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**Published paper**

Field, E.L., Norman, P. and Barton, J. (2008) *Cross-sectional and prospective associations between cognitive appraisals and posttraumatic stress disorder symptoms following stroke*, Behaviour Research And Therapy, Volume 46 (1), 62 -70.

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Running head: POST-STROKE PTSD SYMPTOMS

Cross-sectional and prospective associations between cognitive appraisals and posttraumatic stress disorder symptoms following stroke <sup>#</sup>

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<sup>#</sup> This paper is dedicated to the memory of the second author's father who died following a stroke on 16 August 2007.



**Abstract**

This study examined cross-sectional and prospective associations between cognitive appraisals and posttraumatic stress disorder (PTSD) symptoms following stroke. While in hospital, stroke patients ( $n = 81$ ) completed questionnaires assessing cognitive appraisals (i.e., negative cognitions about the self, negative cognitions about the world, and self-blame) and PTSD symptoms. PTSD symptoms were assessed again 3 months later when all patients had been discharged from hospital ( $n = 70$ ). Significant correlations were found between the time 1 measures of negative cognitions about the self and the world, but not self-blame, and the severity of PTSD symptoms measured at time 1 and at time 2. Regression analyses revealed that cognitive appraisals explained a significant amount of variance in the severity of PTSD symptoms at time 1, with negative cognitions about the self emerging as a significant predictor. In contrast, time 1 cognitive appraisals were unable to explain additional variance in time 2 PTSD severity over and above that explained by time 1 PTSD severity. The findings therefore provide only weak support for Ehlers and Clark's cognitive model of PTSD.

*Key words.* PTSD, anxiety, depression, cognitive appraisals, stroke.

## 1. Introduction

Posttraumatic stress disorder (PTSD) is described in the DSM-IV 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”. [1] The characteristic symptoms include “persistent reexperiencing of the traumatic event, persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness, and persistent symptoms of increased arousal” (APA, 1994, p. 424). Research on PTSD has traditionally focused on traumas such as war, physical and sexual assaults, and road traffic accidents. However, recent research has documented the existence of PTSD symptoms following a range of medical events (Tedstone & Tarrier, 2003) including cancer (e.g., Kangas, Henry & Bryant, 2003), myocardial infarction (MI) (e.g., Kutz, Shabtai, Solomon, Neumann & David, 1994), and subarachnoid haemorrhage (e.g., Berry, 1998). The present study extends this work by focusing on PTSD symptoms following stroke. A stroke occurs when the normal blood supply to the brain is disrupted causing cells in the affected area to become damaged or die. Stroke is the third most common cause of death, and the most common cause of severe disability, in the UK. A 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, 2006). Thus a stroke has many of the characteristics of events likely to trigger PTSD symptoms, in that it is unexpected, uncontrollable and potentially life threatening.

Previous research has estimated the prevalence of PTSD following stroke to be between 10-31%, depending on the method of assessment (Bruggimann et al., 2006; Merriman, Norman

& Barton, 2007; Sembi, Tarrier, O'Neill, Burns & Farragher, 1998). PTSD symptom severity has been found to be independent of age, marital status, memory deficits, neurological impairment, post-stroke disability, and dissociation (Bruggimann et al., 2006; Merriman et al., 2007; Sembi et al., 1998). Instead, a number of factors have been found to be associated with the severity of PTSD symptoms including gender, education, number of previous strokes, time since stroke (negative relationship), neuroticism, negative affect and retrospective perceptions of the stroke (e.g., fear, helplessness), although anxiety and depression have been found to be the most consistent correlates of the severity of post-stroke PTSD symptoms (Bruggimann et al., 2006; Merriman et al., 2007; Sembi et al., 1998). However, previous studies predicting post-stroke PTSD have suffered from a number of limitations. First, they have employed cross-sectional designs, thereby limiting conclusions that can be made regarding the direction of the relationships. Second, they have failed to draw upon recent cognitive models of PTSD which highlight the importance of cognitive appraisals in the development and persistence of PTSD (see Brewin & Holmes, 2003).

