MAJOR RESEARCH PROJECT

ANXIETY IN THE AFTERMATH OF ACQUIRED BRAIN INJURY:
PREVALENCE, COURSE AND CORRELATES

SECTION A
Factors associated with anxiety following stroke:
A review of the empirical evidence and a conceptual model
(Word Count: 5490 plus 714 words)

SECTION B
Anxiety related to discharge from inpatient neurorehabilitation:
Exploring the role of self-efficacy and internal health control beliefs
(Word Count: 7985 plus 64 words)

SECTION C
Critical Appraisal
(Word Count: 1987)

SECTION D
Appendix of Supporting Material

TOTAL WORD COUNT: 15 462 WORDS (plus 778 words)

A thesis submitted in partial fulfilment of the requirements of
Canterbury Christ Church University for the degree of
Doctor of Clinical Psychology

JULY 2013
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Acknowledgements

Firstly, I would like to say a big thank you to the participants who gave their time to take part in this study. Thank you to the three NHS Trusts that supported the project and to Dr Vicky Bancroft, Dr Amanda Nielsen and Beth Stroyde, who were essential to the recruitment of participants. Thank you to my supervisors, Dr Martin Harvey and Professor Paul Camic, for their advice and support in conducting the research and for their valuable ideas and comments on drafts of the work. Thanks to my fellow trainees and friends, who even while managing demands of their own commitments, were there to support me through mine. Last, but by no means least, a big thank you to my family, for their unconditional love and support and for all those positive vibes!
Summary of portfolio

Section A - Factors associated with anxiety following stroke:
A review of the empirical evidence and a conceptual model

A review of the scientific literature was carried-out to elucidate the prevalence, course and correlates of post-stroke anxiety (PSA). A number of potential risk factors for PSA are identified and a conceptual model incorporating some of these factors is presented. Methodological and contextual limitations of the existing evidence-base are discussed, with implications for clinical practice and future research highlighted.

Section B - Anxiety related to discharge from inpatient neurorehabilitation:
Exploring the role of self-efficacy and internal health control beliefs

A cross-sectional study explored the prevalence of anxiety related to discharge from inpatient neurorehabilitation among 42 participants with a diagnosis of acquired brain injury. Differential relationships between psychological factors (self-efficacy and internal health control beliefs) were examined; alongside the relative influence of demographic and clinical characteristics on discharge-related anxiety.

Findings revealed that age, self-efficacy and internal health control beliefs made independent contributions to self-reported discharge-anxiety, with perceived self-efficacy alone explaining 69% of the variance and mediating the effect of internal control beliefs. Implications for clinical practice and future research are discussed.

Section C - Critical Appraisal

An appraisal of the research process is given in answer to four questions. These explore lessons learnt and future training needs, as well as implications for clinical practice and directions for future empirical research in related areas.

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Factors associated with anxiety following stroke:
A review of the empirical evidence and a conceptual model

WORD COUNT: 5490 WORDS (plus 714 words)

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Abstract

Historically, the study of mood-related difficulties following stroke has largely focused on depression, with anxiety conditions having received comparatively little empirical attention. In view of this, the current review sought to compile and critically appraise the literature available on post-stroke anxiety (PSA). In particular, the review aimed to elucidate the prevalence, longitudinal course and correlates of PSA.

A search of the published literature yielded 28 articles that met criteria for inclusion in the review. Of these, 13 described empirical studies which assessed the prevalence of anxiety among stroke survivors' using diagnostic or screening tools.

Findings indicated that Generalised Anxiety Disorder (GAD) was the most commonly assessed diagnosis, with reported prevalence ranging from 17 - 28% in the acute phase post-stroke. Studies using self-report tools reported variable prevalence between 5 - 22%. Inconsistencies in how PSA was conceptualised and measured limited the interpretation and generalisability of these findings.

A number of potential risk factors for PSA were identified. Notably, studies provided limited empirical support for associations between PSA and demographic (age or gender), clinical (stroke pathology or degree of functional impairment) or environmental (e.g. social support) factors. There was some evidence to suggest that cognitive and psychological factors (e.g. confidence in recovery and control cognitions) may be implicated in the development of PSA and may be accessible to intervention. A conceptual model relevant to these ideas is presented. Implications for clinical practice and possible future research in the area are also highlighted.

Key terms: anxiety, emotion, mood, stroke, cerebral vascular accident, brain injury
Factors associated with anxiety following stroke: 
A review of the empirical evidence and a conceptual model

Over the past two decades research interest in psychiatric symptomatology following stroke has grown considerably, with evidence suggesting that mood-related difficulties are a common consequence of stroke (Royal College of Physicians; RCP, 2012). However, studies in the area have largely focused on post-stroke depression, with other emotional outcomes, such as anxiety, having received relatively little empirical attention (Bergersen, Frøslie, Sunnerhagen & Schanke, 2010).

The review that follows seeks to compile and appraise the scientific literature available on post-stroke anxiety (PSA). The aims of the review are (a) to explore the prevalence and longitudinal course of PSA; and (b) to examine aetiological features.

The review begins by outlining the pathophysiology of stroke. Subsequently, studies that have explored PSA are briefly outlined, following which the aims of the review are addressed in more detail. The methodological and contextual limitations of the existing evidence-base are then considered. In addition, a conceptual model, relevant to the development of PSA is presented. Finally, the review concludes by considering implications of its findings, for clinical practice and future research.

Stroke: Cerebral Vascular Accident

Stroke, or Cerebral Vascular Accident (CVA), is defined as “a clinical syndrome consisting of rapidly developing clinical signs of focal (at times global) disturbance of cerebral function, lasting more than 24 hours or leading to death, with no apparent cause other than of vascular origin” (Hatano, 1976, p. 542). In this definition Transient Ischemic Attacks (TIAs), which are denoted by stroke signs that resolve within 24 hours, are excluded (Duncan, Zorowitz & Bates, 2005).
Aetiology and prevalence

Stroke occurs when there is an interruption in the blood supply to parts of the brain; resulting in tissue damage (National Institute for Health and Clinical Excellence; NIHCE, 2008). This can be caused by ischemia (a lack of blood flow, due to an obstruction in a vessel) or haemorrhage (where a blood vessel ruptures and leaks into the surrounding tissue) (RCP, 2012). It is estimated that around 85% of strokes are ischemic in nature, while 15% are due to haemorrhage (RCP, 2012).

In the UK, around 150,000 people suffer a stroke each year (National Audit Office; NAO, 2010). Accordingly, stroke has come to be recognised as the third most common cause of death, after heart disease and cancer, and a leading cause of adult physical disability (Langhorne, Bernhardt & Kwakkel, 2011; NAO 2010).

Repercussions and care

The disabling effects of stroke vary widely and depend on the size and location of the brain areas that have been affected (Coffey et al., 2000). Common repercussions include decreased mobility; loss of functional independence; cognitive and language deficits; and changed relationships (Lai, Studenski, Duncan & Perera, 2002; Langhorne et al., 2011). While some of these effects are transient and likely to resolve over time others tend to be more permanent in nature (Lai et al., 2002). In most cases specialist rehabilitation is required, in order to support individuals’ to reach their optimal levels of physical, cognitive and social functioning (NAO, 2010).

In the UK, the trajectory of care for stroke spans several settings (NIHCE, 2008). In the acute stage, treatment is typically provided on specialist stroke units, where focus is on supporting basic physical functions; the prevention of further strokes; and the initiation of activities that promote rehabilitation (NIHCE, 2008).
Following discharge from acute units, most stroke survivors’ continue to require support and rehabilitation in the longer term (The Stroke Association, 2011). While many engage in community-based programs, some are seen to benefit from further inpatient rehabilitation, on post-acute units (Duncan, Zorowitz & Bates, 2005).

In view of the above, it is unsurprising that stroke survivors tend to perceive post-stroke adjustment to be a long and challenging process (Hackett, Yapa, Parag & Anderson, 2005). Furthermore, that patients’ have reported experiencing a range of negative emotional outcomes, in the aftermath of stroke (Langhorne et al., 2011).

Research has demonstrated that depression is a common consequence of stroke, with studies reporting prevalence between 20 and 50% (Hackett et al., 2005). In line with this, a substantial body of research literature identifies potential risk factors for post-stroke depression (PSD; see Hacket et al., 2005 for a review). In contrast, the literature on PSA remains in its infancy. However, researchers have begun to investigate similar demographic and clinical predictors, as those in PSD.

**Anxiety following stroke**

In some circumstances, anxiety can be considered to be functionally appropriate and a certain amount could be considered an understandable reaction to experiencing a life-threatening event such as stroke. In some cases, anxiety might even be seen to be advantageous. For example, this may promote adaptive health-related behaviours, encourage the mobilisation of social support and other resources, or prepare an individual for challenges which may lie ahead. However, substantially elevated levels of anxiety following stroke has been associated with multiple adverse outcomes, including reduced quality of life, increased healthcare utilisation and greater risk of chronic health conditions (Härtel, Schiling, Sperner & Thyen, 2004).
PSA can be conceptualised as either a diagnosable disorder or as substantially elevated symptomatology (assessed via rating scale and not meeting full diagnostic criteria). The Diagnostic and Statistical Manual of Mental Disorders (DSM; APA, 2000) classifies anxiety disorders as a group of syndromes; including: generalised anxiety disorder (GAD), panic disorder, agoraphobia, specific phobias, obsessive compulsive disorder (OCD) and post-traumatic stress disorder (PTSD). While each of these diagnoses comprises distinctive features, they share characteristic symptoms of excessive fear, distress and consequent difficulties in managing activities of daily living (APA, 2000). Physiological symptoms such as feelings of tension, palpitations and dizziness may also be present (APA, 2000).

One of the diagnostic challenges for assessing anxiety disorder diagnoses following stroke is that some of the physiological symptoms may be attributable to the stroke itself (Langhorne et al., 2011). As a result, the study of self-reported symptoms has proven to be relevant. Nonetheless, the extent to which both anxiety disorders and sub-threshold symptomatology may be a problem after stroke remains uncertain.

**Focus of the current review**

To facilitate a review of the evidence related to PSA a search of the literature was undertaken. Five online databases were searched for the years in which articles were available in electronic format. A web-based search was also conducted using the search engine ‘Google Scholar’. All searches covered studies published in the last 20 years (i.e. January 1992 - October 2012). Searches were conducted in June 2012 and updated in the last week of October 2012. The following injury-related terms were combined, using the Boolean operator ‘OR’: ‘stroke’, ‘cerebral vascular accident’, ‘CVA’, ‘acquired brain injury’, ‘ABI’. These terms were subsequently combined with the following mood-related terms, using the Boolean operator ‘AND’:
'mood', 'anxiety', 'anxious', 'distress*', 'emotion*'. All searches were restricted to the English language. (See Appendix 1 for further details of the search strategies). The review was also limited to research with adult populations. Studies exploring paediatric stroke and other forms of brain injury, such as traumatic brain injury, were excluded (for reviews on these topics see Härtel, Schiling, Sperner & Thyen, 2004; Hiott & Labbate, 2002; Moore, Terryberry-Spohr & Hope, 2006; Soo & Tate, 2007). It should also be noted that the review did not aim to elucidate studies pertaining to PTSD following stroke. This decision was taken in view of a recently published systematic review of studies in this area (Norman, O'Donnell, Creamer & Barton, 2012). The findings of this review will be referred to where appropriate.

**Studies exploring PSA**

The aforementioned literature search yielded 28 articles that met criteria for inclusion in the review. Of these, 13 described empirical studies. The methodological quality of each of these studies was evaluated according to a set of nine criteria developed in a previously published review (see Sherer et al., 2002). An evaluation sheet was completed according to a scoring system (see Table 1 in Appendix 1) wherein data for each criterion were recorded. As discussed in a later section of this review, based on this scoring system, studies rated as ‘acceptable’ or ‘commendable’ were given greater consideration in the evaluation of variables associated with PSA. Articles which do not describe empirical studies, but nonetheless make an important contribution to the scientific evidence-base will be referred to where appropriate.
Study design and setting

Of the 13 empirical studies identified for review, three used a cross-sectional design (Castillo, Starkstein, Fedoroff, Price & Robinson, 1993; Barker-Collo, 2007; Bergersen et al., 2010). Nine described longitudinal studies; two of which employed a prospective design (Åström, 1996; Burvill et al., 1995). The final study detailed the psychometric properties of measures used in related research (Sagen et al., 2010). (See Table 2 in Appendix 1, for an overview of the studies included in the review).

All of the studies involved individuals who had been diagnosed with stroke (cerebral infarction or haemorrhage); however, two also included those with TIA (Åström, 1996; Burvill et al., 1995). Eight studies used a sampling strategy based on consecutive admissions to acute stroke units. Of these, four described follow-up of participants subsequent to their discharge from the unit (Åström, 1996; Morrison, Johnstone, Pollard & MacWalter, 2005; Sagen et al., 2009; Schultz, Castillo, Kosier & Robinson, 1997). One study recruited patients on an acute stroke unit with no further follow-up (Castillo et al., 1993), while two involved stroke survivors who were living in the community (De Wit et al., 2008; Bergersen et al. 2010). The remaining study detailed recruitment from both inpatient and community settings (Burvill et al., 1995).

Methods used in assessing PSA

All of the studies used quantitative methods to examine relationships between variables. Each study declared PSA to be the main outcome of interest. However, the way in which anxiety was defined and assessed varied widely across studies.

Prior to the turn of century, studies focused on anxiety disorder diagnoses. Castillo et al. (1993; 1995) and Schulz et al. (1997) used the Present State Exam (PSE; Wing, Cooper & Sartorius, 1974) to assess for Generalised Anxiety Disorder.
FACTORS ASSOCIATED WITH ANXIETY FOLLOWING STROKE

(GAD); as outlined in the 3rd Edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; American Psychiatric Association; APA, 1980).

Burvill et al. (1995) used the Psychiatric Assessment Schedule (PAS; Dean, Surtees, & Sashidharan, 1983) to assess DSM-III-Revised criteria (DSM-III-R; APA, 1987).

More recent studies used standardised self-report measures to evaluate PSA. Five such studies used the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) to appraise anxiety symptomatology (Bergersen et al., 2010; De Wit et al., 2008; Morrison et al., 2000; Sagen et al., 2009; Townend et al., 2007). In these studies, Townend et al. (2007) and De Wit et al. (2008) interpreted a HADS-Anxiety (HADS-A) score of ≥ eight as indicative of PSA; while Bergersen et al. (2010) chose a cut-off score of seven. In contrast, Morrison et al. (2000) and Sagen et al. (2009) simply noted mean HADS-A scores for their sample, at various assessment intervals.

An exception, in terms of studies that used self-report measures, was a study by Barker-Collo (2007). These authors explored anxiety, at three months post-stroke, via the Beck Anxiety Inventory (BAI; Beck, Epstein, Brown & Steer, 1988).

Prevalence and course of PSA

Anxiety disorders are the most commonly diagnosed group of mental health difficulties in the general population, with a prevalence of around 7% (Martín-Merino, Ruigómez, Wallander, Johansson & García-Rodríguez, 2010). Research has shown that certain anxiety conditions may be even more prevalent in the aftermath of ABI. (See Tables 3 and 4 in Appendix 1, for prevalence of PSA in the reviewed studies).

Four of the reviewed studies assessed stroke survivors primarily for GAD. As can be seen in Table 3, in Appendix 1, prevalence rates in these studies ranged from 17 to 36% (Åström, 1996; Castillo et al., 1993, 1995; Schultz et al., 1997).
Two studies assessed for ‘any anxiety disorder’. Specifically, Sagen et al. (2009) interviewed 184 patients on their admission to an acute stroke unit and noted a prevalence rate of 23% for ‘any anxiety disorder’. Burvill et al. (1995) found a comparable prevalence of 24% (among 294 stroke survivors in the community). Interestingly, in the aforementioned review on PTSD after stroke, Norman et al. (2012) reported that the prevalence of PTSD after stroke ranged from 3 – 31%.

As can be seen from Table 4, in Appendix 1, studies that used self-report measures to gauge PSA also reported disparate prevalence rates. In a study using the BAI, Barker-Collo (2007) purported 21% of a sample of 73 hospitalised stroke patients to be suffering ‘moderate to severe’ symptoms. Studies using the HADS reported prevalence rates which correlated with the amount of time that had elapsed since participants’ stroke event; from 5% at two to five days post-stroke; to 14% at three months; and 22% at four months (Townend et al., 2007; De Wit et al., 2008).

The above findings raised question as to whether, in some cases, PSA might develop some time after stroke (De Wit et al., 2008). Moreover, whether distinction could be drawn between PSA diagnosed while a person was hospitalised for stroke (i.e. during admission to an acute stroke unit) (early-onset) and that which became apparent three months or more after a stroke event (late-onset) (Castillo et al., 1995).

Notwithstanding the above, few studies examined the longitudinal course of PSA. These studies reported mixed findings. Castillo et al. (1995) reported frequency of early-onset PSA (27%) to be higher than that of late-onset PSA (23%); whereas Åström (1996) noted a lower frequency of early-onset PSA (28%) than of late-onset (31%). More recently, De Wit et al. (2008) found that 11% of participants who were initially assessed as ‘not anxious’ met criteria for PSA at four months post-stroke.
These authors further noted that while the prevalence of PSA did not differ over time, the severity of symptomatology changed significantly (with this increasing from two to four months post-stroke and decreasing between four and six months). Notably, the period of two to four months post-stroke is consistent with time-frame in which most stroke survivors would be discharged from acute units (RCP, 2012). Notwithstanding this, the impact of imminent discharge on PSA symptomatology appeared to have been overlooked in the majority of the reviewed studies.

Factors associated with PSA

The review revealed several variables that were examined alongside PSA, including those related to: demographic characteristics; stroke neuropathology and psychosocial factors. The following sections aim to elucidate those factors which have been most consistently correlated with PSA across studies. A conceptual model that incorporates some of these factors will be presented later in the review.

Demographic factors

The most commonly examined demographic factors in the reviewed studies were age and gender. Castillo et al. (1993) and Schultz et al. (1997) found PSA to be more prevalent amongst younger, female stroke survivors. Morrison et al. (2000) similarly reported that women admitted to an acute stroke unit were more anxious than men; while Burvill et al. (1995) found no associations for PSA, age or gender.

