or numbness down one side of the body or face, problems with balance and coordination, problems with communication, confusion) appearing suddenly and without warning (Stroke Association, 2011). Thus, in addition to coping with chronic stressors that may arise from ongoing disability, stroke survivors also have to come to terms with the sudden, unexpected and life-threatening nature of the stroke event itself.

The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) (American Psychiatric Association, 1994, p. 424), defines PTSD as “the development of characteristic symptoms following exposure to an extreme traumatic stressor involving direct personal experience of an event that involves actual or threatened death or serious injury, or other threat to one’s

physical integrity" (Criterion A1) and is associated with feelings of intense fear, helplessness or horror (Criterion A2). The symptoms include "persistent reexperiencing of the traumatic event (Criterion B), persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (Criterion C), and persistent symptoms of increased arousal (Criterion D)". In addition, these symptoms must be present for at least one month (Criterion E) and have a significant negative impact on social, occupational or other areas of functioning (Criterion F). Research on PTSD has traditionally focused on traumas such as war, physical and sexual assaults, and road traffic accidents (Shalev et al., 1993). However, there has been growing recognition that PTSD symptoms may occur after a range of medical events (Tedstone & Tarrier, 2003), including cancer (e.g., Kangas et al., 2005), myocardial infarction (MI) (e.g., Kutz et al., 1994), and subarachnoid haemorrhage (e.g., Berry, 1998). Accordingly, life-threatening illnesses were added as an example of a traumatic event that may lead to the development of PTSD in DSM-IV (APA, 1994).

The experience of PTSD after stroke may have important implications for recovery. For example, PTSD has been related to poorer physical health in the general population (Spitzer et al., 2009) as well as to non-adherence to medication and adverse clinical outcomes in MI patients (Shemesh et al., 2001), worse functional recovery in patients with severe traumatic brain injury (Bryant et al., 2001), and higher levels of disability following hospitalization for physical injuries (e.g., resulting from road traffic accidents) (O'Donnell et al., 2009). PTSD is also likely to have a negative impact on stroke rehabilitation (Williams, 1997). First, the experience of intrusions may place high demands on the already limited cognitive resources of many stroke survivors, which may further restrict their ability to fully process, and come to terms with, the trauma experience. Second, survivors with PTSD are likely to try to avoid reminders of the stroke, which may hinder attempts to integrate them back into the community. Third, survivors with PTSD are likely to engage in catastrophic thinking and to have excessively negative perceptions of possible future harm which may further impede rehabilitation efforts.

Previous reviews have focused on the prevalence and correlates of PTSD following a range of life-threatening physical illnesses (e.g., Pedersen, 2001; Spindler & Pedersen, 2005; Tedstone & Tarrier, 2003). However, these reviews have either been very general in their scope (e.g., Tedstone & Tarrier, 2003) or have focused on specific medical conditions other than stroke (e.g., Spindler & Pedersen, 2005). In relation to the psychological consequences of stroke, reviews to date have only focused on the prevalence (Hackett et al., 2005) and the correlates (Hackett & Anderson, 2005) of depression after stroke. The main aims of the current review were to (i) assess the prevalence of PTSD after stroke, (ii) identify the main correlates of PTSD after stroke, (iii) highlight a range of methodological issues in research on PTSD after stroke, and (iv) make recommendations for future research.

## 2. Methods

The following electronic databases were searched in order to identify relevant studies to include in the review: Web of Knowledge, PsycINFO, and Medline. The searches were restricted to studies published between 1994 (the year life-threatening illnesses were included as an example of a traumatic event in the DSM) and May 2011. The following search terms were used: (i) stroke and cerebrovascular accident, and (ii) post-traumatic stress disorder, posttraumatic stress disorder, PTSD, Impact of Events Scale, IES, Penn, Post

Traumatic Stress Disorder Checklist, PCL, Posttraumatic Diagnostic Scale, PDS, Clinician-Administered PTSD Scale, CAPS, Structured Clinical Interview for DSM-IV, and SCID. Combinations of these two sets of search terms were searched using the Boolean operator "AND". In addition, the reference lists and citation histories of relevant articles were also examined in order to identify further studies to be included in the review. Studies on adult stroke survivors, with a self-report measure of PTSD symptomatology or a clinical interview to diagnose PTSD, that were published in English in peer-reviewed journals were included in the review. Single case studies, qualitative studies, papers without primary data (e.g., editorials), conference abstracts, dissertations, and studies on childhood stroke or subarachnoid haemorrhage were excluded.

The searches identified 411 articles. After applying the inclusion and exclusion criteria detailed above, 10 articles reporting 9 studies were included in the review; one study was reported in two articles (Sagen et al., 2009, 2010). The following data were extracted from each study (see Table 1): date of publication, country of origin, study design, recruitment site, number of patients screened and excluded, main exclusion criteria, response rate, first or recurrent stroke, sample size, age, gender, stroke location, time since stroke, assessment of PTSD, prevalence of PTSD, and significant correlates of PTSD symptom severity.

### **3. Results and discussion**

#### **3.1 Prevalence of PTSD after stroke**

Seven studies reported the prevalence of PTSD after stroke. There was considerable variability in the estimated prevalence of PTSD after stroke, which ranged from 3% (Sagen et al., 2010) to 31% (Bruggimann et al., 2006). This variation is likely to be due to differences in assessment methods, time since stroke, and recruitment procedures (e.g., exclusion criteria, response rates). Nonetheless, despite the heterogeneity in study designs and reported prevalence rates, research to date indicates that stroke survivors are at risk of developing PTSD in line with work on other life-threatening illnesses (Tedstone & TARRIER, 2003). For example, Spindler and Pedersen (2005) reported that the estimated prevalence rate for PTSD after heart disease ranged from 0% to 38%. The estimated prevalence of PTSD after stroke is higher than that found in large-scale community studies which have reported PTSD prevalence rates of less than 1% among older adults (Creamer & Parslow, 2008; Maercker et al., 2008; van Zelst et al., 2003).