Foa and Rothbaum (1998) highlight two cognitions that may contribute to the development of PTSD; namely, that the world is extremely dangerous and that the self is totally incompetent. Similarly, Ehlers and Clark (2000) propose that persistent PTSD occurs when the trauma and/or its sequelae is processed in such a way that leads to a sense of serious current threat. This threat can be external (e.g., the world is a more dangerous place) or internal (e.g., the person no longer views himself or herself as capable/ competent). Ehlers and Clark (2000) outline two processes that can lead to this sense of serious current threat: (i) excessively negative appraisals of the trauma and/or its sequelae and (ii) disturbances in autobiographical memory. The present study focuses on the first of these processes. Previous research in non-medical

contexts has identified significant relationships between a range of negative appraisals (e.g., about oneself, one's world, one's future, interpretations of intrusive memories, perceptions of permanent change, the responses of others and anger) and the severity of PTSD symptoms following a variety of traumatic events (e.g., Dunmore, Clark & Ehlers, 2001; Ehlers, Mayou & Bryant, 2003; Fairbrother & Rachman, 2006; Mayou, Ehlers & Bryant, 2002).

A recent collaboration between the Foa and Ehlers research groups sought to consolidate this work through the development of the Posttraumatic Cognitions Inventory; a reliable and valid measure of trauma-related appraisals (Foa, Ehlers, Clark, Tolin & Orsillo, 1999). In particular, they identified three types of appraisals that may be associated with PTSD: (i) negative cognitions about the self (e.g., "I am inadequate"), (ii) negative cognitions about the world (e.g., "The world is a dangerous place"), and (iii) self-blame (e.g., "The event happened because of the way I acted") (Foa, Ehlers, Clark, Tolin & Orsillo, 1999). A number of studies have examined relationships between these appraisals and the severity of PTSD symptoms. Foa et al. (1999) reported strong, and significant, correlations between all three cognitive appraisals and the severity of PTSD symptoms in a sample of trauma survivors (including accidents, non-sexual assaults, sexual assaults, and illness). Considering studies in medical contexts, Angar, Kennedy and King (2006) found that negative cognitions about the self and the world, but not self-blame, were associated with the severity of PTSD symptoms among a sample of patients with spinal cord injuries. Similar results have been reported by Kangas et al. (2005) who found that negative cognitions about the self and the world assessed within one month of cancer diagnosis were associated with the severity of PTSD symptoms six months following diagnosis, whereas the correlation between self-blame and PTSD severity was non-significant. These studies provide initial evidence that the cognitive appraisals outlined by Foa et al. (1999) are

associated with PTSD symptom severity, although the use of cross-sectional and retrospective designs is a limitation of some of this research. Thus, further research is required to ascertain whether these cognitive appraisals are able to explain variance in the severity of PTSD symptoms over and above that explained by initial symptoms.

The present study therefore sought to assess cross-sectional and prospective associations between cognitive appraisals and PTSD symptoms following stroke. Patients who recently had a stroke completed questionnaires while in hospital and again three months later when they had been discharged from hospital. In line with Ehlers and Clark's (2000) cognitive model of PTSD, it was predicted that negative appraisals (i.e., negative cognitions about the self, negative cognitions about the world, and self-blame) would explain variance in the severity of PTSD symptoms, both cross-sectionally and prospectively, following stroke. A range of demographic (e.g., age, gender), medical (e.g., time since stroke, number of previous strokes) and psychological (e.g., anxiety, depression) variables were also assessed to control for the effects of these factors in the regression analyses.

## **2. Method**

### *2.1 Participants and Procedure*

Ethical approval was obtained from the local NHS Research Ethics Committee. Male and female adults ( $\geq 18$  years) who had recently experienced a stroke were recruited from the stroke wards of a NHS University Teaching Hospital in the UK. Patients who were unable to complete the questionnaire themselves due to cognitive impairment resulting from stroke (e.g., aphasia) and patients experiencing acute medical problems were excluded. Stroke nurse coordinators identified potential patients (i.e., those who met the inclusion criteria) and provided them with an information sheet. Patients who gave initial verbal consent were then approached by the lead



researcher who outlined the aims of the study. Upon gaining written informed consent, patients were asked to complete the time 1 questionnaire. Time 2 questionnaires were sent out to participants' home addresses 3 months later. Before these were sent out, patients' general practitioners were contacted to ascertain that they were still alive. Participants were telephoned to ensure they had received the questionnaire and to answer any questions. If questionnaires were not returned within 21 days, a second questionnaire was sent out.