Findings that PSA may be more prevalent among younger stroke survivors contradict research in other populations; wherein anxiety has been shown to increase with age (e.g. Härter, Conway & Merikangas, 2003). Regarding gender, the higher prevalence of PSA among female stroke survivors is in line with a higher prevalence of anxiety disorders among women in the general population (McIntosh et al., 2004).
Clinical characteristics

Several of the reviewed studies examined associations between PSA and clinical factors, including those related to psychiatric symptoms and stroke pathology.

Premorbid anxiety

Only two studies in the review, considered the implications of premorbid anxiety conditions for PSA. Burvill et al. (1995) noted that 21% of men, who met criteria for PSA, had a pre-stroke history suggestive of anxiety; whereas 30% of women who met criteria had a pre-stroke history. Schultz et al. (1997) noted a similar trend in their study data. Nevertheless, these both sets of researchers noted methodological limitations within their studies; wherein retrospective reports (from participants’ relatives) were used to elucidate information about premorbid anxiety. These methods may have under-estimated difficulties (Schultz et al., 1997).

Psychiatric comorbidity

Anxiety and depression have been found to have high rates of comorbidity in the general population and research has indicated that stroke pathology may amplify this phenomenon (Lindelow, Hardy & Rodgers, 1997). In keeping with this, Castillo et al. (1993) found that 27% of participants in their study met DSM criteria for both GAD and depression at a single assessment point; whereas Castillo et al. (1995) noted that 85% of participants met criteria for both disorders at some point during their two year study period. Studies that used self-report measures reported lower rates of comorbidity. Barker-Collo (2007) revealed that PSD was present in 12% of cases of ‘moderate to severe’ PSA (assessed via the BAI); while Sagen et al. (2010) noted that 14% of their participants exceeded HADS cut-off scores for both PSA and PSD.
**Stroke neuropathology**

Three studies examined the neuropathological correlates of PSA (Åström, 1996; Castillo et al., 1993; Morrison et al., 2000). These studies used computed tomography (CT) scans to examine lesions formed after stroke (Luft et al., 2004).

Two of the studies found that PSA in isolation was associated with right brain hemisphere lesions; whereas PSA comorbid with PSD was associated with lesions in the left hemisphere (Åström, 1996; Castillo et al., 1993). The third study revealed no effect for lesion location (Morrison et al., 2000). These studies were considered to be of high methodological quality. Scan results were evaluated by neurologists who were blind to psychiatric assessment findings and diagnostic interviews were carried-out by psychiatrists who had no knowledge of CT results (Åström, 1996).

**Functional impairment**

Physical functioning was investigated as a potential predictor of PSA in several of the reviewed studies (Castillo et al., 1995; Schultz et al., 1997; Åström, 1996; Morrison et al., 2000; Barker-Collo, 2007; Sagen et al., 2009). The majority of these studies used standardised, observer-rated measures to evaluate stroke survivors’ degree of functional impairment. These measures included the John Hopkins Functional Inventory (Wade 1987); the Functional Independence Measure (FIM; Granger, Hamilton & Sherwin, 1986); the Barthel Index (Wade & Collin, 1988); and the Scandinavian Stroke Scale (Boysen, 1992). Findings found no support for the hypothesis that greater impairment in functioning would be associated with PSA.
**Cognitive impairment**

The review revealed a single study that explored the relationship between PSA and cognitive functioning. In this study, Barker-Collo et al. (2007) used a range of standardised psychometric tests to assess individuals’ cognition at three months post-stroke. These tests included the Victoria Stroop Test (Spreen & Strauss, 1998) and California Verbal Learning Test (CVLT; Delis, Kramer, Kaplan & Ober, 2000).

Results reflected difficulties in the following cognitive domains: verbal memory; attention; and processing speed (wherein the majority of participants’ performances in each domain fell more than two standard deviations below the mean and were considered to be impaired) (Barker-Collo, 2007). Notably, when these cognitive variables were entered into a regression model they were seen to explain 39% of the variance in PSA. Notwithstanding this, the cross-sectional nature of the study meant that the causal direction of associations could not be inferred. Specifically, it was recognised that PSA may exacerbate cognitive deficits (Barker-Collo, 2007).

**Environmental factors**

Environmental factors have been widely recognised as being instrumental in promoting well-being (Baum, 2003). In the present review, various aspects related to stroke survivors’ social environments were explored as potential predictors of PSA.

**Satisfaction with healthcare**

The review revealed a single study which explored satisfaction with healthcare. In this study, Morrison et al. (2000) used a purposely designed measure wherein 71 stroke survivors were asked to rate how satisfied they were with (a) advice and (b) treatment they had received since their stroke. Findings revealed that both greater satisfaction with treatment and with advice were associated with lower PSA.
**Perceptions of social support**

Positive perceptions of social support have widely been shown to improve quality of life following stroke (Ch’Ng, French & Mclean, 2008). Nevertheless, the review revealed only two studies that explored social support as a potential predictor of PSA. In the first study, Castillo et al. (1995) completed the Social Functioning Exam (Starr, Robinson & Price, 1983) with patients on an acute stroke unit and found no association between social support and PSA. In the second study, Townend et al. (2007) completed the Multidimensional Scale of Perceived Social Support (Zimet, Dahlem & Zimet, 1988) with stroke survivors in the community and reported that greater perceptions of support were associated with less PSA. These researchers speculated that social support may be more salient following discharge from inpatient settings, as in this context individuals’ may be more reliant on their support networks.

**Psychological factors**

The potential role of psychological factors was explored in one of the reviewed studies. In this study, Morrison et al. (2000) hypothesised that stroke survivors’ level of confidence in their recovery would be associated with PSA. Further, that stroke survivors’ perception of personal control (as appraised via the Recovery Locus of Control scale; RLOC; Partridge & Johnston, 1989) would be associated with PSA. Findings demonstrated support for both hypotheses. Most notably, lower internality of control was positively correlated with PSA (Morrison et al., 2000). In line with this, research among other illness populations has consistently demonstrated that lower perceived internal control correlates with psychological distress (Kaptein et al., 2003).
Multiple predictors of PSA

Two of the reviewed studies explored multiple predictors of PSA. In the first study, Morrison et al. (2005) included four psychosocial factors (perceived control, recovery confidence, satisfaction with treatment and satisfaction with advice) in a hierarchical regression equation wherein 31% of the variance in PSA was explained. Findings indicated that confidence in recovery explained 25% of the variance in PSA; while treatment satisfaction contributed 7%. Perceived control and satisfaction with advice did not emerge as significant predictors (Morrison et al., 2005). In the second study, Barker-Collo (2007) employed multivariate analyses to determine whether demographic, clinical and cognitive variables would be predictive of PSA at three months post-stroke. Results revealed that age, gender and functional independence together explained 12% of the variance in PSA. This model was not significant. However, with cognitive variables (verbal memory, attention and processing speed) added to the equation 51% of the variance in PSA was explained. This finding was significant, with cognitive variables alone explaining 39% of the total variance in PSA.

The development of PSA

The findings of this review suggest that the aetiology of PSA has been explained in a number of ways. Historically, PSA has been conceptualised as either a pathophysiological mechanism; attributed to damage to the brain and consequent neurochemical responses (Åström, 1996; Castillo et al., 1993; Luft et al., 2004) or as a direct response to physical or cognitive impairment (Barker-Collo, 2007; Sagen et al., 2009). However, more recent research suggests that these understandings may be overly simplistic. In particular, findings suggest that the impact of stroke-related disability may be mediated psychological factors such as mental interpretations and consequent reactions to social or functional changes (Morrison et al., 2000).
A conceptual model

According to Lazarus and Folkman’s (1984) “transactional model of stress”, distress experienced in relation to illness is contingent upon cognitive appraisals of events. Within this model, primary appraisals are mainly concerned with evaluating potential threats of a stressor in the environment, while secondary appraisals involve “a complex evaluative process which takes into account the likelihood that a given coping option will accomplish what it is supposed to do and the likelihood that one can apply a particular strategy effectively” (Lazarus & Folkman, 1984, p. 35).

Lazarus and Folkman (1984) recognise constructs inherent in Bandura’s (1977a) social learning theory as congruent with the transactional model of stress. Specifically, the construct of self-efficacy is seen to influence secondary appraisals, since this relates to a person’s beliefs about whether or not they can successfully execute behaviours necessary to produce a desired outcome (Bandura, 1977b).

In a similar vein, beliefs regarding personal control are seen as influential (Lazarus & Folkman, 1984). According to Rotter (1966) internal locus of control (ILC) refers to beliefs that events are shaped by one’s own behaviour and external locus of control (ELC) refers to beliefs that outcomes are contingent on the actions of others.

Stroke survivors may experience changes in multiple domains of functioning (Lai et al., 2002). These changes may result in unsuccessful attempts at coping, which may influence appraisals (Taylor, Todman & Broomfield, 2011). Accordingly, stroke survivors’ are thought to be at increased risk of developing belief systems characterised by poor self-efficacy and ELC (Endler, Kocovski & Macrondimitris, 2001). Although research exploring these variables among stroke populations is limited, research among other illness populations has shown that poor self-efficacy and ELC beliefs are related to distress (Endler et al., 2001; Wu, Tang & Kwok, 2004).
Limitations of the research

The empirical findings outlined in this review should be considered within the context of methodological and contextual limitations of the included studies.

Summary and weight of findings

Based on a set of predefined quality criteria (see Table 1 in Appendix 1), five of the 13 studies included the review were rated as ‘commendable’, while six were rated as ‘acceptable’ and two as ‘marginal’. Notably, the review indicated that GAD was the most commonly assessed diagnosis in studies of PSA disorders, with reported prevalence ranging from 17 - 28% (Åström, 1996; Castillo et al., 1995). Studies using self-report tools reported prevalence between 5 - 22% (De Wit et al., 2008; Townend et al., 2007). All of these studies were considered to be of acceptable methodological quality. In contrast, a study reporting the self-reported prevalence of PSA to be 36.4% was rated as being of ‘marginal’ quality (Bergersen et al., 2010).

Two studies suggested that PSA may be more common among young women (Castillo et al., 1993); however, others found no effect for age (Burvill et al., 1995) or gender (Morrison et al., 2000). Findings also demonstrated mixed empirical support for hypotheses that PSA may be a direct consequence of stroke pathology. Two studies noted greater frequency of cortical lesions in the right brain hemisphere amongst individuals with PSA (Åström, 1996; Castillo et al., 1993); however, a third found no association (Morrison et al., 2000). All of these studies were considered to be of acceptable methodological quality and therefore no additional weight can be given to specific findings and no further conclusions can be drawn about the nature of the relationships between the variables under investigation and PSA as outcome.

Several studies found no association between PSA and physical impairment (Åström, 1996; Castillo et al., 1995; Morrison et al., 2000), whereas studies revealed
some support for relationships between PSA and environmental factors (e.g. those related to perceptions of social support) (Townend et al., 2007) and psychological factors such as personal confidence in recovery and perceptions of internal control (Morrison et al., 2000). A single study provided support for the predictive role of cognitive functioning / impairment in the development of PSA (Barker-Collo et al., 2007). However, this study was considered to be of ‘marginal’ methodological quality.

Methodological limitations

It has been recognised that accurate measurement of mood disorders among stroke survivors is confounded by symptoms of physical illness (De Wit et al., 2008). In keeping with this, research has demonstrated low sensitivity but high specificity for self-report tools such as the HADS among stroke survivors (Sagen et al., 2009). Research has also highlighted discrepancies between psychiatric symptoms reported by stroke survivors and those obtained from collateral sources; with stroke survivors’ under-reporting symptoms (Sagen et al., 2009). Accordingly, it is possible that the prevalence of PSA assessed via self-report scale may have been underestimated. This may have been compounded by sampling bias, as anxiety may have affected individuals’ decision as to whether to take part in the research. Notably, none of the studies reported on the proportion of eligible participants who chose to participate.

Studies that employed clinical interviews to assess for anxiety disorders may have had greater validity (Sagen et al., 2009). However, these studies were limited by small sample sizes, which made it necessary to collapse anxiety disorder diagnoses into broad diagnostic groups. It would be reasonable to speculate that larger samples may have afforded greater opportunity to assess for the prevalence of varied anxiety disorders such as PTSD or panic disorder. Notably, several studies in the review chose to assess exclusively for GAD, as this constitutes a well-defined
syndrome for research purposes (Åström, 1996). However, these studies modified diagnostic criteria to exclude the recommended symptom duration of six months (APA, 1994) (see Appendix 4 for a summary of DSM diagnostic criteria for GAD).

Although the review highlighted several factors that were assessed alongside PSA, findings were seldom included in sufficient studies to make an accurate interpretation regarding the level of empirical support for associations with PSA.

It is also important to note that findings were unclear as to whether anxiety reported was a consequence of stroke, as most studies failed to consider premorbid difficulties. Studies also neglected to include information about other factors that might impact on PSA, such as medication use or psychological intervention.

Finally, causal associations between study variables were precluded, as most studies carried-out cross-sectional assessments. Moreover, findings did not indicate whether prevalence of PSA was in keeping with the prevalence of anxiety conditions in other populations, as none of the reviewed studies included comparison groups.

**Contextual limitations**

The main contextual limitation of the review relates to generalisability of findings. Studies were carried-out in a variety of different countries, including: America, New Zealand, Australia, Norway, Sweden and Scotland (see Table 3 in Appendix 3 for further details regarding study location). Therefore, it was difficult to gauge the extent to which the reviews findings may be relevant to the UK context.

Variability in assessment intervals, alongside a lack of clarity about length of inpatient stay or discharge destination in longitudinal studies also made it difficult to distinguish between the prevalence of PSA in inpatient and community settings.
Several longitudinal studies noted that at a three-month assessment point, stroke survivors would have been nearing discharge to the community. However, no studies appeared to consider the potential impact of imminent discharge on PSA.

The transition from inpatient neurorehabilitation to home has been recognised as a distinct and important phase in the continuum of care following stroke and other forms of acquired brain injury (ABI; Nalder et al., 2012). In a recent review of the literature, Turner et al. (2008) noted that the hospital-to-home transition was often perceived as a stressful and challenging time by individuals with ABI. Moreover, that while this was associated with positive perceptions of improvement in an individual’s condition, this was also associated with negative perceptions of decreased health monitoring or care and feelings of anxiety linked to ‘relocation stress’ (Carpenito-Moyet, 2006). According to Carpenito-Moyet (2006) ‘relocation stress’ can be defined as: “a state in which a person experiences physiologic or psychological disturbances as a result of transfer from one environment to another” (p. 597).

**Implications for clinical practice**

In some situations, anxiety is seen as functionally appropriate and to a certain extent could be considered a normal reaction to experiencing a life-threatening event such as stroke. However, substantially elevated levels of PSA have been associated with adverse consequences including greater functional dependence (Åström, 1996); reduced well-being (Lai et al., 2002) and poorer quality of life (Härtel et al., 2004). In addition, PSA has been seen to adversely impact on interpersonal relationships, as well as engagement and progress in neurorehabilitation (Sturm et al., 2004).

Given the potential negative impact of PSA, it is important for clinicians to be able to accurately assess and identify those individuals who may be at greatest risk of developing the condition (Barker-Collo, 2007). It is encouraging to note that
National Clinical Guidelines for Stroke (NIHCE, 2012; RCP; 2010) recommend the routine assessment and management of mood-related difficulties following stroke. Moreover, that the National Stroke Strategy (DOH, 2007) highlights the importance of addressing psychological factors, which may impact on post-stroke adjustment.

With regard to treatment for PSA, findings of the current review are encouraging, as they provide limited support for relationships between stable factors (e.g. demographic and clinical characteristics) and PSA; while holding promise that potentially alterable factors (e.g. control cognitions) may be accessible to intervention (Morrison et al., 2005). While there is limited evidence for the value of medication in the treatment of PSA other forms of intervention such as guided self-help, relaxation training or cognitive behavioural therapy may be of benefit (Bergersen et al., 2010).

**Directions for future research**

This review highlights several possible directions for future research. Firstly, while self-report measures such as the HADS hold promise for the detection of PSA, further research is needed to validate these measures and determine cut-off scores for clinically significant anxiety in stroke populations (Sagen et al., 2009).

As the majority of research to date has been conducted outside of the UK, further research may be needed to establish accurate estimates of the prevalence and correlates of PSA at varied points across the stroke care pathway in this country.

Given that transitions from inpatient care to the community have been recognised as a stressful time for stroke survivors (Turner et al., 2008), it might be useful to explore whether imminent discharge from neurorehabilitation impacts on the reporting of anxiety symptomatology. Specifically, further research is needed to substantiate existing anecdotal evidence and quantify the prevalence of anxiety related to discharge. Such research might include mixed diagnostic samples as seen
in neurorehabilitation settings (i.e. stroke and TBI survivors). This may increase the clinical relevance of findings (Bernhardt, Dewey, Thrift, Collier & Donnan, 2008).

Another aim for future research would be to increase current understanding of potential risk factors for PSA. Notably, in the current review a large proportion of the variance in PSA was seen to be unexplained (Morrison et al., 2000; Barker-Collo et al., 2007). Accordingly, findings highlight a need for further exploration of factors that might be related to PSA (e.g. stroke severity or premorbid physical functioning). In particular, research may have a role in exploring factors that may render stroke survivors’ more or less resilient to anxiety. In view of research carried-out among other chronic illness populations, it would be reasonable to hypothesise that further investigation into the role of psychological factors (e.g. self-efficacy, self-concept, control cognitions, self-esteem, personality traits or coping styles) may be of benefit. For example, research might seek to investigate the potential impact of self-efficacy and ILC on PSA; while also considering demographic and clinical factors that have been associated with anxiety conditions (e.g. age, gender, brain lesion locations). Such research may hold promise in supporting clinical decisions regarding treatment.