#### **3.2 Correlates of PTSD after stroke**

Eight studies examined associations between the severity of PTSD symptoms and potential risk factors. In addition, one study also compared stroke survivors with and without PTSD (Sembi et al., 1998), reporting that those with PTSD had higher levels of neuroticism, anxiety and depression and lower levels of psychological well-being. However, the PTSD group was very small ( $n = 6$ ). A range of significant correlates of PTSD symptom severity has been reported, including demographic variables such as age (Sampson et al., 2003; Sharkey, 2007) and gender (Bruggimann et al., 2006), stroke details including the number of previous strokes (Merriman et al., 2007), time since stroke (Merriman et al., 2007) and post-stroke disability (Wang et al., 2011), personality variables such as neuroticism (Sembi et al., 1998), negative affect (Merriman et al., 2007), emotionalism (Eccles et al., 1999) and alexithymia

(Wang et al., 2011), psychological distress including psychiatric morbidity (Wang et al., 2011) as well as anxiety and depression (Bruggimann et al., 2006; Field et al., 2008; Merriman et al., 2007; Sembi et al., 1998), and cognitive appraisals about the stroke (Bruggimann et al., 2006; Field et al., 2008; Merriman et al., 2007; Sharkey, 2007). A number of additional risk factors have been found to have non-significant associations with PTSD symptom severity, including neurological impairment (Bruggimann et al., 2006), lesion site/hemisphere (Bruggimann et al., 2006, Merriman et al., 2007), memory of stroke (Bruggimann et al., 2006), dissociation (Merriman et al., 2007), and consciousness (Field et al., 2008). The strongest and most consistent correlates of PTSD symptom severity have been anxiety, depression and negative cognitive appraisals about the stroke. Considering each correlate in turn, the significant correlations between generalized anxiety and PTSD symptom severity are not unexpected given that PTSD is an anxiety disorder and that several symptoms overlap in the diagnostic criteria for the two disorders. The significant correlations with depression suggest that there might be high levels of psychological co-morbidity following stroke, with many stroke survivors experiencing both mood and anxiety disorders. For example, co-morbidity between anxiety and depression after stroke has been reported to be in the range of 11-18% (Astrom, 1996; Barker-Collo, 2007; Leppavuori et al., 2003; Sagen et al., 2009). In relation to co-morbidity with PTSD, Wang et al. (2011) reported that 93% of their sample of stroke survivors with PTSD scored above the GHQ-28 cut-off for psychiatric morbidity at one month post-stroke, although this figure fell to 50% at three months post-stroke. More generally, large community surveys have revealed that 80-85% of people diagnosed with PTSD also meet the diagnostic criteria for at least one other psychiatric condition (Brady, Killeen, Brewerton, & Lucerini, 2000; Creamer, Burgess, & McFarlane, 2001). While these figures highlight the breadth of psychopathology that may develop following trauma exposure, they may also reflect the lack of specificity of the current PTSD diagnostic criteria (Spitzer, First, & Wakefield, 2007). The significant correlations between negative cognitive appraisals about the stroke and PTSD symptom severity are consistent with psychological models of PTSD (e.g., Ehlers & Clark, 2000; Foa & Rothbaum, 1998) that emphasize that the way in which the trauma is interpreted and processed is important in the development and persistence of PTSD. However, closer inspection of the items used in some of the studies reveals that they may be confounded with Criterion A2 of the DSM-IV (APA, 1994) which states that an event must evoke feelings of intense fear, helplessness or horror to qualify as a traumatic event. Thus, items assessing feelings of hopelessness and helplessness (Bruggimann et al., 2006), fear (Merrimann et al., 2007) and horror (Sharkey, 2007) have been related to the severity of PTSD symptoms after stroke. In contrast, Field et al. (2008) focused on negative cognitions about the self and the world, that do not exhibit this overlap with Criterion A2.

### 3.3 Methodological issues

It is difficult to draw strong conclusions regarding the prevalence and correlates of PTSD after stroke because the majority of studies suffer from a number of important methodological limitations. These include a reliance on self-report measures of PTSD, small sample sizes, a preponderance of cross-sectional designs, a lack of representative samples, the assessment of a limited set of potential risk factors and a failure to fully consider the impact of specific features of stroke, medical events and older adults on PTSD symptomatology.