Of the 90 eligible patients approached by the researcher, 9 declined to participate in the research. Eighty-one patients were therefore recruited into the study. It was possible to follow-up 70 (86%) of the initial 81 patients at 3 months, all of whom had been discharged from hospital. Of the 11 patients for whom it was not possible to collect time 2 data, 3 had died, 4 had been re-admitted to hospital and 2 had specified that they did not want to be contacted at follow-up. No significant differences were found between those patients who provided data at time 2 ( $n = 70$ ) and those who did not ( $n = 11$ ) in terms of demographic and clinical variables, and responses to the time 1 measures detailed below.

## 2.2 Measures

The time 1 questionnaire, which was completed in hospital, contained measures of cognitive appraisals, anxiety, depression and PTSD symptoms. The time 2 questionnaire at 3 month follow-up only assessed PTSD symptoms.

The *Posttraumatic Cognitions Inventory* (PTCI; Foa et al., 1999) is a 33 item measure that includes three scales assessing (i) negative cognitions about the self (e.g., "I feel like I don't know myself anymore") (21 items), (ii) negative cognitions about the world (e.g., "I have to be especially careful because you never know what can happen next") (7 items), and (iii) self-blame for the trauma (e.g., "Somebody else would not have gotten into this situation") (5 items).

Respondents were instructed to answer the items in relation to their stroke and to provide their responses on 7-point response scales (*totally disagree-totally agree*). The scales have been found to have excellent internal consistency with Foa et al. (1999) suggesting that they may be shortened for research purposes, such as predictive studies of PTSD. A shortened version (7 items) of the negative cognitions about the self scale was therefore used in the current study, based on the factor loadings reported by Foa et al. (1999). Responses to the items in each scale were averaged for data analysis. The scales were found to have satisfactory internal reliability in the current study ( $\alpha = .83, .83, .70$ , respectively).

The *Hospital Anxiety and Depression Scale* (HADS; Zigmond & Snaith, 1983) is a 14-item questionnaire comprising separate measures of *anxiety* (e.g., “I get sudden feelings of panic”) and *depression* (e.g., “I look forward with enjoyment to things”). Each sub-scale contains seven items scored on 4-point response scales (e.g., 0 = *not at all* to 3 = *very often indeed*). Responses to the items are coded and summed such that scores on each scale can range from 0 to 21 with high scores indicating high levels of anxiety and depression. Both the anxiety ( $\alpha = .86$ ) and depression ( $\alpha = .78$ ) subscales were found to have satisfactory internal reliability in the current study.

The *Posttraumatic Diagnostic Scale* (PDS; Foa, 1995; Foa, Cashman, Jaycox & Perry, 1997) includes 17 items, rated on 4-point response scales (i.e., 0 = *not at all or only once* to 3 = *5 or more times a week/almost always*), to measure the severity of the PTSD symptoms in the past month. The items assess the extent to which respondents have been bothered by re-experiencing (e.g., “Having bad dreams or nightmares about the event”), avoidance (e.g., “Trying not to think about, talk about, or have feelings about the event”) and arousal (e.g., “Feeling irritable or having fits of anger”) symptoms, as detailed in DSM-IV. In the present

study, respondents were instructed to answer the items in relation to their stroke. Responses to the 17 items are summed to provide a measure of PTSD symptom severity which can be rated by pre-defined categories: Mild = 1-10, Moderate = 11-20, Moderate to Severe = 21-35, and Severe  $\geq 36$  (Foa, 1995). The PDS has been reported to have excellent internal reliability, as was also found in the current study at time 1 ( $\alpha = .89$ ) and time 2 ( $\alpha = .91$ ). The PDS has been shown to have good sensitivity (0.89) and specificity (0.75) when compared against the Structured Clinical Interview for DSM-IV (Foa et al., 1997).

A range of demographic (i.e., age, gender, education level, marital status) and medical (i.e., time since stroke, number of previous strokes, consciousness at time of stroke) variables were also assessed.