**Conclusions**

The findings of the review suggested that anxiety conditions are prevalent post-stroke. However, research in the area continues to struggle with inconsistencies in how PSA is conceptualised and measured, thereby limiting the interpretation and generalisability of findings. Nevertheless, studies have made progress in identifying potential risk factors for PSA. Although causal directions of associations between variables cannot be inferred, findings nonetheless provide evidence to suggest that PSA is likely to be more than a reaction to neurological impairment or functional limitations. Specifically, evidence suggests that social, cognitive and psychological
factors might serve to influence stroke survivors’ reactions to post-stroke changes. Accordingly, findings hold promise that potentially alterable cognitions may be implicated in the development of PSA and may be accessible to intervention. Further research aimed at gaining knowledge of PSA and associated factors may hold implications for the early detection of those at increased risk of significant anxiety following stroke and may support clinical decisions regarding treatment.
References


Anxiety related to discharge from inpatient neurorehabilitation: Exploring the role of self-efficacy and internal health control beliefs

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Abstract

Objectives: This study aimed to determine the prevalence of anxiety specifically related to discharge in a group of 42 individuals who had sustained moderate to severe acquired brain injury and who were imminently due to return home following a period of inpatient neurorehabilitation. The study also aimed to explore differential relationships between psychological factors (self-efficacy and health control beliefs) alongside the relative influence of demographic (age, gender and ethnicity) and clinical (medical diagnosis and injury location) characteristics on discharge-anxiety.

Design: A cross-sectional, single-group design was employed, wherein correlational and multivariate analyses were used to explore relationships between variables. Data were obtained via self-report tools and retrospective reviews of medical files.

Results: While few participants (14%) reported markedly elevated trait-anxiety almost half (45%) of the sample reported levels of transient, state-anxiety which could be considered to be clinically significant. Notably, state-anxiety (appraised via the State-Trait Anxiety Inventory) was strongly associated with discharge-anxiety (appraised via the Patient Anxieties Questionnaire). Age, self-efficacy and internal health control beliefs made independent contributions to the level of discharge-anxiety reported, with perceived self-efficacy alone explaining 69% of the overall variance and mediating the effect of internal control beliefs. No other demographic or clinical characteristics examined were significantly related to discharge-anxiety.

Conclusions: Although causality cannot be inferred, findings suggest that anxiety related to discharge from inpatient rehabilitation is best predicted by poor perceptions of self-efficacy. Implications for clinical practice and future research are discussed.

Key terms: discharge, anxiety, brain injury, self-efficacy, locus of control
Anxiety related to discharge from inpatient neurorehabilitation:

Exploring the role of self-efficacy and internal health control beliefs

Over the past two decades there have been numerous studies reporting on the challenges faced by individuals with a diagnosis of acquired brain injury (ABI), following their discharge from inpatient care to the community. Findings suggest that individuals who have sustained ABI are likely to experience difficulties across multiple domains of physical, cognitive and interpersonal functioning (Fletcher, 2009; Lai, Studenski, Duncan & Perera, 2002) on returning to the community and home life. Notwithstanding this, there has been little research exploring the potential concerns or emotional status of inpatients prior to their discharge from inpatient rehabilitation.

Aetiology and prevalence of ABI

ABI is an inclusive category that describes rapid onset brain injury of varied aetiology; including: accidental or surgical trauma; vascular accident (i.e. stroke); cerebral anoxia; and complications arising from metabolic insult or infection.

The varied conditions that comprise ABI make it difficult to determine overall prevalence. However, it is recognised that stroke and traumatic brain injury (TBI) make up the largest proportion of ABI in the UK (National Audit Office; NAO, 2010).

Around 150,000 people suffer a stroke each year; while moderate to severe TBI affects around 25 per 100,000 people annually (NAO, 2010; National Institute for Health and Clinical Excellence; NIHCE, 2012). It is estimated that 10–20% of people who sustain an ABI will suffer permanent disability; whereas 65–85% will have had a good physical but not necessarily cognitive or social recovery (Turner-Stokes, 2003).
Neurorehabilitation following ABI

Recovery following ABI often involves intensive engagement with specialist neurorehabilitation services (Robertson, 2008). In the UK, rehabilitation starts in the acute stages of hospital care, where focus is on preventing health complications and reducing injury-related impairments (Turner-Stokes, 2003). Although not all people require specialist intervention beyond this acute stage, a proportion may experience significant physical or cognitive difficulties that would likely impact on their daily lives. In these instances, further rehabilitation on post-acute units may promote functional independence and support the person in returning home (Turner-Stokes, 2003).

Experiences of discharge

The transition from inpatient care to home has been recognised as a distinct and critical phase in the rehabilitation continuum following ABI (Ellis-Hill et al., 2009). Although this transition has been linked to positive perceptions of recovery (Turner, Fleming, Ownsworth & Cornwell, 2008) research has indicated that this is also often associated with negative perceptions of decreased health monitoring and support (Dowswell et al., 2000). These factors may compound feelings of anxiety linked to ‘relocation stress’. According to Carpenito-Moyet (2006) ‘relocation stress’ can be defined as: “a state in which a person experiences physiologic or psychological disturbances as a result of transfer from one environment to another” (p. 597).

In keeping with the above, evidence suggests that individuals perceive the transition from inpatient neurorehabilitation to home to be a stressful and testing time. In a review of the relevant literature Turner et al. (2008) highlighted seven qualitative studies that used interviews to explore the experiences of inpatients with ABI during their discharge home. A common theme identified within these studies was that the discharge process was perceived to be a distressing experience. Words used to
describe this process included ‘stressful’ and ‘overwhelming’ (Turner et al., 2008).

In view of these findings the reviewers noted that individuals with ABI may be at increased risk of anxiety at the time of discharge from inpatient care and that further research may be warranted to substantiate anecdotal evidence (Turner et al., 2008).

**Anxiety in the aftermath of ABI**

Notwithstanding the above, the vast majority of studies to date have explored anxiety among ABI populations shortly after admission to inpatient settings or some months after discharge to the community (Turner et al., 2008; Turner, Fleming, Cornwell, Haines & Ownsworth, 2009). Accordingly, studies have explored levels of general, (i.e. trait) anxiety and not anxiety expressly related to discharge *per se*.

In a meta-analysis of 12 studies, Epstein and Ursano (2005) noted the overall prevalence of anxiety disorders to be 29% across all severities of TBI. In this review prevalence of specific diagnoses included: generalised anxiety disorder (3-28%); post-traumatic stress disorder (3-27%) and panic disorder (4-17%). By contrast, studies exploring anxiety disorders after stroke have primarily assessed for GAD, with reported prevalence ranging from 17-28% (Åström, 1996; Castillo et al., 1993).

Recently researchers have noted that: (a) assessment of anxiety disorder diagnoses is limited to the extent that diagnoses themselves are valid constructs; and (b) measurement of anxiety symptomatology (not meeting full diagnostic criteria) may increase clinical relevance of findings (Moore, Terryberry-Spohr & Hope, 2006). Therefore, studies have increasingly begun to explore anxiety using self-report tools such as the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983).

These studies have revealed further information about the nature of anxiety following ABI. For example, in a longitudinal study exploring anxiety following stroke, De Wit et al. (2008) noted that while the prevalence of anxiety did not differ over time,
the severity of symptomatology increased significantly from two to four months post-stroke and decreased between four and six months. Notably, the period of two to four months post-stroke is consistent with the time-frame during which most stroke survivors would be approaching discharge from inpatient care (Duncan, Zorowitz & Bates, 2005). Nonetheless, to date there has been a paucity of research exploring factors related to clinical context, as potential mediators of anxiety following ABI.

**Factors associated with anxiety**

Historically, anxiety following ABI has been conceptualised as either a pathophysiological mechanism, attributed to damage to the brain and consequent neurochemical responses (e.g. Castillo et al., 1993; Epstein & Ursano, 2005) or as a direct response to injury-related impairments (e.g. Rapaport et al., 2002; Sagen et al., 2009). However, studies have found little empirical support for the predictive role of neuropathology (localisation of brain injury) (Åström, 1996; Castillo et al., 1993); physical (Castillo, Schultz & Robinson, 1995; Morrison, Johnston & MacWalter, 2000) or cognitive impairment (Barker-Collo et al., 2007) in the aetiology of anxiety post-ABI. Accordingly, researchers have suggested that anxiety may be mediated by psychological factors which serve to influence reactions to injury-related functional changes and environmental changes (Epstein & Ursano, 2005; Morrison et al., 2000).

Notwithstanding the above, to the author’s knowledge a single study to date has explored psychological factors in relation to anxiety following ABI. In this study Morrison, Pollard, Johnston and MacWalter (2005) noted that both (a) low internal locus of control and (b) poor personal confidence in recovery were related to post-stroke anxiety. In regression analyses, 25% of the variance in anxiety was explained by personal confidence in recovery, while internal control beliefs were non-significant. The authors speculated that control beliefs may have been mediated by self-efficacy.
Notably, in this study stroke survivors were assessed six-months after discharge from an acute unit. Given that hospital environments may render personal control unlikely (Scharloo & Kaptein, 1997), it may be useful to explore whether self-efficacy and control beliefs influence anxiety prior to discharge from inpatient care.

**Self-efficacy and locus of control**

According to Bandura (1977) self-efficacy relates to a person’s beliefs in their capabilities to execute behaviours required to achieve a desired outcome. Numerous studies have shown that poor self-efficacy expectancies perpetuate distress subsequent to chronic health conditions (e.g. Edwards, Cecil & Lenoci, 2001; Johnson, Stone, Altmaier & Berdahl, 1998; Stuifbergen, Seraphine & Roberts, 2000).

A construct closely related to self-efficacy is that of locus of control (Lazarus & Folkman, 1984). According to Rotter (1966) internal locus of control denotes beliefs that events are shaped by one’s own behaviour and external control refers to beliefs that outcomes are contingent on other factors (e.g. chance or the actions of others).

Locus of control beliefs are viewed as domain specific, with one of the most widely studied constructs being health control (Luszczynska & Schwarzer, 2005). Several studies have found a positive association between lower internal health control and distress in relation to chronic illness (e.g. Wu, Tang & Kwok, 2004). However, others have found no such association (Wallston, Stein & Smith, 1994).

Although few studies have explored self-efficacy or locus of control in relation to ABI, theoretical models have highlighted the importance of these constructs. For example, Taylor, Todman and Broomfield (2011) in a “social cognitive transition model of post-stroke emotional adjustment” have suggested that stroke survivors’ may be at risk of developing belief systems characterised by poor self-efficacy, consequent to failed attempts at coping with post-stroke changes.
With regard to TBI, Moore, Stambrook and Wilson (1995) have proposed a “model of cognitive beliefs and appraisals following TBI”, wherein it is suggested that:

“following TBI individuals may interpret poor outcomes as unrelated to efforts to control their environment; thereby creating and reinforcing a belief system characterised by an external locus of control and poor self-efficacy” (p. 113).

Following from the literature, it would be reasonable to assume that individuals who have engaged in rehabilitation following ABI and who feel under confident about their capabilities to organise and execute actions required to adapt to injury-related changes (i.e. who have poor self-efficacy and low internal health control beliefs) might feel more anxious about being discharged from inpatient care to return home.

**The current study**

In view of gaps in the existing knowledge-base, the current study sought to (a) determine the prevalence of self-reported discharge-anxiety in a group of individuals with ABI who were due to return home following a period of inpatient rehabilitation and (b) explore the relative influence of demographic, clinical and psychological factors (self-efficacy and control beliefs) on discharge-anxiety. It was recognised that increased knowledge of these relationships might hold implications for the early detection of individuals at risk of discharge-anxiety and support clinical decisions regarding treatment. Based on theory and past research it was hypothesised that:

- Individuals with ABI would report higher levels of discharge-anxiety (or state-anxiety) than generalised / global anxiety (i.e. trait-anxiety).
- There would be significant differential relationships between (a) self-efficacy and (b) internal health control beliefs and discharge-anxiety.
- Self-efficacy and health control beliefs would interact with (and possibly mediate) each-other in influencing and predicting discharge-anxiety.
Methods

Design

The study employed a cross-sectional, single group design to explore relationships between variables. Quantitative methods were used, wherein data was obtained via self-report tools and retrospective reviews of participants’ medical files.

Setting and inclusion criteria

Recruitment took place across three post-acute neurorehabilitation units in the UK, over a 15 month period (from March 2012 to June 2013). During this period, individuals were invited to take part in the research if they: (a) were aged 18 or over; (b) were inpatient; (c) had a diagnosis of ABI (as recorded in their medical file); (d) had capacity to provide informed consent; (e) had adequate English language comprehension skills; (f) were able to effectively communicate their answers to self-report measures; (g) were due to be discharged from the unit (and had been informed of their discharge date); and (h) were due to return home upon discharge.

Ethical considerations

Prior to the initiation of the study, approval was obtained from an NHS Research Ethics Committee (REC) (Appendix 5). Authorisation was also obtained from Research and Development (R&D) Departments at each of the study sites (Appendix 6). In keeping with guidelines stipulated by these authorities, informed consent was obtained from all participants. All data was stored in accordance with Caldicott Principles (Department of Health; DOH, 2003) and guidance outlined in the British Psychological Society’s (BPS) Code of Ethics and conduct (BPS, 2009).
Procedure

The method of recruitment at each of the study sites was based on guidance from the R&D Departments. At each unit, the study was introduced to individuals who met inclusion criteria by a ‘site advisor’ (a qualified psychologist who was familiar with the study). To minimise potential for distress, only those who were aware of their discharge date were approached. During this approach, individuals were given a brief verbal explanation of the study, along with a participant information sheet and consent form (Appendix 7). Accordingly, individuals’ capacity to provide consent to participate in the research was appraised by site advisors in the first instance.

Once verbal consent had been obtained, a meeting with the researcher was arranged. This meeting was facilitated three days after the initial approach and was scheduled so as not to conflict with therapeutic activities. During the meeting participants were given a detailed explanation of the study and encouraged to ask questions. Written consent was then obtained from each participant, following which four self-report measures were completed (see Appendix 8 for measures). To enhance standardisation, measures were verbally administered by the researcher (in the order presented below). This process took roughly 40 minutes on average.

Variables and measures

Although all of the measures included in the study were reputed to have good validity and reliability, to verify their suitability for the study, the opinions of three inpatients on a neurorehabilitation unit were sought with positive feedback. Measures were also piloted with four patients on the unit, with no concerns noted.

---

1 Individuals were presented with a range of measures (including various control and self-efficacy scales). Those that were included in the study were unanimously seen as being the most suitable.
Discharge-anxiety

Anxiety related to discharge was measured in two ways. Firstly, the State-Trait Anxiety Inventory: Form Y (STAI; Spielberger, 1983) was completed. This measure comprises two distinct, 20-item scales: trait-anxiety (i.e. a relatively stable tendency to attend to and report anxiety across situations) and state-anxiety (anxiety that is transient in nature) (Tilton, 2008). On each scale, items are rated using a 4-point Likert format. Individual item scores are summed to yield a total score on each subscale (ranging from 20-80). Higher scores indicate greater anxiety.

Although the STAI has been infrequently used in ABI populations, it has been shown to be reliable in these populations (Curran, Ponsford & Crowe, 2000). In the current study, the internal consistency reliability of both scales was found to be good (trait subscale Cronbach’s $\alpha = 0.92$; state subscale Cronbach’s $\alpha = 0.86$).

In addition to the STAI, the Patient Anxieties Questionnaire (PAQ; Main & Gudjonsson, 2005) was included. This scale was included in view of the absence of a measure of anxiety related to discharge from neurorehabilitation. Notably, the PAQ was originally developed to appraise anxiety related to discharge from forensic units.

The PAQ comprises six, self-report items, each of which are rated on a scale from 1-7. Scores on each item are summed to yield an overall, total score. Total scores range from 7-42, with higher scores indicating greater discharge-anxiety.

Prior to the scale being submitted for ethical review, the wording of one item (i.e. “how worried are you about leaving the medium secure unit”) was amended. Permission to use the scale in this way was granted by the author (Appendix 9).

To establish face validity, the adapted scale was given to four inpatients on a neurorehabilitation unit for comments, with no concerns noted. Internal consistency of this version was found to be good in the current study (Cronbach’s $\alpha = 0.87$).
Health control beliefs

Participants’ control beliefs were assessed via the Multidimensional Health Locus of Control Scale (MHLC; Wallston et al., 1994). This self-report scale was designed to be used with a range of health conditions and has been shown to possess good construct validity (Wallston et al., 1994). The scale contains 18 items, which are rated using a Likert format (from strongly disagree to strongly agree).

The MHLC yields scores on three distinct subscales: “internal” “chance” and “others”, wherein higher scores reflect greater beliefs about control being governed by that domain. Subscale scores are seen as independent (these are not summed). Therefore, for the purposes of the present study, only the internal subscale (MHLC-Internal) was included in statistical analyses. Scores on this subscale range from 6-36, with higher scores indicating greater perceptions of an internal locus of control. In the current study sample Cronbach’s α of the MHLC-Internal subscale was 0.82.

Self-efficacy beliefs

Self-efficacy beliefs were assessed via the Traumatic Brain Injury Self-Efficacy Questionnaire (TBI-SE; Cicerone & Azulay, 2007). This self-report tool was designed for use with ABI populations and is considered to possess good construct validity and internal consistency reliability (Cicerone & Azulay, 2007). The scale comprises 13 statements (each of which is preceded by the question “how confident are you that you can...”). Responses to individual items are rated on a scale from 1-10 (i.e. from not at all confident to totally confident). Individual item scores are summed for an overall score, which ranges from 13-130. Higher scores reflect greater self-efficacy.

Permission to include the TBI-SE in the current study was obtained from the author (Appendix 9). Cronbach’s α of the total scale was 0.94 in this study.
Other information

Following the administration of self-report measures, demographic data on age, gender and ethnicity was recorded for each participant. The following clinical information was also obtained from their medical files: ABI diagnosis; severity of ABI; injury location (left hemisphere, right hemisphere or diffuse damage); and information related to inpatient care (number of prior admissions, length of current admission and time to discharge) (see ‘demographic and clinical information sheet’ in Appendix 10).

Sample size requirements

Prior to recruitment, power calculations were carried out to determine the size of the sample that would be required in order to attain statistically significant findings. Relevant academic literature, pertaining to statistical analyses, was also consulted.