Authors (Date), Country	Study Design, Recruitment and Main Exclusion Criteria	Sample	Assessment of PTSD	Prevalence of PTSD	Significant Correlates of PTSD Symptom Severity
Sembi et al. (1998) UK	Cross-sectional Recruitment: outpatient stroke prevention clinic, outpatient elderly day care hospital, and inpatient stroke rehabilitation ward N patients screened and excluded not reported Response rate after exclusions = 77% Exclusions: dysphasia	First-ever stroke or transient ischemic attack N = 61 Age M = 66 years % male not reported Stroke details not reported	IES, cut-off score = 30 Penn, cut-off score = 35 CAPS Time since stroke not reported	21% (IES) 7% (Penn) 10% (CAPS) - only those who scored above cut-offs on the IES or Penn were interviewed using the CAPS	IES: physical disability, neuroticism, GHQ-28, anxiety, depression. Penn: neuroticism, GHQ-28, anxiety, depression.
Eccles et al. (1999) UK	Cross-sectional Recruitment: inpatient hospital wards N patients screened = 177 N patients excluded = 112 (63%) Response rate after exclusions not reported Exclusions: poor physical health, cognitive impairment, communication problems	Stroke N = 65 Age M = 72 years % male = 31% Stroke details not reported	IES Administered within 1 month of stroke	Not reported	Emotionalism.
Sampson et al. (2003) UK	Cross-sectional Recruitment: inpatient stroke units, older adult medical wards, standard medical wards N patients screened = 150 N patients excluded = 73 (49%) Response rate after exclusions = 76% Exclusions: cognitive impairment, dysphasia, physically unwell, hearing impairment	Stroke N = 54 Age Median = 72.5 years % male not reported Stroke details: right hemisphere stroke n = 30, left hemisphere stroke n = 22	PCL-S, cut-off score = 44 Median time in hospital = 43.5 days	6%	Age (negative r).

Table 1. Summary of Studies Examining the Prevalence and Correlates of PTSD after Stroke

Authors (Date), Country	Study Design, Recruitment and Main Exclusion Criteria	Sample	Assessment of PTSD	Prevalence of PTSD	Significant Correlates of PTSD Symptom Severity
Bruggimann et al. (2006) Switzerland	Cross-sectional Recruitment: postal questionnaire N patients screened = 142 N patients excluded = 37 (26%) Response rate after exclusions = 52% Exclusions: persistent moderate or severe neurologic deficit, major psychiatric illness prior to stroke, neurologic comorbidity	First-ever non-severe stroke N = 49 Age M = 51 years % male = 67% Stroke details: frontal n = 11, temporal n = 6, parietal n = 13, occipital n = 6, basal ganglia n = 13, cerebellum n = 16	IES, cut-off score = 30 1 year post-stroke	31%	Gender (female), education (negative <i>r</i> ), subjective trauma appraisals, depression, anxiety.
Merriman et al. (2007) UK	Cross-sectional Recruitment: inpatient stroke wards N patients screened = 108 N patients excluded = 50 (46%) Response rate after exclusions = 95% Recruitment: postal questionnaire to discharged patients N patients screened and excluded not reported Response rate after exclusions = 52% Exclusions: dysphasia, acute medical problems	Stroke N = 102 Age M = 74 years % male = 56% Stroke details not reported	PDS Time since stroke M = 123.01 days	31% (fulfilling Criteria B, C and D)	Time since stroke (negative <i>r</i> ), number of previous strokes, anxiety, depression, negative affect (trait measure), trauma appraisals.
Sharkey (2007) UK	Cross-sectional Recruitment: not reported in sufficient detail Exclusions: severe communication difficulties and cognitive impairments	First-ever stroke N = 34 Age M = 73 years % male = 59% Stroke location not reported	IES Penn CAPS Time since stroke M = 62 weeks	3% received a PTSD diagnosis using the CAPS and cut-off scores on the IES and Penn	IES: feeling horrified immediately after stroke, stroke-specific quality of life (negative <i>r</i> ). Penn: age (negative <i>r</i> ), fear of another stroke, stroke-specific quality of life (negative <i>r</i> ).

Table 1. (Continued)

Authors (Date), Country	Study Design, Recruitment and Main Exclusion Criteria	Sample	Assessment of PTSD	Prevalence of PTSD	Significant Correlates of PTSD Symptom Severity
Field et al. (2008) UK	Prospective Recruitment: inpatient stroke wards, followed-up by postal questionnaire N patients screened not reported Response rate after exclusions = 90% Response rate to time 2 questionnaire = 86% Exclusions: cognitive impairment (e.g., aphasia), acute medical problems	Stroke N = 81 at time 1 N = 70 at time 2 Age M = 71 years % male = 53% Stroke details not reported	PDS Time since stroke at time 1 M = 19.94 days Time 2 follow-up 3 months later	Not reported	Cross-sectional correlations at time 1: age (negative <i>r</i> ), anxiety, depression, negative appraisals about the self, negative appraisals about the world. Prospective correlations (time 1 to time 2): anxiety, depression, negative appraisals about the self, negative appraisals about the world, PDS.
Sagen et al. (2009, 2010) Norway	Cross-sectional Recruitment: inpatient stroke unit N patients screened not reported N patients excluded ≥ 81 Response rate after exclusions at recruitment = 84% Response rate after exclusions at 4 months = 69% Exclusions: aphasia, cognitive impairment, transient ischemic attack	Stroke N = 104 Age M = 65 years % male = 59% Stroke details: cerebral infarction <i>n</i> = 99, cerebral haemorrhage <i>n</i> = 5	SCID 4 months post-stroke	3%	Not assessed

Table 1. (Continued)



Authors (Date), Country	Study Design, Recruitment and Main Exclusion Criteria	Sample	Assessment of PTSD	Prevalence of PTSD	Significant Correlates of PTSD Symptom Severity
Wang et al. (2011) UK	Longitudinal Recruitment: stroke rehabilitation unit N patients screened = 191 N patients excluded = 91 (48%) Response rate after exclusions at recruitment = 90% Response rate to time 2 questionnaire = 87% Exclusions: dysphasia, language problems, history of mental health problems	Stroke N = 90 at time 1 N = 78 at time 2 Age M = 75 years % male = 48% Stroke details: cerebral infarction n = 72, cerebral haemorrhage n = 11	PDS Time since stroke at time 1 M = 47 days Time 2 follow-up 2 months later	30.0% at time 1 23.1% at time 2	Cross-sectional correlations at time 1: GHQ, physical disability, alexithymia (identifying and describing feelings). Prospective correlations (time 1 to time 2): GHQ, time since stroke, physical disability, alexithymia (identifying and describing feelings), PDS.