### **3. Results**

#### *3.1 Data Screening*

Prior to analysis the data were examined for the assumptions of multivariate analysis (Tabachnick & Fidell, 1996). Scores on a number of the measures were positively skewed, including negative cognitions about the self ( $z = 3.18, p < .001$ ), self-blame ( $z = 3.92, p < .001$ ), time 1 PDS severity ( $z = 5.36, p < .001$ ) and time 2 PDS severity ( $z = 3.46, p < .001$ ). Square root transformations reduced levels of skewness to non-significance and were used in subsequent analyses. No evidence of multicollinearity was found among the independent variables used in the regression analyses.

#### *3.2 Sample Characteristics*

Participants ranged in age from 39 - 94 years ( $M = 71.23, SD = 11.74$ ), and included 43 males and 38 females. The majority categorized themselves as white British ( $n = 74$ ). Just over half were married ( $n = 44$ ) or cohabiting ( $n = 3$ ), while the remaining participants were either

widowed ( $n = 25$ ), divorced/separated ( $n = 4$ ) or single ( $n = 5$ ). The mean age of leaving full-time education was 14.97 years ( $SD = 1.49$ ). The mean number of strokes ranged from 1 to 4 ( $M = 1.30$ ,  $SD = 0.56$ ) with time since the most recent stroke ranging from 2 to 120 days ( $M = 19.94$ ,  $SD = 22.66$ ). Most participants ( $n = 55$ ) were conscious at the time of their stroke, although 7 were unconscious and 12 were asleep (missing data,  $n = 7$ ). The mean PDS severity score at time 1 ( $M = 9.32$ ,  $SD = 8.73$ ) was near the top of the mild symptom severity range, as described by Foa (1995), whereas at time 2 it was within the moderate symptom severity range ( $M = 11.93$ ,  $SD = 10.47$ ). However, there was no difference between the means of the time 1 and time 2 severity scores,  $t(69) = 1.54$ ,  $p = .13$ .

### 3.3 Correlation Analyses

Correlations were computed between the demographic, medical and psychological variables and PDS severity at time 1 and time 2 (see Table 1). Considering the demographic and medical variables, only age was found to correlate significantly with time 1 PDS severity. However, subsequent analyses revealed the presence of three (younger) outliers that, when removed, reduced the correlation between age and time 1 PDS severity to non-significance,  $r(76) = -.13$ ,  $p = .24$ . Considering the psychological variables, anxiety and depression were found to correlate significantly with both time 1 and time 2 PDS severity, as did negative cognitions about the self and negative cognitions about the world. In contrast, the correlations between self-blame and PDS severity were non-significant. Finally, time 1 PDS severity was found to correlate significantly with time 2 PDS severity.

### 3.4 Regression Analyses

The ability of the PTCI measures to explain variance in PDS severity scores at time 1 was assessed through a hierarchical regression analysis in which the independent variables were

entered in a two blocks (see Table 2). Given their significant correlations with time 1 PDS severity, anxiety and depression were entered in block 1, followed by the PTCI measures (i.e., negative cognitions about the self, negative cognitions about the world, self-blame) in block 2. The variables entered in the first block were able to explain 36% of the variance in time 1 PDS severity scores,  $R^2 = .36$ ,  $F(2,78) = 23.42$ ,  $p < .001$ , although only anxiety emerged as a significant predictor. The addition of the PTCI measures resulted in a significant increment in the amount of variance explained,  $R^2 = .09$ ,  $\Delta F(3,75) = 4.18$ ,  $p = .009$ , with negative cognitions about the self emerging as a significant predictor along with anxiety. The variables in the final regression equation were able to explain 47% of the variance in time 1 PDS severity scores,  $F(5,75) = 13.03$ ,  $p < .001$ .