Literature regarding bivariate correlations indicated that a sample of between 28-35 participants would be needed to achieve a high level of power ($\beta = 0.80$) to attain a statistically significant result ($p < 0.05$; two-tailed) if the magnitude of the correlation co-efficient was moderate ($r = 0.3$; Cohen, 1988; Bonnett & Wright, 2000).

It was planned that, in the current study, no more than three predictor variables would be included in a regression model. A calculation revealed that at least 36 participants would be required for a regression analysis with three predictors (where the level of power was 0.80 and the effect size was large - $R^2 = 0.26$; Cohen, 1992). Academic literature further indicated that between 10-15 participants per predictor would be needed to achieve sufficient power in testing a regression model (Field, 2009). Therefore, the study aimed to recruit between 36-45 participants.
Characteristics of the sample

The final sample comprised 31 men (74%) and 11 women (26%) (N= 42). The average age of the sample was 44.74 years (SD =13.35, range 19-74 years). (See Table 4 in Appendix 11 for a summary of the characteristics of the sample).

In terms of ethnicity: 31 participants (74%) self-identified as White British or Irish; three (7%) as Black British and two (5%) as Afro-Caribbean. The remaining six participants (14%) self-identified as belonging to various other ethnic groups (including Indian, Pakistani, South-Asian and mixed-heritage backgrounds).

Medical records indicated that 22 participants (52%) had suffered a stroke (cerebral infarction or haemorrhage), while 18 (43%) had sustained TBI (through: road traffic accidents; complications from falls; tumoral or vascular neurosurgery). Two participants (5%) had a diagnosis of anoxic brain injury; a consequence of areas of the brain being deprived of oxygen (Peskine, Picq & Pradat-Diehl, 2004).

With regard to the location of trauma to the brain: 20 participants (47%) had sustained injury to the right hemisphere; 10 (24%) had left hemisphere injury; and four (10%) had diffuse brain damage. In eight cases (19%), ABI location was not specified. Information regarding ABI severity was unavailable for the majority (72%) of participants; however, admission to a post-acute rehabilitation unit would suggest that they sustained moderate to severe injuries (Maas, Stocchetti & Bullock, 2008).

The majority of participants (n = 37; 83%) had been admitted to the unit from an acute ward, while five (12%) had been transferred from another post-acute unit. At the time of the study, the mean length of time since injury was 4.52 months (SD = 2.59, range 1-12) and mean length of stay on the unit was just under three months (M = 82.29 days; SD = 49.75; range 27-240 days). On average, participants were assessed 4.24 days (SD = 3.87; range 0-14) prior to discharge from the unit.
Statistical analyses

Data were analysed using SPSS for Windows; Version 17.0 (SPSS, 2009). Descriptive statistics were used to detail the characteristics of the study sample (as reported above). The prevalence of anxiety within the sample was appraised by comparing scores on the two STAI subscales, alongside the adapted-PAQ.

The aims of the research were subsequently addressed in more detail, by exploring associations between demographic, clinical and psychological factors and discharge-anxiety (as determined by the adapted-PAQ). Differences in reported discharge-anxiety between groups were appraised using Mann-Whitney \( U \) tests, while bivariate correlations were performed among continuous variables.

Variables which were found to be independently associated with discharge-anxiety were subsequently included in a hierarchical regression model (with the aim being to test the effects of predictor variables, independent of the influence of others) (Field, 2009). In addition, tests of indirect effects (meditation) were carried-out using bootstrapping techniques (Hayes, 2009), which were not restricted by assumptions of normality and were considered suitable for small samples (Hayes, 2009; Russel & Dean, 2000). Variables appraised in this way were self-efficacy, health locus of control and discharge-anxiety. Due to the exploratory nature of the research the level of statistical significance for all analyses was defined as \( p < 0.05 \) (two-tailed).

Normality of distributions

Data were prepared for analysis using guidelines by Pallant et al. (2010). Descriptive statistics confirmed that there were no missing data or significant outliers. Normality of distributions’ were explored via the use of histograms and Shapiro-Wilk tests. Results indicated normal distributions of data for: age (years); state-anxiety (STAI-State); Internal control beliefs (MHLC-Internal); and self-efficacy (TBI-SE).
However, the following data was positively skewed: trait-anxiety (STAI-Trait); discharge-anxiety (adapted-PAQ); time since injury; length of admission and time until discharge (see Appendix 12 for histograms). These variables were therefore square-root transformed (Field, 2009). This achieved a normal distribution of data for both trait- and discharge-anxiety. However, data related to time since injury, length of admission and time until discharge remained skewed following transformations (see Appendix 13). Therefore, non-parametric tests were used where appropriate.

**Differences between study sites**

A Kruskal-Wallis test revealed no significant differences in terms of: length of time since ABI \( [H(2) = 4.05, p = 0.13] \); length of current admission \( [H(2) = 2.74, p = 2.56] \) or time until discharge \( [H(2) = 2.61, p = 0.27] \) across the three study sites.

A one-way ANOVA further confirmed that scores on study measures did not differ significantly between sites. Table 5 (below) presents the results of this ANOVA. Therefore, subsequent analyses were carried-out using the entire sample \( (N = 42) \). (See Table 8 in Appendix 14 for a summary of scores obtained on each measure).

**Table 5**

<table>
<thead>
<tr>
<th>Measure</th>
<th>( df ) between groups</th>
<th>( df ) within groups</th>
<th>( F )</th>
<th>( p )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adapted-PAQ(^a)</td>
<td>2</td>
<td>39</td>
<td>1.17</td>
<td>0.32</td>
</tr>
<tr>
<td>STAI: State-Anxiety</td>
<td>2</td>
<td>39</td>
<td>0.80</td>
<td>0.46</td>
</tr>
<tr>
<td>STAI: Trait-Anxiety(^a)</td>
<td>2</td>
<td>39</td>
<td>1.15</td>
<td>0.37</td>
</tr>
<tr>
<td>MHLC: Internal</td>
<td>2</td>
<td>39</td>
<td>0.15</td>
<td>0.86</td>
</tr>
<tr>
<td>TBI Self-Efficacy</td>
<td>2</td>
<td>39</td>
<td>0.76</td>
<td>0.47</td>
</tr>
</tbody>
</table>

\(^a\)Scores were square-root transformed prior to analyses
Results

Prevalence of anxiety

The final sample ($N = 42$) had a mean STAI trait-anxiety score of 37.29 ($SD = 10.52$, range 20-68) and a mean state-anxiety score of 42.40 ($SD = 14.68$, range 20-79). A students $t$ test (in which both trait and state scores were square root transformed) revealed that this difference was statistically significant [$t(41) = 2.81, p = 0.008$], with state-anxiety being higher. According to the developer of the STAI, subscale scores of $\geq 45$ are suggestive of clinically significant anxiety (Spielberger, 1983). Descriptive statistics revealed that only six participants (14%) had a trait-anxiety score of $\geq 45$, while 19 participants (45%) had a state-anxiety score of $\geq 45$.

Discharge-related anxiety

The mean adapted-PAQ score for the sample was 18.12 ($SD = 8.27$, range 6-36). A moderate positive correlation was found between this score and STAI trait-anxiety ($r = 0.51$, $p < 0.01$; Cohen, 1992) and a large positive correlation between this score and STAI state-anxiety ($r = 0.77$, $p < 0.001$; Cohen, 1992).

Factors associated with discharge-anxiety

In exploring relationships between variables the following adjustments were made

- In a point-biserial correlation exploring ethnicity, participants from all black and minority ethnic (BME) groups were combined to form one group ($n = 11$) that was compared with a group comprising Caucasian participants ($n = 31$)

- In a point-biserial correlation exploring ABI diagnosis, participants with a diagnosis of anoxic brain damage ($n = 2$) were included in the TBI category.

- In a one-way ANOVA exploring brain areas affected by ABI, participants for whom relevant clinical information was unavailable ($n = 8$) were excluded.
Table 9
Associations between variables and discharge-anxiety

<table>
<thead>
<tr>
<th>Variable / outcome measure</th>
<th>Adapted-PAQ score&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Test statistic</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographic factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td>( r = 0.31^* )</td>
<td>0.04*</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td>( U = 125.50^\dagger )</td>
<td>0.20</td>
</tr>
<tr>
<td>Ethnicity&lt;sup&gt;b&lt;/sup&gt; (Caucasian vs. BME)</td>
<td></td>
<td>( r_{pb} = 0.12^T )</td>
<td>0.49</td>
</tr>
<tr>
<td><strong>Clinical characteristics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ABI diagnosis&lt;sup&gt;b&lt;/sup&gt; (Stroke vs. TBI)</td>
<td></td>
<td>( r_{pb} = -0.01^T )</td>
<td>0.97</td>
</tr>
<tr>
<td>Brain areas affected by injury&lt;sup&gt;b&lt;/sup&gt; (n =32)</td>
<td></td>
<td>( F(2,31) = 0.076^\dagger )</td>
<td>0.92</td>
</tr>
<tr>
<td>(right hemi-, left hemi- or diffuse damage)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Length of time since ABI event (months)</td>
<td></td>
<td>( r = 0.18^* )</td>
<td>0.25</td>
</tr>
<tr>
<td>Length of current admission (days)</td>
<td></td>
<td>( r = 0.05^* )</td>
<td>0.73</td>
</tr>
<tr>
<td>Length of time until discharge (days)</td>
<td></td>
<td>( r_s = -0.06^p )</td>
<td>0.70</td>
</tr>
<tr>
<td><strong>Psychological factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Internal control beliefs (MHLC-Internal)</td>
<td></td>
<td>( r = -0.37^* )</td>
<td>0.02*</td>
</tr>
<tr>
<td>Self-efficacy beliefs (TBI-SE Scale)</td>
<td></td>
<td>( r = -0.84^* )</td>
<td>&lt; 0.001**</td>
</tr>
</tbody>
</table>

<sup>a</sup>Variable was square-root transformed; <sup>b</sup>Variable was dichotomised; <sup>\dagger</sup>Mann-Whitney \( U \)-test; <sup>\ddagger</sup>One-way ANOVA; <sup>T</sup>Point-biserial correlation; <sup>*</sup>Pearson’s \( r \) correlation; <sup>p</sup>Spearman’s \( \rho \) correlation

\( N = 42 \) unless otherwise stated \( ^* p < 0.05 \quad ^{**} p < 0.001 \)
Demographic, clinical and psychological correlates

As can be seen from Table 9 (overleaf), there was a small but significant positive correlation between age and discharge-anxiety ($r = 0.31$, $p = 0.04$) and a small but significant negative correlation between internal control beliefs and discharge-anxiety ($r = -0.37$, $p = 0.02$). There was a large negative correlation between self-efficacy beliefs and discharge-anxiety ($r = -0.84$, $p < 0.001$). No other demographic or clinical variables were correlated with discharge-anxiety at a statistically significant level.

Internal health control and self-efficacy beliefs

MHLC-Internal scores ranged from 9-35 ($M = 23.05$, $SD = 5.88$).
When the median score of 22.50 was used to divide the sample into ‘low’ and ‘high’ internal control (IC) groups, participants in the low IC group were seen to have significantly higher discharge-anxiety ($M = 4.55$, $SD = 0.97$) than those in the high IC group ($M = 3.75$, $SD = 0.82$) [$t(40) = 2.932$, $p = 0.006$] (see Figure 1 in Appendix 15).

TBI-Self-Efficacy scores ranged from 51-128 ($M = 91.93$, $SD = 22.18$).
When the median score of 90.50 was used to divide the sample into ‘high’ and ‘low’ self-efficacy (SE) groups, those in the low SE group were seen to have significantly higher discharge-anxiety ($M = 4.89$, $SD = 0.65$) than those in the high SE group ($M = 3.40$, $SD = 0.60$) [$t(40) = 7.69$, $p < 0.001$] (see Figure 2 in Appendix 15).

Potential predictors of discharge-anxiety

Three variables (age, control beliefs and self-efficacy) were significantly associated with discharge-anxiety in univariate analyses. These variables were included in a hierarchical regression model (with discharge-anxiety as the criterion variable). As can be seen from Table 10 (overleaf) at step one of the model, age alone accounted for 10% of the variance in discharge-anxiety. This model was
significant \( F(1,40) = 4.32, p = 0.45 \). At the second step, internal control beliefs were included. This model was again significant \( F(2,39) = 5.57, p = 0.007 \). Internal control beliefs explained a further 12% of the variance in discharge-anxiety when age was taken into account. At the third step self-efficacy was added to the model. This overall model (with all three predictors) was significant \( F(3,38) = 31.15, p < 0.001 \) and accounted for 71% of the variance in discharge-anxiety. However, at this third and final step, age and control beliefs did not contribute significantly to the model.

The largest variance inflation factor value was < 3 (with all tolerance values < 0.2) indicating that multicollinearity among the predictors did not unduly influence the aforementioned regression estimates (Tabachnick, Fidell & Osterlind, 2001).

Table 10
Results of hierarchical regression for variables predicting discharge-anxiety

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE B</th>
<th>( \beta )</th>
<th>t</th>
<th>( R^2 )</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Step 1</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>0.02</td>
<td>0.01</td>
<td>0.31</td>
<td>2.08</td>
<td>0.10</td>
<td>0.05*</td>
</tr>
<tr>
<td><strong>Step 2</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>0.02</td>
<td>0.01</td>
<td>0.29</td>
<td>2.06</td>
<td>0.22</td>
<td>0.007*</td>
</tr>
<tr>
<td>MHLC-Internal</td>
<td>-0.06</td>
<td>0.02</td>
<td>-0.35</td>
<td>-2.50</td>
<td></td>
<td>0.02*</td>
</tr>
<tr>
<td><strong>Step 3</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.71</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>Age (years)</td>
<td>0.01</td>
<td>0.007</td>
<td>0.12</td>
<td>1.18</td>
<td>0.01</td>
<td>0.24</td>
</tr>
<tr>
<td>MHLC-Internal</td>
<td>-0.001</td>
<td>0.02</td>
<td>-0.01</td>
<td>-0.78</td>
<td></td>
<td>0.94</td>
</tr>
<tr>
<td>TBI-Self-Efficacy</td>
<td>-0.04</td>
<td>0.004</td>
<td>-0.80</td>
<td>-8.06</td>
<td></td>
<td>&lt; 0.001**</td>
</tr>
</tbody>
</table>

*Square-root transformed adapted-PAQ score (dependent variable)

(\( N = 42 \)) \( \beta = \) Unstandardised co-efficient \( * \ p = 0.05 \) \( ** \ p < 0.001 \)
A univariate regression, in which only self-efficacy was entered as a predictor with discharge-anxiety as the dependent variable, was performed. This model was significant \[ R^2 = 0.69, t = -9.66, F(1,40)= 93.44, p < 0.001 \] and demonstrated that self-efficacy beliefs alone accounted for 69% of the variance in discharge-anxiety.

**Tests of indirect effects: meditation**

Mediatational analysis focuses on the difference between the *direct effect* of an independent variable (X) on an outcome variable (Y) and the *indirect effect* of this relationship, after an interceding or mediating variable (M) has been taken into account (Preacher & Hayes, 2008). In this study, two meditational models were tested. The first model explored the indirect effect of internal health control beliefs (X) on discharge-anxiety (Y) through self-efficacy (M) (see Figure 4 in Appendix 16). The second model explored the indirect effect of self-efficacy (X) on discharge-anxiety (Y) through health control beliefs (M) (see Figure 5 in Appendix 16).

Using an SPSS macro (syntax; Preacher & Hayes, 2004) 1,000 identically sized datasets were created by iteratively resampling cases from the original dataset. Confidence intervals for the direct path coefficients were then derived. Using this method, mediation was considered to be significant if the upper and lower bounds of the bias-corrected confidence intervals did not contain zero (Hayes, 2009).

As can be seen in Table 11, results indicated that control beliefs were *not* a significant mediator of the relationship between self-efficacy and discharge-anxiety \[ R^2 = 0.84, F(1,40)= 47.36, 95\% \text{ BCaCI}= (-0.003, +0.01), p=(ns) \]. Conversely, self-efficacy was a significant mediator of the relationship between control beliefs and discharge-anxiety \[ R^2 = 0.12, F(1,40)= 7.63, 95\% \text{ BCaCI}= (-0.11, -0.02), p < 0.05^* \].
Table 11
Bootstrapped point estimates and bias-corrected confidence-intervals for the meditational (indirect) effects of locus of control and self-efficacy

<table>
<thead>
<tr>
<th>Partial effects of variables on discharge-anxiety</th>
<th>$\beta$</th>
<th>$SE$</th>
<th>$t$</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MHLC-Internal</td>
<td>-0.01</td>
<td>0.14</td>
<td>-0.09</td>
<td>0.93</td>
</tr>
<tr>
<td>TBI Self-efficacy</td>
<td>-0.31</td>
<td>0.04</td>
<td>-8.45</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

**Indirect effect:** Internal locus of control as a mediator of the relationship between self-efficacy and discharge-anxiety

<table>
<thead>
<tr>
<th>Bootstrap results</th>
<th>$M$</th>
<th>$SE$</th>
<th>LLCI</th>
<th>ULCI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>-0.001</td>
<td>0.02</td>
<td>-0.03</td>
<td>0.04</td>
</tr>
</tbody>
</table>

**Indirect effect:** Self-efficacy as a mediator of the relationship between internal locus of control and discharge-anxiety

<table>
<thead>
<tr>
<th>Bootstrap results</th>
<th>$M$</th>
<th>$SE$</th>
<th>LLCI</th>
<th>ULCI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>-0.52</td>
<td>0.21</td>
<td>-1.01</td>
<td>-0.16*</td>
</tr>
</tbody>
</table>

Bootstrap sample size = 1000  
$\beta$ = unstandardised regression co-efficient  
LL = lower limit; UL = upper limit  
CI = 95% confidence interval

On conclusion of the study, a summary of the above findings was made available on a website; that was accessible to participants (as detailed in the participant information sheet). An end of study report was also sent to: the NHS REC; site advisors; and relevant NHS Trust R&D Departments (see Appendix 17).
Discussion

This study explored the prevalence of discharge-related anxiety amongst 42 individuals who had sustained moderate to severe ABI and who were imminently due to return home following a period of inpatient neurorehabilitation. The study also investigated differential relationships between psychological factors, alongside the relative influence of demographic and clinical characteristics, on discharge-anxiety.