Table 1. (Continued)

### 3.3.1 Assessment of PTSD

Most studies have used self-report measures to assess the prevalence of PTSD, including the Impact of Events Scale (IES; Horowitz et al., 1979), the Penn Inventory of PTSD (Penn; Hammarberg, 1992), the Post Traumatic Stress Disorder Checklist (PCL-S; Weathers et al., 1993) and the Posttraumatic Diagnostic Scale (PDS; Foa et al., 1997). A major limitation of most of these measures is that they only assess the severity of PTSD symptoms (Criteria B, C and D) and fail to consider the length of time symptoms have been present (Criterion E), the impact of symptoms on daily functioning (Criterion F), or powerful emotional reactions (Criterion A2). Indeed, one of the most frequently used measures, the IES (Horowitz et al., 1979), only assesses two symptom clusters: intrusive thoughts and avoidance. Such measures are therefore likely to over-estimate the prevalence of PTSD as they do not assess all the DSM-IV criteria (A-F) for a PTSD diagnosis (APA, 1994).

Clinical diagnostic interviews were used in three studies to provide a diagnosis of PTSD. The Clinician-Administered PTSD Scale (CAPS; Blake et al., 1992) was used in two studies in conjunction with self-report measures. Thus, Sembi et al. (1998) first screened stroke survivors using the IES and Penn self-report scales; those scoring above cut-off points on both measures were then interviewed using the CAPS. Similarly, Sharkey (2007) used a "multi-modal" assessment of PTSD using the CAPS in conjunction with the IES and Penn. The Structured Clinical Interview for DSM-IV (SCID; First et al., 1995) was used in one study (Sagen et al., 2009, 2010) to provide a PTSD diagnosis. There was some evidence that studies employing diagnostic interviews to assess PTSD reported lower prevalence estimates than those using self-report PTSD measures. The prevalence rates reported in studies that included a clinical diagnostic interview ranged from 3% (Sagen et al., 2010) to 10% (Sembi et al., 1998), whereas the frequencies reported in studies only employing self-report measures ranged from 6% (Sampson et al., 2003) to 31% (Bruggiman et al., 2006).

### 3.3.2 Sample sizes

The samples sizes for studies included in the review were small, ranging from 34 (Sharkley, 2007) to 104 (Sagen et al., 2009, 2010). This has important consequences for research on the prevalence of PTSD after stroke. Small sample sizes are likely to lead to large confidence intervals and unreliable prevalence estimates. In addition, they increase the probability that outliers may have a disproportionate impact on prevalence rates (O'Donnell et al., 2003). For example, with a sample size of 50, each additional PTSD diagnosis increases the estimated prevalence rate by 2%. This issue is likely to be exacerbated when cut-off scores on self-report measures, rather than clinical interviews, are used to provide a PTSD diagnosis. Small sample sizes also impact on research on the correlates of PTSD after stroke. In particular, they are likely to lead to many analyses being under-powered, thereby increasing the probability of Type II errors (Cohen, 1992). In addition, outliers may have a disproportionate influence on the strength of correlations, leading to potentially spurious findings.

### 3.3.3 Study design

One of the most limiting aspects of research on the prevalence of PTSD after stroke is the lack of longitudinal studies. To date, all but one study (Wang et al., 2011) have employed cross-sectional designs. Moreover, the time since stroke at which PTSD was assessed varied considerably between studies. For example, the average time since stroke ranged from 43.5 days (Sampson et al., 2003) to 62 weeks (Sharkey, 2007). Moreover, there was also considerable variability within studies. For example, the standard deviation for time since

stroke reported by Sharkey (2007) was 26.3 weeks, and the time since stroke reported by Merriman et al. (2007) ranged from 27 to 365 days. Only three studies assessed PTSD at fixed time points post-stroke. Sagen et al. (2009, 2010) assessed PTSD at four months post-stroke, Bruggimann et al. (2006) assessed PTSD at one year post-stroke, and Wang et al. (2011) assessed PTSD at approximately one and three months post-stroke, although the mean time since stroke at time 1 was 47.1 days with a standard deviation of 26.0 days. It is therefore difficult to compare reported prevalence rates or to provide an accurate point prevalence of PTSD. As a result, there are no accurate data on the natural course of post-stroke PTSD over time. The preponderance of cross-sectional designs also limits conclusions regarding the direction of relationships between risk factors and PTSD after stroke. For example, the experience of PTSD may result in excessively negative appraisals about the stroke, rather than negative appraisals determining PTSD as proposed in cognitive models of PTSD (Brewin & Holmes, 2003). As result, prospective designs in which potential risk factors are assessed shortly after stroke and related to the development of PTSD at a later time point are essential. To date, only two studies have employed such a design (Field et al., 2008; Wang et al., 2011). Moreover, only three studies (Field et al., 2008; Merrimann et al., 2007; Wang et al., 2011) have employed multivariate analyses to examine associations between potential risk factors and PTSD symptom severity; all other studies only examined bivariate associations.