A second hierarchical regression analysis was conducted to assess the ability of the baseline PTCI measures to explain variance in time 2 PDS severity scores, controlling for time 1 severity scores. The independent variables were entered in three blocks: (i) time 1 PDS severity, (ii) anxiety and depression and (iii) negative cognitions about the self, negative cognitions about the world, and self-blame (see Table 3). In this way it was possible to test the ability of the PTCI measures to predict time 2 PDS severity over and above the effect of baseline PDS severity scores as well as anxiety and depression which were found to correlate with time 2 PDS severity. Time 1 PDS severity explained 17% of the variance in time 2 PDS severity scores,  $R^2 = .17$ ,  $F(1,68) = 13.95$ ,  $p < .001$ . Neither the addition of anxiety and depression at step 2,  $\Delta R^2 = .03$ ,  $\Delta F(2,66) = 1.13$ ,  $p = .33$ , nor the addition of the PTCI measures at step 3,  $\Delta R^2 = .04$ ,  $\Delta F(3,63) = 1.07$ ,  $p = .37$ , produced a significant increment in the amount of variance explained. However, the previously significant effect of time 1 PDS severity became non-significant when these variables were added. The variables in the final regression equation were able to explain 24% of

the variance in time 2 PDS severity scores,  $F(6,63) = 3.25, p = .008$ , although none of the independent variables made a significant contribution to the regression equation. [2]

#### 4. Discussion

The current study assessed both cross-sectional and prospective associations between cognitive appraisals and PTSD symptoms following stroke. In line with Ehlers and Clark's (2000) cognitive model of PTSD, negative cognitions about the self and the world were found to correlate with the severity of PTSD symptoms both cross-sectionally, at time 1, and prospectively, at 3 month follow-up. In contrast, self-blame was unrelated to the severity of PTSD symptoms at both time points. The present results are similar to those reported in other medical contexts by Agar et al. (2006), in relation to spinal cord injuries, and by Kangas et al. (2005), in relation to cancer diagnosis. However, these results can be contrasted with those reported by Foa et al. (1999) who found significant correlations between all three cognitive appraisals and the severity of PTSD symptoms in a sample of trauma survivors (including accidents, non-sexual assaults, sexual assaults, and illness).

The non-significant correlations found for self-blame in the present study may reflect the nature of the trauma event, i.e., a stroke. First, compared to other traumatic events, such as sexual/physical assaults and road traffic accidents, the causes of a stroke are likely to be more distal. Thus, the main risk factors for stroke centre on various lifestyle factors including exercise, diet, smoking and alcohol consumption. In contrast, the causes of other trauma events, that have traditionally been the focus of PTSD research, may be more proximal in nature centring, for example, on what the person was doing just before the event. Second, stroke may have more similarities with natural disasters (i.e., "acts of God") than with interpersonal traumas (e.g., physical assaults) where specific individuals and/or actions can be readily identified as potential

causes of the event. Thus, given the nature of the trauma event, issues of self-blame may be less relevant to the psychological reactions of stroke patients than of other trauma survivors.

Regression analyses revealed that at time 1 (i.e., cross-sectionally) the cognitive appraisal measures explained a significant amount of the variance in the severity of PTSD symptoms, although only negative cognitions about the self made a significant contribution to the regression equation. Interestingly, this effect occurred despite controlling for the effects of anxiety and depression which have been found to consistently correlate with post-stroke PTSD symptom severity in previous studies (Bruggimann et al., 2006; Merriman et al., 2007; Sembi et al., 1998). In contrast, the baseline measures of cognitive appraisals were unable to explain additional variance in the severity of time 2 PTSD symptoms after controlling for baseline PTSD symptom severity. Similar results have been reported by Kangas et al. (2005) who found that negative cognitions about the self failed to emerge as a significant predictor of PTSD severity at six months following cancer diagnosis when entered into a regression analysis with a range of demographic, medical and psychological variables (i.e., age, gender, treatment complications, peritraumatic dissociation, anxiety, depression, social support), including Acute Stress Disorder, assessed at one month. Overall, the present findings provide only weak support for Ehlers and Clark's (2000) cognitive model of PTSD.