Recruitment to the study took place across three post-acute neurorehabilitation units in the UK. At the time of the study, the mean length of time since ABI was just over four months and the mean length of stay on the unit was around three months. On average, participants were assessed four days prior to their discharge home.

Whilst few participants (14%) recorded substantially elevated trait-anxiety almost half (45%) of the sample recorded clinically significant levels of transient, state-anxiety symptomatology. Notably, state-anxiety (appraised via the STAI) was highly associated with discharge-anxiety (assessed via the adapted-PAQ).

The majority of past studies have used the HADS to explore anxiety in ABI populations, limiting direct comparison of prevalence rates. Nevertheless, studies that have used the HADS in inpatient settings (up to four months post-ABI) have noted comparable trait-anxiety prevalence between 14-22% (De Wit et al., 2008; Jorge, Robinson & Starkstein, 1993; Townend et al., 2007), while community-based studies (exploring anxiety up to five years post-ABI) have reported prevalence rates to be as high as 36% (Bergersen, Froslie, Sunnerhagen & Schanke, 2010).

A literature search revealed a single relevant study by Curran et al. (2000) wherein the STAI was used. This study explored coping up to five years post-TBI. Interestingly, findings revealed only slightly lower state-anxiety ($M=41.89$, $SD=16.20$) than in the current study, but significantly higher trait-anxiety ($M=44.45$, $SD=15.14$).
Taken together, the above findings suggest that transient, state-anxiety may be more prevalent in the lead up to discharge from inpatient care (possibly as individuals experience uncertainty about their ability to cope with impending change) (Rusconi & Turner-Stokes, 2003); whereas following discharge to the community (several years after TBI) individuals may be at risk of developing a propensity to experience anxiety across situations (i.e. trait-anxiety) (possibly as a consequence of injury-related difficulties and multiple experiences of failure) (Fletcher, 2009).

Of primary interest in the study were relationships between psychological factors and discharge-anxiety. Findings provided support for hypotheses that there would be associations between both (a) self-efficacy and (b) internal health control beliefs and discharge-anxiety. Although a lack of similar research in ABI populations makes it difficult to compare findings with other studies in this area, the link between self-efficacy and discharge-anxiety could well be explained by evidence from other fields showing that poor perceptions of self-efficacy perpetuate emotional distress subsequent to health conditions (e.g. Edwards et al., 2001; Stuifbergen et al., 2000).

The literature investigating the role of health locus of control beliefs in relation to chronic health conditions was also supported by the findings of the current study, in that participants who reported low internal health control beliefs were significantly more anxious about discharge than those who reported high internal control beliefs. For example, Wu et al. (2004), in a study examining emotional distress amongst 159 elderly women suffering from varied chronic illnesses, reported a significant association between lower internal health control and greater emotional distress. While the average age of participants in the study by Wu et al. (2004) ($M=74$ years, $SD=6.80$) was much higher than that of the current sample ($M=45$ years, $SD=13.35$) both studies also revealed that age was correlated with negative emotional states
(such that older participants reported greater anxiety or distress). These findings were in keeping with research in other illness populations, wherein anxiety has been shown to increase with advancing age (e.g. Härter, Conway & Merikangas, 2003). However, findings contradicted studies showing anxiety to be more prevalent among younger stroke survivors (Castillo et al., 1993; Schultz, Castillo, Kosier & Robinson, 1997). One explanation may be that, since stroke is infrequent in younger cohorts, such an event may cause greater distress in such cohorts (Petrea et al., 2009).

Hierarchical regression analyses facilitated an investigation of the relative influence of age, internal health control and self-efficacy beliefs on discharge-anxiety. Together these three variables explained 71% of the variance in discharge-anxiety. However, while age and locus of control beliefs accounted for 10% and 12% of the variance at step one and two of the regression model respectively, when self-efficacy was entered at the third step, this emerged as the most salient predictor. Specifically, at the final step, age and internal control beliefs did not contribute significantly to the model, with self-efficacy alone explaining 69% of the variance in discharge-anxiety.

Investigation of the indirect (meditational) effects of psychological factors revealed that internal control beliefs did not significantly mediate the relationship between self-efficacy and discharge-anxiety; whereas self-efficacy mediated the relationship between internal control beliefs and discharge-anxiety. Accordingly, findings supported theoretical assumptions that self-efficacy may be derived through causal attributions (Schwarzer & Renner, 2000). For example, Moore et al. (1995) proposed an overlap between control and self-efficacy beliefs following TBI, such that “generalized expectancies [...] arising from pervasive non-contingent and suboptimal outcomes in many aspects of the TBI patient’s life, lead to feelings of low personal control over the environment and contribute to lowered self-efficacy” (p. 118).
In keeping with the above, findings of the current study also supported the premise that self-efficacy beliefs may be more proximal to the consequences of illness conditions, whereas internal control beliefs exert their influence at a more distal level (Endler, Kocovski & Macrodimitris, 2001). It is worth noting that research among other illness populations has also demonstrated support for this hypothesis; such that perceptions of self-efficacy mediate the relationship between internal health control beliefs and distress (e.g. Schiaffino & Revenson, 1992; Wu et al., 2004).

Lastly, findings that none of the clinical characteristics examined in the current study were significantly related to discharge-anxiety were somewhat surprising, given that past research has demonstrated associations between anxiety following ABI and lesions in the right brain hemisphere (Åström, 1996; Castillo et al., 1993). Although the relatively small sample in the current study may have reduced statistical power to detect a significant effect, it is worth noting that studies with larger ABI samples have also found no effect for location of ABI on anxiety (e.g. Morrison et al., 2000; N=71).

**Limitations of the study**

The results of this study should be considered in light of several limitations. Firstly, the study sample was voluntary and fairly small. Recruitment took place across three post-acute neurorehabilitation units which were located in suburban settings in England. In addition, most participants were around three month’s post-ABI and all had sustained moderate to severe injury. As such, caution should be taken not to generalise the specifics of findings to other ABI populations or settings.

It is also worth noting that individuals from BME groups may have been under-represented in the current sample (RCP, 2012). A greater proportion of participants from BME groups would have enabled further scrutiny of the manner in which culture impacts on anxiety, self-efficacy and control beliefs. This may have been particularly
relevant as culture influences the development of belief systems (Bandura, 2002).

With regard to gender, 31 men and 11 women were recruited to the study, possibly reflecting that men are more likely than women to sustain ABI (Turner-Stokes, 2003).

Secondly, the reliance on self-report measures raised question as to whether findings may have been distorted by bias. In the case of ABI, self-report data may be adversely affected by cognitive difficulties (Port, Willmott & Charlton, 2008). However, the use of Likert scales comprising relatively short and concrete items has been shown to yield accurate data in such populations (Moore et al., 1995). This seemed to be the case in the current study as all measures demonstrated good internal consistency reliability (Cronbach, 1951). It is worth noting that reliability may have been enhanced through verbal administration of measures (Lezak, 2004). However, this non standardised method of delivery would have likely adversely affected the original psychometric properties of these measures (Port et al., 2008).

With regard to the PAQ and STAI, there is the issue of whether these scales sufficiently embodied the constructs under study. For example, it may be that health professionals and family carers have different understandings of discharge-anxiety, while the STAI may have been confounded by anxiety related to the assessment situation (Spielberger, 1983). Nonetheless, strengths of both these measures lay in that they did not include items that may have related to somatic or physical complaints as is a recognised limitation of the HADS measure (Tilton, 2008).

It is also possible that findings of the current study may have been subject to response bias, as more anxious individuals may have been more or less likely to participate in the research. This is particularly noteworthy given the high prevalence of self-reported anxiety in the sample. Unfortunately a lack of information relating to reasons for non-participation limited the accuracy with which interpretations about
prevalence of anxiety in the study population could be made. The absence of a comparison group also precluded exploration as to whether anxiety prevalence varied in other illness populations (e.g. patients with ABI vs. orthopaedic injuries).

Another set of implications from the current study relates to the locus of control construct. Given that the MHLC tapped internal health control beliefs, findings raised query as to whether domain specific or global measures of control may be more useful (Wallston et al., 1994). Unfortunately, because all of the MHLC subscales would have yielded too many comparisons, only the internal subscale was included in statistical analyses. Further examination of the other subscales may be a useful area for future research.

In addition, some potentially relevant factors for anxiety outcome may not have been included in the study. For example, elements of individuals’ rehabilitation programs were not quantified. Although efforts were made to include information related to the management of anxiety (e.g. pharmacological or psychotherapeutic intervention) information pertaining to these factors was limited. Information from collateral sources (e.g. clinicians) may have supplemented file information.

Lastly, information relating to anxiety prior to ABI was limited. Therefore, it was not possible to determine whether anxiety reported in the current study was new or ongoing. This was compounded by limitations inherent in the cross-sectional design of the study wherein it was not possible to determine causal associations between variables. It may be that individuals who were more anxious were more likely to report poor perceptions of self-efficacy or that anxiety renders a person more vulnerable to experiences of failure, which in turn impacts on self-efficacy beliefs. Nonetheless, regardless of causal pathways, findings of the study suggest that health locus of control and self-efficacy may be important in addressing anxiety post-ABI.
Clinical implications of findings

Substantially elevated levels of anxiety following ABI have been associated with numerous adverse consequences, including increased dependence (Rapoport et al., 2002) and reduced quality of life (Åström et al., 1996; Sturm et al., 2004). Therefore, findings of this study may hold important implications for clinical practice.

Firstly, findings suggest that it may be important for clinicians to inquire about individuals’ concerns in the lead-up to their discharge from inpatient care, so that the nature and degree of anxiety can be determined. This may enable anxiety-provoking issues to be addressed while the person is still resident on the inpatient unit (as opposed to post-discharge when professional support may be less accessible).

Secondly, findings that individuals with lower internal control and poorer self-efficacy were more anxious about discharge (than those with higher control and self-efficacy expectancies) may have implications for interventions. For example, where appraisals are inaccurate, behavioural experiments could be used to promote more realistic evaluations; whereas where appraisals are accurate, mindfulness-based interventions may be of benefit (Hofmann, Sawyer, Witt & Oh, 2010). In addition, opportunities to develop competence through mastery experiences could be used to enhance self-efficacy (Bandura, 1989). One such intervention, which has shown to be effective in supporting successful transitions from inpatient care to the community for individuals with ABI, is the transitional living unit (TLU; Minnes, Harrick, Carlson & Johnston, 1998; Olver & Harrington, 1996; Simpson et al., 2004). TLU’s involve programs that are conducted in a home-like environment with an emphasis on the development of skills necessary for community living (Kendall, Ungerer & Dorsett, 2003). However, such units are not in widespread use and research is needed to validate their effectiveness (Kendall et al., 2003).
Lastly, it is notable that most participants in the current study reported having received no formal therapeutic intervention, aimed at addressing anxiety, in the lead-up to their discharge. While the possibility of self-report bias is acknowledged, it is also recognised that empirical support for the effectiveness of both medication and traditional psychotherapeutic approaches (e.g. cognitive-behavioural therapy; CBT) for targeting anxiety in people with ABI is limited (Campbell-Burton et al., 2011; Soo & Tate, 2007; Williams, Evans & Fleminger, 2003). Notably, evidence suggests that cognitive impairments can limit the effectiveness of CBT in this context (Anson & Ponsford, 2006). Accordingly, interventions aimed at addressing self-efficacy and control cognitions (as outlined above) may be of particular benefit; both in supporting individuals with ABI during discharge from inpatient care and in helping to reduce strain placed on their informal networks during this transition (Turner et al., 2009).

**Directions for future research**

Findings of this study highlight a number of possibilities for future research. Firstly, while age, self-efficacy and internal control beliefs were seen to explain a substantial proportion (71%) of the variance in discharge-anxiety, further research may be warranted to identify factors that explain the remaining variance. Several potentially important variables (e.g. perceptions of support and coping strategies) were not addressed in this study and may have implications for anxiety outcome.

Secondly, it may be useful to undertake research in order to validate the use of self-report measures of anxiety (e.g. the STAI and PAQ) in ABI populations. Conspicuously, measures that are currently in widespread use (e.g. the HADS) have demonstrated poor sensitivity for anxiety in ABI populations (Sagen et al., 2009).

In addition, longitudinal research aimed at investigating the way in which self-efficacy and control beliefs influence anxiety throughout the course of inpatient
admissions may yield interesting insights. This may be particularly relevant given that the current study found no association between discharge-anxiety and number of days until discharge. While the relatively small sample may have reduced statistical power to detect a significant effect, it may also be the case that anxiety fluctuates throughout the course of inpatient admission or that this peaks when individuals are imminently due to return home but discharge preparation is not yet complete.

Lastly, it is recognised that the study adopts a negative approach by examining relationships between psychological constructs and discharge-anxiety. Future studies may seek to investigate how control and self-efficacy beliefs relate to positive adjustment to ABI or support experiences of successful discharge to the community and home life. For example, it may be that more anxious individuals have a more realistic appreciation of the difficulties inherent in community living (e.g. the challenges involved in adapting to ABI-related functional impairment), but that their anxiety prevents them from meeting these challenges successfully, or alternatively that lower levels of anxiety enable individuals to function efficiently in the community by being pro-active in seeking support. Perhaps more importantly, somewhat overlooked in the research is the fact that some individuals with ABI experience minimal levels of anxiety. Their coping strategies should be explored, alongside outcome studies of interventions aimed at targeting discharge-related anxiety.
Conclusions

Findings of the current study suggest that: (a) anxiety is prevalent in the lead-up to discharge from inpatient neurorehabilitation to home following ABI and (b) age, internal health control and self-efficacy beliefs may play an important role in influencing discharge-related anxiety. These findings are consistent with theoretical models of emotional adjustment following ABI, as well as existing empirical evidence relating to anxiety in the context of chronic health conditions. They also supplement existing anecdotal accounts (from qualitative research), which suggest that adjusting emotionally to transitions from inpatient care settings to the community and home-life poses a significant challenge for individuals with ABI.

Findings do not support relationships between gender, ethnicity or clinical factors (specifically ABI diagnosis and location of brain injury) and discharge-anxiety. Although the cross-sectional nature of the research precludes any inferences about direction of causality, findings nonetheless provide some evidence to suggest that psychological factors (self-efficacy and control beliefs) may exert a stronger influence on discharge-anxiety than neuropathology and that these factors may be important in developing interventions aimed at addressing this phenomenon. However, in view of the limitations of the current study and the early stages of related research, further investigations aimed at replicating and expanding on current findings, are warranted.
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Critical appraisal of the research process and key learning points

The current study aimed to explore relationships between psychological constructs and self-reported discharge-related anxiety in a group of 42 participants with a diagnosis of acquired brain injury (ABI). The following sections present an appraisal of this research; by addressing four questions outlined by the Doctorate in Clinical Psychology Program at Canterbury Christ Church University for this purpose.

What research skills and abilities have you developed from carrying-out this project and what do you think you need to learn further?

Carrying out this project has developed my skills in a number of areas; from identifying gaps in the existing evidence-base and consulting with clinicians and service-users who have in-depth knowledge of the area of interest; to designing information sheets, selecting measures and obtaining ethical approval; through to data collection and analysis; and the final writing-up of findings for dissemination.

In terms of gaining access to carry-out research within the National Health Service (NHS), I have developed an appreciation of the time this can take and the obstacles that can be encountered. For example, efforts to make contact with clinicians (who might act as “site advisors” and facilitate access to participants) at various neurorehabilitation units proved unsuccessful. On reflection, this could have been for any number of reasons. It may be that following ABI individuals’ may be perceived as “too unwell” to take part in research (Slyter, 1998) or that clinicians working on busy wards simply do not have the time to invest in research activities. Therefore, in carrying-out future research in this area, I would need to gain specific knowledge of local services, in order to identify individuals who might be able to facilitate access to participants’. Involving local clinicians in the early design stages
of future projects may also help to encourage “buy-in”. This may be particularly relevant, as at two potential study sites feedback from Research and Development (R&D) Departments indicated a need to prioritise research carried-out by clinicians who were internal to the organisation. I am therefore mindful of the move toward a commercially viable, business model within the NHS (Ward, 2011) and the possible research implications of initiatives such as “Payment by Results” (e.g. a need to prioritise service evaluation or funded projects). I am also conscious that I will need to develop an awareness of how to secure funding through grant applications in the future, as this is likely to be a key feature of future research endeavours.

With regard to recruitment, carrying-out a multi-site project necessitated efficient liaison with site advisors and sharpened my time-management skills. Nevertheless, time constraints and competing commitments meant that it was not always possible to meet with participants in the immediate lead-up to their discharge (particularly at times when individuals were simultaneously discharged from different units and it was not possible to attend both sites on the same day). Therefore I learnt to be realistic in terms of what could be achieved within the time-frames available for the project (e.g. some participants were seen almost two weeks prior to discharge).

On a more positive note, I thoroughly enjoyed meeting with participants’ and noted that as data collection progressed, I became skilled at adapting my interactions in view of the communication and visual difficulties they sometimes presented with. Although verbal administration of measures was time consuming, I recognised that this enabled individuals’ who might otherwise not have been able to participate in the research to do so (e.g. those with visual field neglect or expressive aphasia). I also learnt a great deal about the use of computerised communication aids and feel that this is an area in which I could continue to expand my knowledge in the future.
Lastly, in carrying-out this project my understanding of multivariate analysis techniques has increased. Although I came to the project with some knowledge of regression approaches and how to examine indirect effects, using steps outlined by Baron and Kenny (1986), this was my first experience of “bootstrapping techniques” (Hayes, 2009). I found supervision and key publications (e.g. Shrout & Bolger, 2002) invaluable in supporting this new learning. Nonetheless, I recognise that there is a lot more to learn in relation to this area. I also recognise that, while enabling me to develop skills in quantitative research, this project did not afford opportunities to increase my knowledge of qualitative methodologies. Therefore, I would like to develop skills in utilising qualitative or mixed-methods approaches in the future.