### 3.3.4 Sample representativeness

The number of stroke survivors excluded from participating in the studies was either not reported (Field et al., 2008; Sembi et al., 1998; Sharkey, 2007) or was considerable, ranging from 26% (Bruggimann et al., 2006) to 63% (Eccles et al., 1999). The main exclusion criteria included cognitive impairment, communication difficulties (e.g., aphasia), and poor physical health. Stroke survivors were usually recruited from inpatient stroke wards (Field et al., 2008; Merriman et al., 2007; Sagen et al., 2009, 2010; Sembi et al., 1998), stroke rehabilitation units (Wang et al., 2011) or general hospital wards (Eccles et al., 1999; Sampson et al., 2003). A couple studies also recruited participants using postal questionnaires (Bruggimann et al., 2006; Merriman et al., 2007). Response rates (after exclusions) among studies recruiting from inpatient wards were generally high, ranging from 76% (Sampson et al., 2003) to 95% (Merriman et al., 2007). Studies employing postal questionnaires obtained lower response rates (52%) (Bruggimann et al., 2006; Merriman et al., 2007).

High exclusion rates raise questions regarding the representativeness of the samples recruited as stroke survivors who experienced more severe strokes are likely to have been excluded. None of the studies reported whether the samples were representative of the populations from which they were drawn. As a result, generalisability is limited. Moreover, it is possible that these exclusion criteria may themselves be risk factors for the development of PTSD. If so, this would imply that most studies have underestimated the prevalence of PTSD after stroke.

### 3.3.5 Assessment of risk factors

Studies on the correlates of PTSD after stroke have focused on a limited set of variables and have not assessed variables that have been identified as having strong associations with PTSD symptomatology in response to other traumas (Brewin et al., 2000; Ozer et al., 2003). For example, Ozer et al. (2003) conducted a meta-analysis of seven potential predictors of PTSD diagnosis or symptoms and found that more distal characteristics related to the individual or their life history (e.g., prior trauma, family history of psychopathology) had

smaller correlations with PTSD symptomatology than more proximal psychological factors (e.g., perceived life threat, dissociation). Such findings are in line with current psychological models of PTSD that emphasize the importance of appraisal and memory processes in the development of PTSD (Brewin & Holmes, 2003). Few studies on PTSD after stroke have drawn on such models to guide the selection of independent variables. Ehlers and Clark's (2000) cognitive model, which according to Brewin and Holmes (2003) provides the most detailed account of PTSD, proposes that PTSD is likely to develop and persist when the trauma and/or its sequelae is processed in such a way that leads to a sense of serious current threat, as a result of (i) making excessively negative appraisals and (ii) disturbances in autobiographical memory.

Considering negative appraisals, only one study has tested the Ehlers and Clark (2000) model in relation to stroke. Field et al. (2008) reported that negative cognitions about the self (e.g., "I am inadequate") and about the world (e.g., "The world is a dangerous place"), assessed shortly after the stroke ( $M = 20$  days), were significantly correlated with the severity of PTSD symptoms both cross-sectionally and prospectively three months later. However, the prospective correlations became non-significant after controlling for the effect of initial PTSD symptoms. Considering disturbances in autobiographical memory, Ehlers and Clark (2000) propose that the overwhelming experience of a traumatic event may disrupt peritraumatic cognitive processing resulting in trauma memories that are disorganised and poorly elaborated. This, in turn, may make trauma memories more vulnerable to triggering by matching cues, thereby increasing the frequency of reexperiencing symptoms. Three aspects of cognitive processing during the trauma have been related to poorly elaborated/organised trauma memories and subsequent PTSD (Halligan et al., 2003); namely, (i) engaging in surface level, or data-driven processing (e.g., "It was just like a dream of unconnected impressions following each other"), (ii) a lack self-referential processing (e.g., "I felt as if it was happening to someone else"), and (iii) dissociation (e.g., reduced awareness of the self, time and/or environment at the time of the trauma). Halligan et al. (2003) reported that measures of these memory processes, assessed within three months after assault, were predictive of the severity of PTSD symptoms at three and six months follow-up. Only two studies have examined memory variables after stroke. Bruggiman et al. (2006) reported no differences in the symptom severity scores of survivors with fragmented versus complete memories of their stroke, whereas Merriman et al. (2007) reported that peritraumatic dissociation was related to the number, but not the severity, of PTSD symptoms after stroke.

A related strand of work has noted that people with PTSD have difficulty recalling specific autobiographical memories (e.g., "When I watched the football on the television last Sunday") in response to cue words (e.g., "happy"). Instead, they recall abstract or more general memories that cover several different events or time points (e.g., "When I watch football on the television"). This tendency has been termed overgeneral memory bias (Williams & Broadbent, 1986). An inability to retrieve specific autobiographical memories may prevent the trauma memory from being integrated with other autobiographical memories and with the person's schemas about the self and the world (Kleim & Ehlers, 2008), thereby contributing to the development of PTSD. A number of studies have reported that PTSD is associated with overgeneral memory bias (Bryant et al., 2008; Schönfeld & Ehlers, 2006; Schönfeld et al., 2007). Dagleish et al. (2008) have presented evidence to suggest that people with PTSD may avoid retrieving specific personal information as a means of affect regulation, in support of a functional avoidance account of overgeneral

memory bias (Williams et al., 2007). Thus, intentional memory searches may be stopped prematurely at an abstract level in order to avoid retrieving potentially distressing material related to the trauma. To date, no studies have examined the relationship between overgeneral memory bias and PTSD after stroke.

### 3.3.6 Stroke details

Few studies have reported details of the type (e.g., ischemic vs. haemorrhagic), hemisphere (left vs. right), site (e.g., frontal, temporal, etc.) or severity of the stroke. Sampson et al. (2003) reported details of the hemisphere of stroke, Sagen et al. (2009, 2010) reported whether the stroke was a cerebral infarction or a cerebral haemorrhage, whereas Bruggiman et al. (2006) and Wang et al. (2011) provided detailed information regarding the location of the stroke and neurological deficit. Moreover, these aspects of the stroke have rarely been related to PTSD symptom severity. One exception is Bruggiman et al. (2006) who reported that lesion site and neurological deficit were unrelated to PTSD symptomatology, although it should be noted that the sample included only non-severe strokes.