The current findings are of theoretical importance as they provide the first prospective investigation of the predictors of the severity of post-stroke PTSD symptoms as well as the first test of the Ehlers and Clark (2000) cognitive model of PTSD following stroke. However, it is clear that further, prospective, tests of the Ehlers and Clark (2000) model are required to assess the ability of cognitive appraisals to predict PTSD symptom severity, controlling for the effect of initial symptoms. In addition, future research attention should also focus on the mechanisms

through which negative cognitions may lead to the development and persistence of PTSD symptoms. For example, Ehlers and Clark (2000) propose that negative cognitions are likely to lead to various behavioural and cognitive coping strategies which, although intended to control the sense of current threat, may actually exacerbate PTSD symptoms. For example, a negative view of oneself (e.g., “My reactions since the event mean that I am going crazy”) and/or self-blame (e.g., “The event happened because of the way I acted”) may lead to excessive rumination which may provide internal retrieval cues that trigger re-experiencing symptoms (Ehlers & Clark, 2000). Similarly, negative cognitions about the world (e.g., “I have to be especially careful because you never know what can happen next”) may lead to avoidance and safety behaviors that serve to maintain a sense of serious current threat by preventing disconfirmation of negative beliefs (Dunmore, Clark & Ehlers, 1999). Interestingly, a number of studies have reported associations between such dysfunctional coping strategies and the severity of PTSD symptoms (e.g., Dunmore et al., 2001; Ehlers et al., 2003; Mayou et al., 2002).

The current study represents only a partial test of Ehlers and Clark’s (2000) model. In addition to the role of negative cognitive appraisals, the model proposes that disturbances in autobiographical memory are also important in the development and persistence of PTSD. For example, one factor that may lead to the trauma memory being poorly elaborated and inadequately integrated with other autobiographical memories is dissociation during and immediately after the trauma event, and a range of evidence has linked dissociation with PTSD symptom severity (e.g., Ehlers et al., 2003; Kangas et al., 2005). On a related point, only 55 of the sample were conscious at the time of their stroke, with 7 being unconscious and 12 asleep. As result, some patients may have had little or no recollection of their stroke. There has been considerable debate regarding whether or not PTSD symptoms can develop under such



circumstances (Harvey, Brewin, Jones & Kopelman, 2003; Klein, Caspi & Gil, 2003), with recent research producing conflicting findings (e.g., Caspi, Gil, Ben-Ari, Koren, Aaron-Peretz & Klein, 2005; Creamer, O'Donnell & Pattison, 2005). In the present study, consciousness at the time of stroke was found to be unrelated to PTSD symptom severity. One explanation for such a finding is that impaired consciousness may not last throughout the trauma experience 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). Alternatively, it is possible that processing of the trauma experience may occur at an implicit level during periods of impaired consciousness (Bryant, 2001).

There are a number of study limitations that should be noted. First, the sample size was relatively small which may have reduced the power of the regression analyses, especially at time 2. [3] It is therefore important for future work to replicate the current findings with larger sample sizes. Second, given the restricted ethnic range of the sample, the generalisability of the current findings may be limited. Third, it is possible that, in addition to the potentially traumatic nature of the stroke, patients' responses to the PDS may also be influenced by secondary appraisals regarding ongoing disability, although previous studies have shown levels of post-stroke disability to be unrelated to PTSD symptom severity (e.g., Merriman et al., 2007; Sembi et al., 1998). Fourth, the identification of PTSD symptoms following medical events, such as stroke, is further complicated by high co-morbidity with other emotional disorders such as anxiety and depression (Shalev, Schreiber, Galai & Melmed, 1993) which could elevate PDS scores and/or lead to the symptoms of PTSD being misinterpreted and the disorder being unrecognised or under-diagnosed in clinical practice (Zimmerman & Mattia, 1999).

Notwithstanding the study limitations, the current findings are of clinical importance. In particular, PTSD may interfere with rehabilitation programs and thereby impair adjustment (Williams, 1997). For example, PTSD has been linked with nonadherence to medication and adverse clinical outcomes in MI patients (Shemesh et al., 2001). Given that stroke patients are required to adhere to exercise/physiotherapy as well as medication as part of their rehabilitation programs, it is important to identify and treat at risk patients. Current guidelines in the UK recommend trauma-related cognitive behavioural therapy for persistent PTSD (NICE, 2005) as also recommended by Nemeroff et al. (2006) in their recent review of work on PTSD. On the basis of the present findings, negative cognitions about the self and, to a lesser extent, the world may provide appropriate targets for the treatment of post-stroke PTSD (cf., Foa & Rothbaum, 1998).