**If you were to do this project again, what would you do differently and why?**

If I were to undertake this project again, there are four key areas I would strive to improve upon. Firstly, as stipulated by the relevant authorities, recruitment to the study was wholly reliant on site advisors, who identified potential participants. While I feel that this was appropriate, given that the clinicians involved were best placed to consider issues related to mental capacity, I was frustrated by the limited information available with regards to the study’s inclusion criteria; with this being available for only the largest of the three sites. At this site, 31 individuals with a diagnosis of ABI were discharged during the 15 month recruitment period. Of these, five were seen to lack capacity to provide consent; four were discharged to another inpatient setting; and one was unable to attend a meeting with me due to a medical appointment. Therefore, 21 participants were recruited from this unit. With hindsight, I would be more proactive in encouraging the advisors at other sites to keep similar records.

Secondly, I feel that the study could have been improved by piloting measures with a larger sample of participants’ with mixed ABI diagnoses, in order to determine
which were most appropriate for the study population. Notably, the Traumatic Brain Injury Self-Efficacy Questionnaire (TBI-SE; Cicerone & Azulay, 2007) was designed to assess self-efficacy following TBI and may not have been as valid and reliable amongst stroke survivors as more generalised measures (e.g. Sherer et al. 1982).

Third, a recognised limitation of the study was the lack of ethnic diversity within the sample. Had more time been available, it may have been helpful to identify and seek access to units in areas where populations were known to be ethnically diverse. This would have potentially enabled greater generalisability of findings.

Lastly, with regard to the write-up of the project, I recognise that Section B necessitated an inclusive approach to ABI; while Section A focused primarily on post-stroke anxiety (PSA). The decision to focus on stroke in Section A was taken in view of the numerous reviews that have been published in relation to anxiety following TBI (e.g. Epstein & Ursano, 2005; Moore, Terryberry-Spohr & Hope, 2006). and in considering the absence of a review of the empirical evidence pertaining to stroke. I also hoped that this strategy would enable Section A and B to exist as stand-alone papers, avoiding repetition yet maintaining continuity between them. I was therefore disappointed to discover, shortly after completing a draft of my report, that an article had just been published, purporting to be the first review of anxiety prevalence post-stroke (Campbell-Burton et al., 2012). Nevertheless, on reading this article I realised that the degree of overlap with my own findings was minimal, as the authors had focused on calculating the pooled estimate of anxiety prevalence (across the reviewed studies) simply noting that: “while there has been a large number of studies investigating and reporting on the frequency of anxiety after stroke, there is scant information about timing of onset, risk factors and outcomes” (p.12). Therefore, I hope that my own findings might make an interesting follow-up to this article.
Clinically, as a result of doing this study, would you do anything differently?

Prior to carrying-out this study, I had little prior experience of engaging with individuals with chronic health conditions (particularly stroke survivors’ and individuals with a diagnosis of moderate to severe TBI). As a result, I have learned a great deal about the difficulties they might face (e.g. physical and communication difficulties) and processes involved in psychological adjustment. I have also gained an appreciation of the complex interplay between mental and physical well-being. I hope to continue to build on these experiences during my future career in this area. In particular, I will seek to actively enquire about how individuals’ feel prior to their discharge from inpatient care, in the hope that potentially anxiety-provoking issues may be elucidated and, where possible, addressed. I was surprised to learn that a number of participants had not disclosed that they were feeling anxious about discharge, to clinicians on the unit. Anecdotal reports suggested that they may have had some concerns about what doing so might mean in terms of their discharge (e.g. whether this might be delayed). Published accounts of stroke survivors’ experiences of discharge from inpatient care (e.g. Ellis-Hill et al., 2009) appeared to echo these sentiments. I would therefore pro-actively seek to address any such concerns and would also seek to increase other clinicians’ awareness and understanding in relation to the assessment of mood-related difficulties within this context.

As a result of carrying-out this project, I have also developed an increased understanding of factors (e.g. self-efficacy and control cognitions) that might play a role in the development of, and potentially help to alleviate, discharge-related anxiety and would seek to use this knowledge to help inform interventions. For example, on hearing about my project, a nurse on my clinical placement suggested that we might invite clients who had previously been discharged from the unit to speak to inpatients about their experiences of this process. Given that vicarious experience
has been shown to influence expectations of personal efficacy (Bandura, 1997),
I encouraged this idea. A member of The Stroke Association subsequently attended
a ward-based group and feedback suggested that this experience was highly valued
by all parties involved. I would therefore promote similar pursuits in the future. I would
also seek to collaborate with staff-members from other professional disciplines (e.g.
physiotherapists and occupational therapists) in the development and evaluation of
interventions aimed at enhancing experiences of personal mastery (Bandura, 1997).

If you were to undertake further research in this area what would that
research project seek to answer and how would you go about doing it?

This was the first study to seek to quantify the prevalence of discharge-related
anxiety in individuals with a diagnosis of ABI and the first to explore relationships
between this phenomenon and psychological constructs (specifically, self-efficacy
and control cognitions). Therefore, in the first instance, I would seek to replicate and
expand on the current findings using a larger and more ethnically diverse sample.
I would also be interested in extending the research, in order to explore other
variables that might have direct, mediating or moderating effects in relation to
discharge-anxiety (e.g. external locus of control or perceptions of social support).

In the absence of a self-report measure that has consistently demonstrated
good psychometric properties in the assessment of anxiety following ABI (Epstein &
Ursano, 2005; Sagen et al., 2009) I would also seek to validate the STAI with these
client groups. However, since the STAI is a relatively lengthy measure (comprising
40 items) and given that, following ABI, individuals with more severe physical and
cognitive difficulties might be more susceptible to fatigue (Lynch et al., 2007) I would
seek to use factor analysis to develop and validate a shorter version of this scale.
Finally, although the current study appeared to substantiate existing qualitative accounts of the emotional challenges involved in moving from inpatient care to the community (see Turner, Fleming, Ownsworth & Cornwell, 2008) findings did not provide insights into the specific nature of individuals’ concerns. Therefore, I would seek to undertake a qualitative investigation, aimed at increasing professionals’ understanding of ‘what people worry about prior to their discharge from inpatient neurorehabilitation following ABI’. This could possibly be accomplished using interpretative phenomenological analysis (IPT; Smith, Flowers, & Larkin, 2009).
References


SECTION D:

APPENDIX OF SUPPORTING MATERIAL
Appendix 1

Literature search strategy

The current review sought to explore the following (a) how PSA has been assessed, in studies to date (i.e. methods and measures that have been employed); (b) the prevalence of PSA; (c) the longitudinal course of PSA; and (d) aetiological features (factors that have been associated with increased anxiety following stroke).

Search databases and parameters

To locate relevant articles, for inclusion in the review, searches of the following electronic databases were undertaken: PsycINFO, Ovid MEDLINE, IngentaConnect, the Current Index to Nursing and Allied Health Literature (CINAHL) and the Cochrane Database of Systematic Reviews. Each of these five databases was searched for the years in which articles were available in electronic format.

A web-based search was also conducted using the engine ‘Google Scholar’. In order to promote compatibility in how study variables were assessed, this search covered studies published in the last 20 years (i.e. January 1992 - October 2012).

Searches were conducted in June 2012 and updated in the second week of October 2012. The following injury-related terms were combined, using the Boolean operator ‘OR’: ‘stroke’, ‘cerebral vascular accident’, ‘CVA’, ‘acquired brain injury’, ‘ABI’. These terms were then combined with the following mood-related terms, using the Boolean operator ‘AND’: ‘mood’, ‘anxiety’, ‘anxious’, ‘distress’, ‘emotion’. All searches were restricted to the English language and duplicates were removed.

2 Indicates the use of truncation that was in keeping with the requirements of the database searched
Inclusion and exclusion criteria

The initial search yielded 41 articles, book chapters and dissertation abstracts. These were subsequently examined to determine if the research that was described met the following inclusion criteria: (a) studies were published in a peer-reviewed scientific journal; (b) studies were published in the English language (since it was not considered feasible or cost-effective to obtain translations of materials); (c) studies involved adult stroke survivors; (d) studies described the measurement of anxiety. Studies that used diagnostic classification systems (e.g. DSM criteria); self-report screening tools; and qualitative descriptions of anxiety, were included.

Studies that were not specific to stroke (e.g. those which described birth trauma or neurodegenerative conditions) were excluded, as were those that did not involve human participants (i.e. those using animals or mechanical simulations). Studies describing mixed samples, including those with other forms of brain injury, were considered for inclusion if analyses were performed according to type of injury.

Following the application of the above criteria, 26 articles emerged as being relevant to the review. References of these articles were manually cross-checked for other relevant papers. This yielded two additional articles that met inclusion criteria. Articles that did not describe empirical studies, but nonetheless made an important contribution to the literature, were used to supplement the review where appropriate.

Figure 1 (overleaf) provides a visual depiction of the number of articles retrieved at each stage of the literature search. In evaluating the findings of the studies described, each was appraised for methodological quality using a framework devised by Sherer et al. (2002). This involved the consideration of several areas, including: study design, sample and analyses. Table 1 (overleaf) summarises the criteria used to appraise the methodological quality of each of the reviewed studies.
Table 1

Criteria used to evaluate the quality of the reviewed studies

<table>
<thead>
<tr>
<th>Quality of methodology criteria</th>
<th>Score (yes = 1, no = 0)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A Prospective or well designed longitudinal study</td>
<td></td>
</tr>
<tr>
<td>B Use of multivariate modelling to examine relationships</td>
<td></td>
</tr>
<tr>
<td>C Clear attempts made to adjust analyses for other potential predictors of outcome (step-wise procedures considered acceptable for this criterion)</td>
<td></td>
</tr>
<tr>
<td>D Characteristics of the study sample and selection criteria were clearly stated.</td>
<td></td>
</tr>
<tr>
<td>E Participants lost at follow-up described and reason for loss to follow-up explained. For cross-sectional studies, a comparison was conducted between those considered eligible and took part and those who did not participate.</td>
<td></td>
</tr>
<tr>
<td>F The final sample size used in analyses comprised ≥10 participants per variable. Alternatively, if an appropriate adjustment was made to control for the high number of predictors relative to sample size, this was acceptable.</td>
<td></td>
</tr>
<tr>
<td>G Participants were representative: sampling based on a multi-centre study or single site with relatively large sample of consecutive admissions / discharges</td>
<td></td>
</tr>
<tr>
<td>H The majority of variables were assessed using standardised measures (for which normative data and reliability / validity analyses had been conducted).</td>
<td></td>
</tr>
<tr>
<td>I Outcome measures were collected independently to assessment of predictors</td>
<td></td>
</tr>
</tbody>
</table>

Total Score: / 9

Subjective quality of ratings:

- 8 – 9  Commendable
- 6 – 7  Acceptable
- 4- 5  Marginal
- < 3  Flawed
Figure 1

Search flow diagram

Step 1: Initial search of databases → 41 articles, book chapters and thesis abstracts identified

Step 2: Review of titles and key words of all sources → 37 articles identified as being relevant to the current review

Step 3: Examination of abstracts of relevant articles → 28 articles identified as being relevant to the current review

Step 4: Full reading of identified articles (in their entirety) → 26 articles found to be relevant to the current review → 2 additional articles identified through searching reference lists

28 articles included in review > 13 of which reported on outcome-based research studies
### Appendix 2

#### Table 2: Overview of empirical studies included in the review

<table>
<thead>
<tr>
<th>Study</th>
<th>Q/Rating</th>
<th>Sample</th>
<th>Design</th>
<th>Measures</th>
<th>Key findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Castillo, Starkstein, Fedoroff, Price &amp; Robinson (1993)</td>
<td>6 / 9 Acceptable</td>
<td>309 patients diagnosed with a cerebral infarction (86.7%) or intra-cerebral haemorrhage (13.3%) and who were admitted to an acute stroke unit in America.</td>
<td>Cross sectional study, including between group comparisons.</td>
<td>Anxiety: The Present State Exam (PSE) was used to assess DSM-III criteria for generalised anxiety disorder (GAD). Stroke was diagnosed using CT and MRI scans. Level of social support: Social Functioning Exam.</td>
<td>26.9% of participants met criteria for GAD. 13.9% of participants were purported to be ‘worried’ but did not meet diagnostic thresholds for GAD. Anxiety was associated with right hemisphere brain lesions, anxiety comorbid with depression was associated with left-hemisphere lesions. ‘Anxious’ and ‘non-anxious’ groups did not differ in terms of demographic characteristics, family history, level of support or functional abilities.</td>
</tr>
<tr>
<td>Castillo, Schultz &amp; Robinson (1995)</td>
<td>8 / 9 Commendable</td>
<td>142 patients with cerebral infarction (89%) intra-cerebral haemorrhage (12%) who were admitted to an acute stroke unit in America and were followed-up once discharged home.</td>
<td>Longitudinal design. Participants were initially assessed on admission to the stroke unit and followed-up at three (n=78), six (n=80), 12 (n= 70) and 24 (n=66) months post-stroke event.</td>
<td>Anxiety: Present State Exam (PSE), was used to determine DSM-III criteria for generalised anxiety disorder (GAD). Stroke was diagnosed by way of CT scans and neurological symptoms. Functional impairment: John Hopkins Inventory. Level of social support: Social Ties Checklist.</td>
<td>Prevalence of GAD on admission: 27%; at six months: 36%; 12 months: 17%; 24 months: 27%. On admission: GAD was associated with impairment in activities of daily living. Those who developed late-onset anxiety were no more functionally impaired than those who did not.</td>
</tr>
<tr>
<td>Schultz, Castillo, Kosier, Robinson (1997) – second paper reporting on above study.</td>
<td>8 / 9 Commendable</td>
<td>142 patients with cerebral infarction (89%) intra-cerebral haemorrhage (12%) who were admitted to an acute stroke unit in America and were followed-up once discharged home.</td>
<td>Longitudinal design. Participants were initially assessed on admission to the stroke unit and followed-up at three (n=78), six (n=80), 12 (n= 70) and 24 (n=66) months post-stroke event.</td>
<td>Anxiety: Present State Exam (PSE), was used to determine DSM-III criteria for generalised anxiety disorder (GAD). Stroke was diagnosed by way of CT scans and neurological symptoms. Functional impairment: John Hopkins Inventory. Level of social support: Social Ties Checklist.</td>
<td>Late-onset post-stroke anxiety occurred in 31% of patients who were not assessed as anxious (did not meet GAD criteria) at initial evaluation. GAD more prevalent amongst younger women.</td>
</tr>
</tbody>
</table>

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3 Quality rating is based on criteria outlined by Sherer et al. (2002).
<table>
<thead>
<tr>
<th>Study</th>
<th>Q/Rating</th>
<th>Sample</th>
<th>Design</th>
<th>Measures</th>
<th>Key findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Burvill et al. (1995)</td>
<td>7 / 9</td>
<td>294 patients with acute stroke or TIA living in Perth, Western Australia [patients were assessed across a range of inpatient and community settings].</td>
<td>Population-based cohort; longitudinal, prospective design. All people with a suspected stroke or TIA, who were resident in Perth, were registered prospectively and followed up at four (n =294) and 12 (n =205) months [post-stroke event].</td>
<td>Anxiety: Psychiatric Assessment Schedule (PAS) was used to assess DSM-III-R criteria (non-hierarchic approach used so that participants were assigned all diagnoses for which they met criteria).</td>
<td>At four month evaluation:  5% of men and 19% of women met criteria for 'any anxiety disorder’. Most ‘anxiety cases’ were of agoraphobia: 4% in men and 17% in women. Anxiety not associated with demographic variables. One-third of the men and half of the women who met criteria for PSA had a pre-stroke history of anxiety</td>
</tr>
<tr>
<td>Åström (1996)</td>
<td>7 / 9</td>
<td>80 patients diagnosed with cerebral infarction (79%), intra-cerebral haemorrhage (5%) or TIA (16%) who were admitted to an acute stroke unit in Sweden (all were admitted from ICU /emergency wards) and were followed-up once discharged [66% of participants returned to their own homes]. 80% of participants survived first stroke.</td>
<td>Population-based cohort, longitudinal, prospective design. All patients admitted to the unit during a one year period were considered for study inclusion. Participants were assessed shortly after admission to and then at three (n=76), 12 (n=70) 24 (n=58), and 36 (n=49) months post discharge.</td>
<td>Anxiety: [Unspecified] psychiatric interviews were used to assess DSM-III-R criteria for GAD and major depressive disorder. Stroke diagnosed by CT scans and neurological sx. Functional impairment: Study designed measure of activities of daily living Level of social support: Study designed measure of psycho-social contact.</td>
<td>Prevalence of GAD on admission was 28%; Prevalence of GAD at three months: 31%; 12 months: 24%; 24 months: 25%; 36 months: 19% GAD not associated with demographic variables. Based on retrospective assessment and informant interviews, none of the participants would have met criteria for GAD prior to the stroke event. GAD comorbid with major depressive disorder was associated with left-hemispheric lesions; whereas GAD alone was more associated with right hemisphere lesions.</td>
</tr>
<tr>
<td>Study</td>
<td>Q/Rating</td>
<td>Sample</td>
<td>Design</td>
<td>Measures</td>
<td>Key findings</td>
</tr>
<tr>
<td>-------------------------------------</td>
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<td>--------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Morrison, Johnstone &amp; MacWalter (2000)</td>
<td>7 / 9</td>
<td>101 patients diagnosed with ‘an acute stroke’, who were admitted to an acute stroke unit in Scotland and followed-up once discharged [discharge location was ‘usually home’]. 68% of the participants had a right hemisphere lesion. 76% had survived a first stroke.</td>
<td>Longitudinal study. All patients admitted to the unit over a 13 month period were first assessed 10-20 days after admittance and followed-up at one (n = 78) and six (n = 71) months post discharge [patients mostly returned to their own homes]. Participants (n=40) were then followed-up at three years.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Acceptable</td>
<td></td>
<td></td>
<td>Anxious and depression: The Hospital Anxiety and Depression Scale (HADS) [with no cut-off score] Neurological impairment; level of functional ability: Neurological Index, Bartel Index, observer ratings. Perceived control was assessed using the Recovery Locus of Control Scale. Study designed scales were used to assess: confidence in recovery and treatment satisfaction</td>
<td></td>
</tr>
<tr>
<td>Morrison, Johnstone, Pollard &amp; MacWalter (2005) – three year follow-up of the above.</td>
<td>7 / 9</td>
<td>73 patients diagnosed with cerebral infarction (79.5%) or cerebral haemorrhage (20.5%) who were admitted to an inpatient unit in New Zealand. 42.5% of participants had left hemisphere damage; 45.6% had right hemisphere damage [no info for the remaining 12.3%].</td>
<td>Cross sectional study. Aimed to assess all new admissions to the rehabilitation unit during a one year period [participants were assessed at three months after the stroke event].</td>
<td></td>
<td>Mean HADS-Anxiety score – at one month was 7.7; at six months: 7.5 Levels of anxiety were higher among women At one month post-stroke, lower internality of control was associated with greater anxiety. Control beliefs did not emerge as predictive in regression analyses. At six months, confidence in recovery explained 25% of the variance in anxiety; satisfaction with treatment explained 6.8% (initial anxiety accounted for 14.5%)</td>
</tr>
<tr>
<td>Barker-Collo (2007)</td>
<td>5 / 9</td>
<td>73 patients diagnosed with cerebral infarction (79.5%) or cerebral haemorrhage (20.5%) who were admitted to an inpatient unit in New Zealand. 42.5% of participants had left hemisphere damage; 45.6% had right hemisphere damage [no info for the remaining 12.3%].</td>
<td>Cross sectional study. Aimed to assess all new admissions to the rehabilitation unit during a one year period [participants were assessed at three months after the stroke event].</td>
<td>Anxiety and depression: Beck Anxiety and Depression Inventories (BAI and BDI) [using cut-offs /descriptive ranges]. Functional impairment: Functional Index Measure Cognitive impairment: Victoria Stroop, Digit and Spatial Span, Paired Associates and California Verbal Learning Tests.</td>
<td>Prevalence of ‘mild anxiety’ was 17.5%; ‘moderate to severe anxiety’ was 21.1%, (with comorbidity ‘moderate to severe depression’ in 12% of cases). Patient gender and hemisphere of lesion explained 12.1% of the variance in anxiety [non-significant] Cognitive performance alone explained 38.5% of the variance in anxiety. Cognitive performance and functional ability together explained 50.7% of the variance in anxiety.</td>
</tr>
</tbody>
</table>
### Studies using self-report measures (continued)