Psychobiological models of PTSD (Charney et al., 1993) and neuroimaging evidence (Lanius et al., 2006) suggest that the development of PTSD is related to impaired functioning of the medial prefrontal cortex which limits regulation of the amygdala. Neural networks involving these areas have been implicated in fear processing. As a result, damage to such networks may be uniquely related to anxiety disorders (Rauch, 2003). Bryant et al. (2010) reported that traumatic injury survivors who also sustained a mild traumatic brain injury (which tends to be associated with damage to frontal regions of the brain) were more likely to develop anxiety disorders, including PTSD, but not depressive disorders. Other research on stroke has noted that left hemispheric, subcortical and large lesions are associated with verbal memory deficits (Godefroy et al., 2009; Schoten et al., 2009) which, more generally, have been related to PTSD (Brewin et al., 2007; Johnsen & Asbjørnsen, 2008). Such deficits may lead to poorer processing of trauma memories thereby contributing to the development of PTSD.

Many stroke survivors are asleep or unconscious during their stroke. Whether or not PTSD symptoms can develop under such circumstances has been the subject of much debate (e.g., Harvey et al., 2003; Klein et al., 2003). For example, it has been argued that individuals who are amnesic of the trauma event, by definition, cannot meet Criterion A2 of DSM-IV for PTSD (i.e., experience feelings of intense fear, helplessness or horror in response to the trauma event) (O'Donnell et al., 2003). Studies have produced conflicting results on this issue (e.g., Bryant et al., 2009; Caspi et al., 2005; Creamer, O'Donnell, & Pattison, 2005). For example, Bryant et al. (2009) found that longer periods of post-traumatic amnesia were related to less severe intrusive memories one week post-injury, suggesting a protective effect. Thus, individuals who experience post-traumatic amnesia may have fewer or incomplete mental representations of the trauma which are less likely to be triggered by matching cues. However, post-traumatic amnesia was unrelated to the severity of PTSD symptoms at three months, suggesting that it does not protect against the development of PTSD over longer time periods. In relation to stroke, Field et al. (2008) reported that PTSD symptom severity at three months was unrelated to whether the survivor was conscious or not at the time of their stroke. There are a number of ways in which PTSD could develop following post-traumatic amnesia or impaired consciousness. First, it is possible that impaired consciousness does not last throughout the traumatic event, and that PTSD

symptoms may develop in relation to those aspects of the trauma experience that individuals are able to encode (Creamer et al., 2005). Second, individuals may retrospectively reconstruct memories of the trauma experience, for example from witnesses' reports, which subsequently develop into intrusive memories or flashbacks (Bryant et al., 2009). Third, processing of the trauma experience may occur at an implicit level during periods of impaired consciousness (Bryant, 2001).

### 3.3.7 Chronic stressors

Stroke is a leading cause of severe disability. Survivors typically experience a range of ongoing problems (e.g., weakness or paralysis, cognitive impairment, communication difficulties, problems with balance and coordination). One important question is the extent to which PTSD symptom severity reflects the impact of these ongoing stressors, rather than reactions to the stroke event itself. For example, in relation to MI, Shemesh et al. (2001) found that patients who experienced ongoing physical symptoms (e.g., angina) reported more intrusion and avoidance PTSD symptoms than those who were asymptomatic. In addition, in relation to stroke, Wang et al. (2011) reported that the level of physical disability was related to the severity of PTSD symptoms at three months post-stroke. There are a number of ways in which chronic stressors may contribute to the severity of PTSD symptoms. First, chronic stressors may erode individuals' resources, or their ability, to deal with their psychological reactions to the acute stressor (Adams & Boscarino, 2006). Second, chronic stressors may evoke reminders of the stroke which may, more directly, act as triggering cues for the reexperiencing symptoms of PTSD. Third, the experience of ongoing disability may be perceived by the individual to signify permanent negative change. Fourth, some disabilities experienced after stroke, including cognitive and language impairments, may impede the person's ability to fully process and integrate trauma memories with other autobiographical material. For example, cognitive impairment has been related to difficulties in recalling specific autobiographical memories in the elderly (Phillips & Williams, 1997) and in stroke survivors (Sampson et al., 2003). Fure et al. (2006) reported that cognitive impairment was related to elevated levels of anxiety in stroke patients; however, to date, no studies have examined the relationship between cognitive impairment and PTSD. In addition, Thomas and Lincoln (2008) reported that stroke survivors with aphasia had higher levels of emotional distress at one and six months post-stroke, with more detailed analyses revealing that this was the result of expressive, but not receptive, communication impairments. It is possible that stroke survivors with aphasia may also experience more PTSD symptoms.