**Footnotes**

1. PTSD symptoms may also develop following witnessing (or learning about) an extreme traumatic event that affects another person (or family member/close associate).
2. Recent work (e.g., Marshall, Schell, Glynn & Shetty, 2006) has highlighted the importance of distinguishing between the individual PTSD symptom clusters (i.e., re-experiencing, avoidance and arousal). The correlation and regression analyses were therefore repeated considering each symptom cluster in turn. The pattern of results remained the same for each symptom cluster and therefore, for the sake of brevity, only the results pertaining to total PDS symptom severity scores are reported.
3. A power analysis was conducted to examine the ability of the PTCI measures to explain additional variance in time 2 PDS severity scores after controlling for the effect of 1 PDS severity, anxiety and depression. The PTCI measures were only able to explain a small additional amount of variance in time 2 PDS severity scores ( $\Delta R^2 = .04, f^2 = .05$ ) (Cohen, 1992). The power analysis also revealed that the time 2 sample size ( $n = 70$ ) was sufficient to detect a medium effect size ( $f^2 = .17$ , which equates to  $\Delta R^2 = .11$ ) with power set at .80 and alpha set at .05. These analyses suggest that the inability of the PTCI measures to explain additional variance in time 2 PDS severity scores is due to their small effect, rather than to an insufficient number of participants.

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Table 1

*Correlations Between the Independent Variables and (Time 1 and Time 2) PDS Severity*

Variable	Time 1 PDS severity	Time 2 PDS severity
Age	-.23*	-.23
Gender	.13	.18
Marital status	.18	.14
Education level	.19	.02
Number of previous strokes	.17	.04
Time since stroke	.09	.03
Consciousness at time of stroke	-.06	-.11
Anxiety	.60***	.34**
Depression	.45***	.37***
PTCI - Self subscale	.56***	.39***
PTCI - World subscale	.38***	.36***
PTCI - Self-blame subscale	.13	.02
Time 1 PDS severity	–	.41***

*Note.* Variables were coded as follows. Gender: 1 = male, 2 = female; marital status:

1 = single/divorced/widowed, 2 = married/cohabiting; consciousness at time of stroke;

1 = unconscious/asleep, 2 = conscious. Time 1  $N = 81$ . Time 2  $N = 70$ . \*  $p < .05$ . \*\*  $p < .01$ .

\*\*\*  $p < .001$



Table 2

*Summary of Regression Analysis for Variables Predicting Time 1 PDS Severity (N = 81)*

Variable	<i>B</i>	<i>SE B</i>	$\beta$
Step 1			
Anxiety	.18	.04	.51***
Depression	.06	.04	.16
Step 2			
Anxiety	.16	.04	.44***
Depression	-.005	.04	-.01
PTCI - Self subscale	1.44	.50	.38**
PTCI - World subscale	-.01	.12	-.01
PTCI - Self-blame subscale	-.06	.35	-.01

*Note.*  $R^2 = .37$  for Step 1,  $p < .001$  ;  $\Delta R^2 = .09$  for Step 2,  $p = .009$ .

\*\*  $p < .01$ . \*\*\*  $p < .001$ .

Table 3

*Summary of Hierarchical Regression Analysis for Variables Predicting Time 2 PDS Severity**(N = 70)*

Variable	<i>B</i>	<i>SE B</i>	$\beta$
Step 1			
Time 1 PDS Severity	.46	.12	.41***
Step 2			
Time 1 PDS Severity	.30	.17	.27
Anxiety	.03	.06	.07
Depression	.07	.06	.17
Step 3			
Time 1 PDS Severity	.23	.18	.21
Anxiety	.02	.06	.04
Depression	.05	.06	.12
PTCI - Self subscale	.40	.76	.10
PTCI - World subscale	.19	.17	.16
PTCI - Self-blame subscale	-.48	.53	-.11

*Note.*  $R^2 = .17$  for Step 1,  $p < .001$  ;  $\Delta R^2 = .03$  for Step 2,  $p = .33$ .

$\Delta R^2 = .04$  for Step 2,  $p = .37$ . \*\*\*  $p < .001$ .