<table>
<thead>
<tr>
<th>Study</th>
<th>Q/Rating</th>
<th>Sample</th>
<th>Design</th>
<th>Measures</th>
<th>Key findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Townend et al. (2007)</td>
<td>6 / 9 Acceptable</td>
<td>125 patients diagnosed with cerebral infarction (96%) or cerebral haemorrhage (4%), admitted to an acute stroke unit in New South Wales, Australia.</td>
<td>Longitudinal design. Participants were assessed within 2-5 days of the stroke event and at one (n=118) and three (n=105) months [while on the unit]</td>
<td>Anxiety and depression: Hospital Anxiety and Depression Scale (HADS) Perceived social support: Multi-dimensional Scale of Perceived Social Support.</td>
<td>Prevalence of anxiety (i.e. HADS-A score ≥ 8) at 2-5 days post the stroke event was 5%; at one month: 8%; and at three months 14%. Anxiety was not associated with any demographic variables (age, gender, ethnicity or marital status). Anxiety was not associated with perceived support.</td>
</tr>
<tr>
<td>De Wit et al. (2008)</td>
<td>8 / 9 Commendable</td>
<td>532 patients diagnosed with a first ever stroke were recruited from four rehabilitation centres (in Belgium, Scotland, Switzerland, Germany) and followed up once discharged [discharge locations unspecified].</td>
<td>Longitudinal study Participants were assessed at two (n =491), four (n = 464) and six (n =426) months post-discharge</td>
<td>Anxiety and depression screen: Hospital Anxiety and Depression Scale with cut-off scores of ≥8. Stroke was diagnosed using CT scans and neurological symptoms (laterality of hemiplegia)</td>
<td>Prevalence of anxiety (i.e. HADS-A ≥8) at two months: 25%; four months: 22%; six months: 22% The severity of anxiety (i.e. mean HADS scores) decreased between four and six months 11% of those initially not anxious became anxious at the four month evaluation (four months post-stroke).</td>
</tr>
<tr>
<td>Bergersen, Frøslie, Sunnerhagen &amp; Schanke (2010)</td>
<td>4 / 9 Marginal</td>
<td>162 patients diagnosed with cerebral infarction (86%) or cerebral haemorrhage (14%), who were discharged from an inpatient centre in Norway.</td>
<td>Cross sectional study. Patients were mailed questionnaires (all had been discharged between two - five years previously)</td>
<td>Anxiety and depression: Hospital Anxiety and Depression Scale (HADS) A study measure asked about periods of low mood and / or anxiety symptoms following discharge</td>
<td>Mean HADS-Anxiety subscale score was 5.8 The prevalence of anxiety (HADS-A &gt; 7) was 36.4% (17.3% of 'anxious' respondents reported comorbid depression; HADS-D &gt; 7). 54.5% of respondents self-reported having been through periods of anxiety following discharge.</td>
</tr>
<tr>
<td>Study</td>
<td>Q/Rating</td>
<td>Sample</td>
<td>Design</td>
<td>Measures</td>
<td>Key findings</td>
</tr>
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</tr>
<tr>
<td>Sagen et al. (2009)</td>
<td>8 / 9 Commendable</td>
<td>184 patients diagnosed with cerebral infarction (93%) or cerebral haemorrhage (7%), admitted to an acute stroke unit in Norway and followed-up once discharged [discharge location unspecified].</td>
<td>Longitudinal design. Aimed to assess all new / consecutive admissions to the stroke unit, during a 2.5 year period. Participants were initially assessed while on the unit and followed-up four months after discharge (n=104).</td>
<td>Anxiety: Structured Clinical Interview (SCID) for DSM-IV was used to assess for ‘any anxiety disorder’. Comorbidity: Hospital Anxiety and Depression Scale, Apathy Evaluation Scale, Montgomery and Asberg Depression Scale. Functional impairment: The Barthel Index and Scandinavian Stroke Scale</td>
<td>On initial assessment: The SCID interview revealed a prevalence rate of 23.1% for ‘any anxiety diagnosis’. The HADS demonstrated low sensitivity but high specificity for anxiety among the study participants. On the basis of the above findings, lower cut-off scores of ≥4 for HADS among stroke survivors was recommended.</td>
</tr>
<tr>
<td>Sagen et al. (2010) – second paper reporting on findings from the above study.</td>
<td>8 / 9 Commendable</td>
<td>82% of participants had survived a first stroke event, the remainder had a recurrent stroke.</td>
<td></td>
<td></td>
<td>At four month follow-up: 14% of participants had co-morbid anxiety and depression (HADS-A ≥ 8). Anxiety unrelated to physical or functional abilities.</td>
</tr>
</tbody>
</table>
Table 3: Prevalence of post-stroke anxiety (PSA) in the reviewed studies

<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Setting</th>
<th>Assessment of anxiety</th>
<th>Assessment points and prevalence of anxiety</th>
</tr>
</thead>
</table>
| Castillo, Starkstein, Fedoroff, Price & Robinson (1993) | Baltimore, America | All assessments took place on an acute stroke unit                       | Present State Exam (PSE) to assess [modified] DSM-III criteria for Generalised Anxiety Disorder (GAD) | Time since the stroke event: Mean 11.2 days (SD 9.3)  
  - assessed as reasonably 'worried': 13.9%  
  - meeting [modified] criteria for GAD: 27% |
| Castillo, Schultz & Robinson (1995)   | Baltimore, America | Participants were initially assessed on an acute stroke unit and followed-up after discharge (discharge locations not specified). | PSE to assess [modified] DSM-III-R criteria for GAD                                     | Meeting [modified] DSM-III criteria for GAD  
  - on admission to the unit: 27%  
  - at three months post-stroke: 27%  
  - at six months post-stroke: 36%  
  - at 12 months post-stroke: 17%  
  - at 24 months post-stroke: 27% |
| Schultz, Castillo, Kosier & Robinson (1997) | Umeå, Sweden     | Participants were initially assessed while resident on an acute stroke unit and followed-up after discharge from the unit | [Unspecified] psychiatric interviews to evaluate [modified] DSM-III-R diagnostic criteria for GAD | Meeting [modified] DSM-III-R criteria for GAD  
  - on admission to the unit: 28%  
  - at three months post-discharge: 31%  
  - at 12 months post-discharge: 24%  
  - at 24 months post-discharge: 25% |
| Åström (1996)                         |                  | Participants were recruited from a range of inpatient and community settings | Structured Clinical Interview for DSM-III-R (SCID) to assess ‘any anxiety disorder’      | Meeting criteria for ‘any anxiety disorder’  
  - at four months post-stroke: 24%  
  (agoraphobia: 21%and GAD: 3%) |
| Burvill et al. (1995)                 | Perth, Australia | Participants were assessed while on an acute stroke unit and followed-up four months after discharge | Compared the Hospital Anxiety and Depression Scale (HADS) to a Structured Clinical Interview for DSM-IV | Meeting criteria for ‘any anxiety disorder’  
  - on admission to the unit: 23% |
<p>| Sagen et al. (2009, 2010)             | Skien, Norway    | Participants were assessed while on an acute stroke unit and followed-up for four months after discharge |                                                                                       |                                                                                                             |</p>
<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Setting</th>
<th>Assessment of anxiety</th>
<th>Assessment points and prevalence of anxiety</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morrison, Johnstone &amp; MacWalter (2000)</td>
<td>Dundee, Scotland</td>
<td>Initial assessments took place on an acute stroke unit. Participants were later followed-up after discharge from the unit</td>
<td>The Hospital Anxiety and Depression Scale (HADS) (did not use cut-off scores).</td>
<td>Average HADS-Anxiety score &lt;br&gt; - on admission to the unit: 7.7 &lt;br&gt; - at one month post-discharge: 7.5 &lt;br&gt; - at six months post-discharge: 7.5</td>
</tr>
<tr>
<td>Morrison, Johnstone, Pollard &amp; MacWalter (2005)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Barker-Collo (2007)</td>
<td>Auckland, New Zealand</td>
<td>All assessments took place while participants were resident on a post acute rehabilitation unit</td>
<td>The Beck Anxiety Inventory (BAI) using cut-off scores to depict qualitative ranges.</td>
<td>BAI range, at three months post-stroke &lt;br&gt; - 'mild' anxiety symptoms: 18% &lt;br&gt; - 'moderate to severe' anxiety: 21%</td>
</tr>
<tr>
<td>Townend et al. (2007)</td>
<td>New South Wales, Australia</td>
<td>All assessments took place while participants were resident / inpatient on an acute stroke unit.</td>
<td>The Hospital Anxiety and Depression Scale (HADS) (with cut-off scores of ≥ 8).</td>
<td>HADS-Anxiety score of ≥ 8 &lt;br&gt; - at 2-5 days post-stroke: 5% &lt;br&gt; - at one month post-stroke: 8% &lt;br&gt; - at three months post-stroke: 14%</td>
</tr>
<tr>
<td>De Wit et al. (2008)</td>
<td>Belgium, Scotland, Switzerland and Germany</td>
<td>Study participants were followed-up on discharge from rehabilitation units [Average length of stay on the unit was 48 days]</td>
<td>The Hospital Anxiety and Depression Scale (HADS) (with cut-off scores of ≥ 8).</td>
<td>HADS-Anxiety score of ≥ 8 &lt;br&gt; - at four months post-stroke: 22% &lt;br&gt; - at six months post-stroke: 22%</td>
</tr>
<tr>
<td>Bergersen, Frøslie, Sunnerhagen &amp; Schanke (2010)</td>
<td>Oslo, Norway</td>
<td>Participants were mailed assessment measures two to five years after discharge from a post-acute rehabilitation unit.</td>
<td>The Hospital Anxiety and Depression Scale (HADS) (with cut-off scores of &gt;7).</td>
<td>Average HADS-Anxiety score &lt;br&gt; - two to five years post-stroke: 5.8 &lt;br&gt; HADS-Anxiety score of &gt;7 &lt;br&gt; - two to five years post-stroke: 36%</td>
</tr>
</tbody>
</table>
Appendix 4

Diagnostic Criteria for Generalised Anxiety Disorder (GAD)

A. At least 6 months of "excessive anxiety and worry" about a variety of events and situations. Generally, "excessive" can be interpreted as more than would be expected for a particular situation or event. Most people become anxious over certain things, but the intensity of the anxiety typically corresponds to the situation.

B. There is significant difficulty in controlling the anxiety and worry. If someone has a very difficult struggle to regain control, relax, or cope with the anxiety and worry, then this requirement is met.

C. The presence for most days over the previous six months of 3 or more of the following symptoms:
   1. Feeling wound-up, tense, or restless
   2. Easily becoming fatigued or worn-out
   3. Concentration problems
   4. Irritability
   5. Significant tension in muscles
   6. Difficulty with sleep

D. The symptoms are not part of another mental disorder.

E. The symptoms cause "clinically significant distress" or problems functioning in daily life. "Clinically significant" is the part that relies on the perspective of the treatment provider. Some people can have many of the aforementioned symptoms and cope with them well enough to maintain a high level of functioning.

F. The condition is not due to a substance or medical issue

Appendix 5

Approval letters from Research Ethics Committee

*This information has been removed from the electronic version*
Appendix 6

Permissions from Research and Development Departments

This information has been removed from the electronic version
Appendix 7

Research Participant Information Sheet and Consent Form

*This information has been removed from the electronic version*
Appendix 8

Study Measures

*This information has been removed from the electronic version*
Appendix 9

Permissions to use Discharge-Anxiety and TBI Self-Efficacy Scales

*This information has been removed from the electronic version*
Appendix 10

DEMOGRAPHIC AND CLINICAL INFORMATION

Age

_______ years _______ months

Gender

Male              Female              Not stated

Ethnicity

White British              Black Caribbean
White Other              Black African
Asian              Pakistani
Indian              Bangladeshi
Other (specify): __________________

Type of Injury

Stroke (Ischemic)
Stroke (haemorrhagic)
Trauma (e.g. accident or blow)
Anoxia / hypoxia (lack of oxygen)

Other not listed (specify): __________________

Brain area affected

Right hemisphere              Left hemisphere
Diffuse damage              Unknown

Severity of Injury

<table>
<thead>
<tr>
<th></th>
<th>GCS</th>
<th>PTA</th>
<th>LOC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate</td>
<td>9–12</td>
<td>&gt;1 to &lt;7 days</td>
<td>&gt;30 min to &lt; 24 hours</td>
</tr>
<tr>
<td>Severe</td>
<td>3–8</td>
<td>&gt;7 days</td>
<td>&gt;24 hours</td>
</tr>
</tbody>
</table>

Admitted from

home     acute unit     post-acute

Comorbid diagnoses

______________________________

Interventions re mood

(medication, therapy)

______________________________

Previous admissions

___________________________ (number)

Time since event

___________________________ (months)

Current admission

___________________________ (days)

Time until discharge

___________________________ (days)

Version 1.2.
### Appendix 11

**Table 4**

Characteristics of the study sample ($N = 42$)

<table>
<thead>
<tr>
<th></th>
<th>$n$ (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender</strong></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>31 (74%)</td>
</tr>
<tr>
<td>Female</td>
<td>11 (26%)</td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
</tr>
<tr>
<td>White British or Irish</td>
<td>31 (74%)</td>
</tr>
<tr>
<td>Black British</td>
<td>3 ( 7%)</td>
</tr>
<tr>
<td>Afro-Caribbean</td>
<td>2 ( 5%)</td>
</tr>
<tr>
<td>Other BME group$^a$</td>
<td>6 (14%)</td>
</tr>
<tr>
<td><strong>ABI diagnosis</strong></td>
<td></td>
</tr>
<tr>
<td>Stroke (Ischemic or Haemorrhagic)</td>
<td>22 (52%)</td>
</tr>
<tr>
<td>Traumatic Brain Injury (TBI)</td>
<td>18 (48%)</td>
</tr>
<tr>
<td>Anoxic Brain Damage</td>
<td>2 ( 5%)</td>
</tr>
<tr>
<td><strong>Brain area affected</strong></td>
<td></td>
</tr>
<tr>
<td>Left hemisphere</td>
<td>10 (24%)</td>
</tr>
<tr>
<td>Right hemisphere</td>
<td>20 (47%)</td>
</tr>
<tr>
<td>Diffuse damage</td>
<td>4 (10%)</td>
</tr>
<tr>
<td>Not recorded</td>
<td>7 (19%)</td>
</tr>
<tr>
<td><strong>Admitted from</strong></td>
<td></td>
</tr>
<tr>
<td>ICU / acute unit</td>
<td>37 (88%)</td>
</tr>
<tr>
<td>Post-acute unit</td>
<td>5 (12%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Range</th>
<th>$M$</th>
<th>$SD$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of participant (years)</td>
<td>19 – 74</td>
<td>44.74</td>
<td>13.35</td>
</tr>
<tr>
<td>Time since injury (months)</td>
<td>1 – 12</td>
<td>4.52</td>
<td>2.59</td>
</tr>
<tr>
<td>Length of admission (days)</td>
<td>27 – 240</td>
<td>82.29</td>
<td>49.75</td>
</tr>
<tr>
<td>Time until discharge (days)</td>
<td>0 – 14</td>
<td>4.24</td>
<td>3.87</td>
</tr>
</tbody>
</table>

$^a$Black and minority ethnic (BME) groups included those of Indian, South Asian and mixed heritage
Appendix 12