### 3.3.8 PTSD and older adults

The majority of stroke survivors are older adults, with almost 80% of first-ever strokes occurring in people aged 65 years or older (Stroke Association, 2011). Knowledge regarding the prevalence and determinants of PTSD as well as its phenomenology in older adults, more generally, is limited (Averill & Beck, 2000; Cook & O'Donnell, 2005). Moreover, the majority of previous research on PTSD in older adults has focused on holocaust survivors, combat veterans and survivors of natural disasters, rather than on survivors of life-threatening illnesses such as stroke (Cook & O'Donnell, 2005). Some studies have highlighted differences between younger and older adults in the experience and/or reporting of PTSD symptoms (Acierno et al., 2002; Davidson et al., 1990; Fontana & Rosenheck, 1994), although other work has suggested that their PTSD reactions are quite

similar (Bleich et al., 2005; Chung et al., 2005; Kohn et al., 2005). In relation to stroke, a number of studies have reported negative correlations between age and the severity of PTSD symptoms (Field et al., 2008; Sampson et al., 2003; Sharkey, 2007), although other studies have reported non-significant correlations (Bruggimann et al., 2006; Merriman et al., 2007). Nonetheless, there are a number of specific factors that may need to be considered when assessing PTSD in older people (Cook & O'Donnell, 2005). First, older adults are likely to have experienced multiple lifetime traumas (Creamer & Parslow, 2008) which may compound the impact of the current trauma, and vice-versa (Bechtle-Higgins & Follette, 2002). Second, older adults are more likely to suffer from cognitive impairments and dementia which may impact on their ability to fully process trauma memories. Third, older adults may have a more accepting attitude to illness and its psychological consequences.

### 3.3.9 PTSD and medical events

The assessment of PTSD symptoms following medical events, such as stroke, is complicated by the possibility that such symptoms may be confounded with the effects of physical illness and/or its treatment. For example, some of the hyperarousal (e.g., disturbed sleep, irritability, difficulty concentrating) and avoidance (e.g., diminished interest, detachment) symptoms that are used in the diagnosis of PTSD are also common problems experienced by survivors as a consequence of their stroke. Similarly, psychogenic amnesia is also included in the diagnostic criteria for PTSD. However, stroke is often associated with periods of amnesia that may have an organic (i.e., physical), rather than psychogenic, origin. The difficulty in differentiating between organic versus psychogenic causes of specific symptoms has implications for the assessment of PTSD. If such symptoms are simply taken to be part of PTSD rather having an organic origin, this is likely to lead to inflated estimates of the prevalence of PTSD. This effect is likely to be amplified when self-report measures are used to assess PTSD as alternative explanations for such symptoms cannot be explored.

Further, Mundy and Baum (2004) have argued that the nature of PTSD intrusion symptoms for medical events might be qualitatively different to those for other traumatic events. Whereas the focus of intrusions for more traditional traumas, such as combat injuries and assaults, is on the past events, intrusions for medical events may also be future-oriented focusing on concerns about treatment, disease recurrence and ongoing functional impairment. Thus, in addition to having flashbacks to the traumatic event (in the past), individuals who have survived a life-threatening illness such as stroke may also have intrusive negative thoughts about the future (e.g., "Will I live to see my grandchildren grow up?", "Will I be able to work again?"). To date, there has been no phenomenological studies on the experience of PTSD after stroke. If the intrusions experienced by stroke survivors are found to be predominantly future-oriented, this would raise serious questions as to whether such clinical presentations are best thought of as PTSD – which, by definition, is *post*-trauma and characterized by being haunted by past horror – rather than anxiety about the (future) consequences of the stroke.

### 3.4 Recommendations for future research

On the basis of the review of studies on the prevalence and correlates of PTSD after stroke, five main recommendations for future research are made focusing on (i) the measurement of PTSD, (ii) study design, (iii) sample sizes, (iv) sample representativeness, and (v) the assessment of risk factors.

### 3.4.1 Measurement of PTSD

In order to provide accurate prevalence rates of PTSD after stroke, it is essential that structured clinical interviews are routinely employed. The Clinician-Administered PTSD Scale (Weathers et al., 2001) is widely regarded as the measure of choice for PTSD assessment as it is standardized, and can be used to provide both a PTSD diagnosis and a continuous measure of symptom severity. The PTSD module of the SCID (First et al., 1995) is also recommended for PTSD diagnosis, although it fails to provide a measure of PTSD symptom severity. When administering a structured clinical interview the researcher should consider whether the symptoms of PTSD, such as disturbed sleep, difficulty concentrating and amnesia, are better accounted for by alternative explanations (e.g., physical effect of the stroke, medication use, hospital environment) before they are categorized as having a psychogenic origin (O'Donnell et al., 2003). Moreover, this type of more in-depth questioning will also help the researcher to distinguish PTSD from generalized anxiety disorder and/or major depression.

### 3.4.2 Study design

Future research on the prevalence of post-stroke PTSD should employ longitudinal designs with multiple assessments of PTSD at fixed time points after stroke in order to provide accurate point prevalence rates and to chart the natural course of PTSD after stroke. Similarly, research on the correlates of PTSD after stroke should utilize prospective designs in which potential risk factors are assessed shortly after stroke (e.g., within one month) and related to PTSD caseness and symptom severity at subsequent time points while controlling for the effects of initial PTSD symptoms.

### 3.4.3 Sample sizes

Large samples are essential to establish the prevalence of psychiatric disorders in new populations (O'Donnell et al., 2003). Future research on the prevalence of PTSD after stroke should therefore aim to recruit larger sample sizes than have been recruited to date. The actual sample size required to accurately estimate different prevalence rates in a population can be calculated (Daniel, 1999). Naing et al. (2006) recommend that the precision of the estimate should be  $\pm 5\%$  when the expected prevalence rate is greater than 10%, and half the expected prevalence rate when less than 10%. Current prevalence estimates for PTSD after stroke range from 3% to 31%. The sample size required to estimate a 3% prevalence rate with 95% confidence intervals at 1.5% precision (i.e., between 1.5% to 4.5%) is 497, whereas the sample size required to estimate a 31% prevalence rate with 95% confidence intervals at 5% precision (i.e., between 26% and 36%) is 329. The adoption of multi-site studies are likely to aid the recruitment of such sample sizes and also address possible population differences across sites. In addition, studies on the predictors of PTSD after stroke need to be sufficiently powered to assess the impact of a full range of independent variables. Tabachnick and Fidell (2007) recommend that, in order to have adequate statistical power, the sample size for a regression analysis should be at least  $50 + 8k$  (where  $k$  = number of independent variables). For example, for a regression analysis with 25 independent variables the sample size should be at least 250.

### 3.4.4 Sample representativeness

Future studies should provide more information on the representativeness of their samples. Ideally, where there are common care pathways for stroke victims as in the UK, consecutive



admissions to stroke units/wards should be recruited. The resultant sample should then be compared with the patient population from which it was drawn in order to assess its representativeness. Given the severity of stroke and the ensuing levels of disability, it is likely that many stroke survivors will be unable to give informed consent and/or complete self-report measures or clinical diagnostic interviews. This is likely to affect the representativeness of the sample and restrict the extent to which the findings can be generalized to all stroke survivors. Studies should therefore provide detailed information on their recruitment procedures and exclusion criteria. In addition, future work should attempt to amend recruitment and assessment procedures in order, as far as possible, to recruit stroke survivors with communication and cognitive impairments into studies on post-stroke PTSD. For example, research on depression and aphasia (Thomas & Lincoln, 2008) has used visual analogue scales (Brumfitt & Sheeran, 1999) to assess emotional distress in stroke survivors with communication difficulties. Future work should therefore also focus on developing measures of PTSD symptom severity that can be completed by stroke survivors with communication difficulties and/or cognitive impairments.

#### **3.4.5 Assessment of risk factors**

Future research should assess a comprehensive range of potential risk factors, including stroke details, when assessing the predictors of post-stroke PTSD. In particular, future research should draw upon current models of PTSD (Brewin & Holmes, 2003) to assess the impact of more proximal psychological variables that have been found to be the strongest correlates of PTSD symptomatology across a range of traumas in meta-analytic reviews (e.g., Brewin et al., 2000; Ozer et al., 2003). Future research should routinely employ prospective designs and conduct multivariate analyses in which proximal psychological variables (e.g., appraisals, memory processes) are assessed shortly after stroke (e.g., within one month) and are related to the subsequent development of PTSD and/or symptom severity at later time points, while controlling for the influence of more distal factors (e.g., stroke details, demographics) and initial PTSD symptoms. In this way, future studies may assess the extent to which the effects of distal variables are mediated by these more proximal variables, thereby increasing our understanding of the mechanisms, or processes, underlying the development of PTSD after stroke.

#### **3.5 Ethical considerations**

When studying psychological reactions to life-threatening illnesses, such as stroke, that are associated with severe levels of disability and instability in the patient's medical condition, researchers need to be cognisant of ethical as well as scientific considerations (Tedstone & Tarrier, 2003). Particular attention needs to be paid to issues of informed consent given the cognitive and communication impairments experienced by many stroke survivors. PTSD has become a popular diagnosis over recent years (Summerfield, 2001), as evidenced by the increasing range of events, including life-threatening illnesses, that have been the focus of PTSD research (Tedstone & Tarrier, 2003). While such research has increased our understanding of psychological reactions to life-threatening illnesses, researchers should be aware of the risk of pathologising normal reactions to a traumatic event that may naturally remit over time (Middleton & Shaw, 2000).

#### 4. Conclusions

Post-stroke traumatic stress is an important but relatively neglected psychological consequence of stroke. It would be valuable to have reliable and accurate prevalence data from clinical diagnostic interviews with large, representative samples of stroke survivors collected over several time points. In addition, further work is required on the assessment of potential risk factors for the development of PTSD. This should include assessment of a full range of risk factors, including variables from current models of PTSD, shortly after stroke that can be related to subsequent PTSD caseness and symptom severity at later time points. A better understanding of the risk factors for PTSD after stroke has important clinical implications for the management of stroke survivors. It may assist in better differentiating the organic effects of stroke from the behavioural and psychological symptoms of the psychiatric disorder. More importantly, there is now a large body of evidence to guide the effective treatment of PTSD (Ponniah & Hollon, 2009). Appropriate use of such interventions has the potential to improve the quality of life, and reduce the care costs, of this population.

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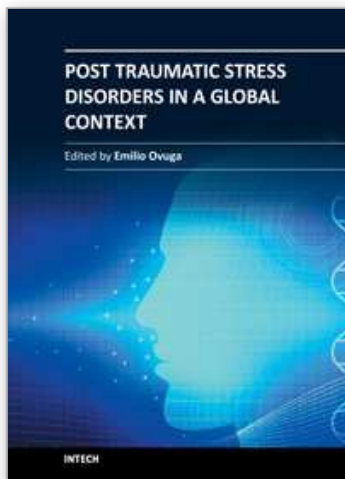
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## **Post Traumatic Stress Disorders in a Global Context**

Edited by Prof. Emilio Ovuga, Md, PhD

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If, as a health care or social service provider, one was called upon to help someone who has experienced terror in the hands of a hostage taker, an irate and chronically abusive spouse or parent, or a has survived a motor vehicle accident, landslide, earthquake, hurricane or even a massive flood, what would be one's priority response? What would be considered as the most pressing need of the individual requiring care? Whatever the answer to each of these questions, people who have experienced terror, suffer considerable psychological injury. Post-Traumatic Stress Disorder in a Global Context offers some answers to meet the needs of health care and social service providers in all settings, whether in a hospital emergency room, at the war front, or natural disaster site. The take home message is, after providing emergency care, there is always a pressing need to provide mental health care to all victims of traumatic stress.

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