Histograms showing distributions of data

Demographic and clinical variables

Mean = 44.74
Std. Dev. = 13.351
N = 42

Mean = 4.52
Std. Dev. = 2.597
N = 42

Mean = 92.39
Std. Dev. = 48.746
N = 42
Scores on self-report measures

Number of days until discharge

Mean = 4.24
Std. Dev. = 3.039
N = 42

Adapted-PAQ Score

Mean = 18.12
Std. Dev. = 8.27
N = 42

STAI-State Score

Mean = 42.4
Std. Dev. = 14.68
N = 42
### Appendix 13

**Table 6**  
Test statistics (Shapiro-Wilk) for untransformed continuous variables

<table>
<thead>
<tr>
<th>Variable / Measure</th>
<th>Test statistic</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of participant (years)</td>
<td>0.97</td>
<td>0.44</td>
</tr>
<tr>
<td>Time since ABI (months)</td>
<td>0.89</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td>Length of admission (days)</td>
<td>0.83</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td>Time until discharge (days)</td>
<td>0.83</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td>Discharge-anxiety (Adapted-PAQ)</td>
<td>0.95</td>
<td>0.05*</td>
</tr>
<tr>
<td>State-Anxiety (STAI-State)</td>
<td>0.96</td>
<td>0.15</td>
</tr>
<tr>
<td>Trait-Anxiety (STAI-Trait)</td>
<td>0.96</td>
<td>0.04*</td>
</tr>
<tr>
<td>Internal control (MHLC-Internal)</td>
<td>0.99</td>
<td>0.96</td>
</tr>
<tr>
<td>Self-Efficacy (TBI-SE)</td>
<td>0.95</td>
<td>0.07</td>
</tr>
</tbody>
</table>

* A p-value of ≤ 0.05 rejects the null hypothesis that data follows the normal distribution

**Table 7**  
Test statistics (Shapiro-Wilk) after square-root transformations of variables

<table>
<thead>
<tr>
<th>Transformed Variable / Measure</th>
<th>Test statistic</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Discharge-anxiety (Adapted-PAQ)</td>
<td>0.97</td>
<td>0.23</td>
</tr>
<tr>
<td>Trait-Anxiety (STAI-Trait)</td>
<td>0.97</td>
<td>0.37</td>
</tr>
<tr>
<td>Time since ABI (months)</td>
<td>0.95</td>
<td>0.05*</td>
</tr>
<tr>
<td>Length of admission (days)</td>
<td>0.92</td>
<td>&lt; 0.01*</td>
</tr>
<tr>
<td>Time until discharge (days)</td>
<td>0.92</td>
<td>&lt; 0.01*</td>
</tr>
</tbody>
</table>

* Data for clinical variables (time since injury, length of admission and time to discharge) continued to violate assumptions of normality following square-root transformations.
## Appendix 14

### Table 8

Summary of scores obtained on self-report measures \((N = 42)\)

<table>
<thead>
<tr>
<th>Measure</th>
<th>Possible range</th>
<th>Observed range</th>
<th>(M)</th>
<th>(SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adapted-PAQ</td>
<td>6 - 42</td>
<td>6 - 36</td>
<td>8.12</td>
<td>8.27</td>
</tr>
<tr>
<td>STAI: State-Anxiety</td>
<td>20 - 80</td>
<td>20 - 79</td>
<td>42.40</td>
<td>14.68</td>
</tr>
<tr>
<td>STAI: Trait-Anxiety</td>
<td>20 - 80</td>
<td>20 - 68</td>
<td>37.29</td>
<td>10.52</td>
</tr>
<tr>
<td>MHLC: Internal</td>
<td>6 - 36</td>
<td>9 - 35</td>
<td>23.05</td>
<td>5.88</td>
</tr>
<tr>
<td>TBI Self-Efficacy</td>
<td>13 - 130</td>
<td>51 – 128</td>
<td>91.93</td>
<td>22.18</td>
</tr>
</tbody>
</table>
Appendix 15

Mean differences in discharge-anxiety: high versus low internal locus of control groups (figure 2) and high versus low self-efficacy groups (figure 3)

Figure 2. Mean differences in discharge-anxiety for high vs. low internal locus of control groups.

Figure 3. Mean differences in discharge-anxiety for high vs. low self-efficacy groups.
Appendix 16

Figures depicting tests of indirect effects: mediational analyses

Figure 4

*Internal health control as a mediator of the relationship between self-efficacy and discharge-anxiety*

**Direct effect**

\[ \text{Self-efficacy (X)} \rightarrow \text{Internal locus of control (M)} \rightarrow \text{Discharge-anxiety (Y)} \]

**Indirect effect**

\[ \text{Self-efficacy (X)} \rightarrow \text{Internal locus of control (M)} \rightarrow \text{Discharge-anxiety (Y)} \]

Figure 5

*Self-efficacy as a mediator of the relationship between internal health control and discharge-anxiety*

**Direct effect**

\[ \text{Internal locus of control (X)} \rightarrow \text{Discharge-anxiety (Y)} \]

**Indirect effect**

\[ \text{Internal locus of control (X)} \rightarrow \text{Self-efficacy (M)} \rightarrow \text{Discharge-anxiety (Y)} \]
Appendix 17

End of Study Reports to Ethics Committee and Trust Departments

Department of Applied Psychology
David Salomons Estate
Broomhill Road
Southborough
Tunbridge Wells
Kent, TN3 0TG

Research Ethics Committee Offices
Room 4W/12, 4th Floor
Charing Cross Hospital
Fulham Palace Road
London, W6 8RF

10 August 2013

Dear Ms Marshall,

REC reference number: 12/LO/0349
Study Title: Anxiety related to discharge from inpatient neurorehabilitation

Please find enclosed end of study report for the abovementioned project.

This study was reviewed by the South East Coast – Surrey REC on 6 March 2012. After receiving confirmation that approval conditions, as suggested by the committee, had been satisfactorily adhered to, the study formally commenced on 24 April 2012. R&D approval from all three study sites was obtained, without further amendments to the study protocol being made and data collection progressed smoothly with no ethical issues or concerns raised. The study concluded on 10 July 2013. I hope that the committee will find the enclosed report, detailing the findings of the research to be of interest. Should you have any other queries, I can be contacted on 078 037 76861.

Yours sincerely,

Michelle Genis
Trainee Clinical Psychologist
Canterbury Christ Church University
# DECLARATION OF THE END OF A STUDY

(For all studies except clinical trials of investigational medicinal products)

To be completed in typescript by the Chief Investigator and submitted to the Research Ethics Committee that gave a favourable opinion of the research ("the main REC") within 90 days of the conclusion of the study or within 15 days of early termination. For questions with Yes/No options please indicate answer in bold type.

## 1. Details of Chief Investigator

| Name: | [Redacted] |
| Address: | [Redacted] |
| Telephone: | [Redacted] |
| Email: | [Redacted] |
| Fax: | [Redacted] |

## 2. Details of study

| Full title of study: | Anxiety related to discharge from inpatient neurorehabilitation: Exploring the role of self-efficacy and locus of control beliefs |
| Research sponsor: | Canterbury Christ Church University |
| Name of main REC: | [Redacted] |
| Main REC reference number: | [Redacted] |

## 3. Study duration

| Date study commenced: | 10 April 2012 |
| Date study ended: | 10 July 2013 |
| Did this study terminate prematurely? | Yes / No |

*If yes please complete sections 4, 5 & 6, if no please go direct to section 7.*
4. **Circumstances of early termination**

<table>
<thead>
<tr>
<th>What is the justification for this early termination?</th>
</tr>
</thead>
</table>

5. **Temporary halt**

<table>
<thead>
<tr>
<th>Is this a temporary halt to the study?</th>
<th>Yes / No</th>
</tr>
</thead>
<tbody>
<tr>
<td>If yes, what is the justification for temporarily halting the study? When do you expect the study to re-start?</td>
<td>e.g. Safety, difficulties recruiting participants, trial has not commenced, other reasons.</td>
</tr>
</tbody>
</table>

6. **Potential implications for research participants**

<table>
<thead>
<tr>
<th>Are there any potential implications for research participants as a result of terminating/halting the study prematurely? Please describe the steps taken to address them.</th>
</tr>
</thead>
</table>

7. **Final report on the research**

<table>
<thead>
<tr>
<th>Is a summary of the final report on the research enclosed with this form?</th>
<th>Yes / No</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>If no, please forward within 12 months of the end of the study.</em></td>
<td></td>
</tr>
</tbody>
</table>

8. **Declaration**

<table>
<thead>
<tr>
<th>Signature of Chief Investigator:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Print name:</td>
</tr>
<tr>
<td>Date of submission:</td>
</tr>
</tbody>
</table>
Anxiety Related to Discharge from Inpatient Neurorehabilitation

Objectives
This study aimed to determine the prevalence of anxiety specifically related to discharge in a group of 42 individuals who had sustained moderate to severe acquired brain injury (ABI) and who were imminently due to return home following a period of inpatient neurorehabilitation. The study also aimed to explore differential relationships between psychological factors (self-efficacy and health control beliefs) alongside the relative influence of demographic (age, gender and ethnicity) and clinical (medical diagnosis and injury location) characteristics, on discharge-anxiety.

Hypotheses
Based on past research in the area and in view of relevant conceptual models, it was hypothesised that: (1) individuals with ABI would report higher levels of discharge-anxiety (or state-anxiety) than generalised anxiety (i.e. trait-anxiety); (2) there would be significant differential relationships between (a) self-efficacy and (b) internal health control beliefs and discharge-anxiety; and (3) self-efficacy and health control beliefs would interact with each-other in influencing discharge-anxiety.

Design
A cross-sectional, single-group design was employed, wherein correlational and multivariate analyses were used to explore relationships between variables. Data was obtained via self-report tools and reviews of participants’ medical files.

Setting and inclusion criteria
Prior to the initiation of the study, authorisation was obtained from the NHS Research and Development (R&D) Departments at each of the study sites. Recruitment took place across three post-acute neurorehabilitation units in the South East of England, over a 15 month period (from March 2012 to June 2013). During this period, individuals were invited to take part in the research if they: (a) were aged 18 or over; (b) were inpatient; (c) had a diagnosis of ABI (as recorded in their medical file); (d) had capacity to provide consent; (e) had adequate English language comprehension skills; (f) were able to effectively communicate answers to self-report scales; (g) were due to be discharged from the unit (and had been informed of their discharge date); and (h) were due to return home upon discharge.
Procedure
The method of recruitment at each of the study sites was based on guidance from the R&D Departments. At each unit, the study was introduced to individuals who met inclusion criteria by a local site advisor (in each instance, a qualified clinical psychologist who was familiar with the study). To minimise potential for distress, only those who were aware of their discharge date were approached. During this approach, individuals were given a brief verbal explanation of the study, along with a participant information sheet and consent form. Accordingly, individuals’ capacity to provide consent to participate in the research was appraised by site advisors in the first instance. Once verbal consent had been obtained, a meeting with the researcher was arranged. This meeting was scheduled in advance, so as not to conflict with therapeutic activities. During the meeting participants were given a detailed explanation of the study and encouraged to ask questions. Written consent was then obtained, following which four self-report measures were administered by the researcher. This was followed by a verbal debrief. No participant reported any concerns with regard to their participation in the study and no risk issues were noted.

Measures
The following self-report measures were verbally administered by the researcher:
- **State-Trait Anxiety Inventory; Form Y** (STAI; Spielberger, 1983)
- **Patient Anxieties Questionnaire** (PAQ; Main & Gudjonsson, 2005)
- **Multidimensional Health Locus of Control Scale** (Wallston et al., 1994)
- **Traumatic Brain Injury Self-Efficacy Questionnaire** (Cicerone & Azulay, 2007)

Demographic data on age, gender and ethnicity was recorded for each participant and (with their explicit consent) the following clinical information was obtained from their medical files: ABI diagnosis; severity of ABI; injury localisation (left hemisphere, right hemisphere or diffuse damage); and information related to their inpatient stay (number of prior admissions, length of current admission and time until discharge).

Findings
While few participants (14%) reported markedly elevated trait-anxiety almost half (45%) of the sample reported levels of transient, state-anxiety which could be considered to be clinically significant. Notably, state-anxiety (appraised via the STAI) was strongly associated with discharge-anxiety (appraised via the adapted-PAQ).
Factors associated with discharge-anxiety

There was a small but significant positive correlation between age and discharge-related anxiety ($r = 0.31$, $p = 0.04$) and a small but significant negative correlation between internal health control beliefs and discharge-anxiety ($r = -0.37$, $p = 0.02$). There was a large negative correlation between self-efficacy beliefs and discharge-related anxiety ($r = -0.84$, $p < 0.001$). Notably, no other demographic or clinical variables correlated with discharge-anxiety at a statistically significant level.

Three variables (age, control beliefs and self-efficacy) were included in a hierarchical regression model (with discharge-anxiety as the criterion variable). At step one of the model, age alone accounted for 10% of the variance in discharge-anxiety. At the second step, internal control beliefs were included. This model was again significant, with internal control beliefs explaining a further 12% of the variance in discharge-anxiety when age was already taken into account. At the third step, self-efficacy was added to the model. This overall model (with all three predictors) was also significant and accounted for 71% of the variance in discharge-anxiety. However, at this third and final step, age and control beliefs did not contribute significantly to the model.

Conclusions

Findings of the current study suggest that: (a) anxiety is prevalent in the lead-up to discharge from inpatient neurorehabilitation following ABI and (b) age, internal health control and self-efficacy beliefs may play an important role in influencing discharge-anxiety. These findings are consistent with theoretical models of emotional adjustment following ABI, as well as existing empirical evidence relating to anxiety in the context of chronic health conditions. They also supplement published anecdotal accounts, which suggest that adjusting emotionally to transitions from inpatient care to the community and home-life poses a significant challenge for individuals with ABI.

Findings did not support relationships between gender, ethnicity or clinical factors examined and discharge-anxiety. Although the cross-sectional nature of the research precludes inferences about direction of causality, findings nonetheless provide some evidence to suggest that psychological factors (self-efficacy and control beliefs) may exert an influence on discharge-anxiety and that these factors may be important in developing interventions aimed at addressing this phenomenon. However, in view of the limitations of the current study and the early stages of related research, further investigations aimed at replicating and expanding on current findings, are warranted.
Appendix 18

Journal Submission Guidelines: Neuropsychological Rehabilitation

Neuropsychological Rehabilitation considers all manuscripts on the strict condition that they have been submitted only to Neuropsychological Rehabilitation, that they have not been published already, nor are they under consideration for publication elsewhere. Authors who fail to adhere to this will be charged all costs that Neuropsychological Rehabilitation incurs and their papers will not be published. Contributions to Neuropsychological Rehabilitation must report original research and will be subjected to review by referees at the discretion of the Editorial Office.

Manuscript preparation

See the APA Publication Manual (6th Ed.) for specific style guidelines

- Papers are accepted only in English. British English spelling and punctuation is preferred. Any consistent spelling style may be used. Please use double quotation marks, except where “a quotation is ‘within’ a quotation”.

- There is no word limit for manuscripts submitted to this journal. Authors should include a word count with their manuscript.

- Manuscripts should be compiled in the following order: title page; abstract; keywords; main text; acknowledgments; appendixes (as appropriate); references; table(s) (on individual pages); figure caption(s) (as a list).

- Abstracts of 150-200 words are required for all papers submitted. Avoid abbreviations, diagrams, and references to the text in the abstract.

- Each paper should have 5 keywords.
• All the authors of a paper should include their full names, affiliations, postal addresses, telephone numbers and email addresses on the cover page of the manuscript. One author should be identified as the corresponding author. The affiliations of all co-authors should be the affiliation where the research was conducted. If any of the named co-authors moves affiliation during the peer review process, the new affiliation can be given as a footnote. Please note that no changes to affiliation can be made after the article is accepted.

• Please note that the email address of the corresponding author will be displayed in the article PDF (depending on style) and the online article.

• Biographical notes on contributors are not required for this journal.

• For all manuscripts non-discriminatory language is mandatory.

• Authors must adhere to SI units. Units are not italicised.

• When using a word which is or is asserted to be a proprietary term or trade mark, authors must use the symbol ® or TM.

• Authors should supply a shortened version of the title suitable for the running head, not exceeding 50 character spaces. Section headings should be concise and should not contain numbering.

• Acknowledgements should be gathered into a brief statement at the end of the text. All sources of financial sponsorship are to be acknowledged, including the names of private and public sector sponsors. This includes government grants, corporate funding, trade associations and contracts.

• Tables should be kept to the minimum. Each table should be typed double spaced on a separate page, giving the heading, e.g., "Table 2", in Arabic numerals, followed by the legend, followed by the table.
Results of statistical tests should be given in the following form:

"... results showed an effect of group, \( F(2, 21) = 13.74, MSE = 451.98, p < .001, \) but there was no effect of repeated trials, \( F(5, 105) = 1.44, MSE = 17.70, \) and no interaction, \( F(10, 105) = 1.34, MSE = 17.70.\)"

Abbreviations that are specific to a particular manuscript or to a very specific area of research should be avoided, and authors will be asked to spell out in full any such abbreviations throughout the text. Standard abbreviations such as RT for reaction time, SOA for stimulus onset asynchrony or other standard abbreviations that will be understood by readers of the journal are acceptable.

Experimental conditions should be named in full, except in tables and figures.

**Figures**

- It is in the author's interest to provide the highest quality format possible. Please be sure that all imported material is scanned at the appropriate resolution: 1200 dpi for line art, 600 dpi for grayscale and 300 dpi for colour.
- Figures must be saved separate to text.
  
  Please do not embed figures in the paper file.
- Files should be saved as one of the following formats: TIFF (tagged image file format), PostScript or EPS (encapsulated PostScript), and should contain all the necessary font information and the source file of the application (e.g. CorelDraw/Mac, CorelDraw/PC).
- All figures must be numbered in the order in which they appear in the paper (e.g. Figure 1, Figure 2). In multi-part figures, each part should be labelled (e.g. Figure 1(a), Figure 1(b)).
- Figure captions must be saved separately, as part of the file containing the complete text of the paper, and numbered correspondingly.

- The filename for a graphic should be descriptive of the graphic, e.g. Figure1

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Once logged on to the site, submissions should be made via the Author Centre. Online user guides and access to a helpdesk are available on this website.

Manuscripts may be submitted in any standard format, including Word, EndNote and PDF. These files will be automatically converted into a PDF file for the review process. LaTeX files should be converted to PDF prior to submission because ScholarOne Manuscripts is not able to convert LaTeX files into PDFs directly.

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