AN INVESTIGATION INTO PREDICTIVE FACTORS OF POST-TRAUMATIC STRESS DISORDER FOLLOWING ACUTE MEDICAL TRAUMA

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ABSTRACT

Early investigations into responses to traumatic events tended to focus upon large-scale external traumatic events such as wars and natural disasters. This has resulted in traumatic responses being codified into the diagnostic syndrome of post-traumatic stress disorder (PTSD). Research that is more recent has addressed traumatic responses to smaller events such as the onset of medical conditions. Research has indicated several factors that are associated with the development and maintenance of the symptoms of PTSD.

The current study aimed to investigate the role of illness representations, social support, and thought control strategies in predicting PTSD symptoms in myocardial infarction and subarachnoid haemorrhage samples. The study also explored changes in PTSD symptoms and illness representations over time. In a prospective study, individuals who had experienced a myocardial infarction or subarachnoid haemorrhage completed measures of PTSD, illness representations, social support and thought control strategies at three time points over a three-month period. Statistical analysis demonstrated that the illness representations of emotional representation, illness coherence, consequences, and treatment control and the thought control strategy of worry were found to be significant predictors of PTSD symptomatology. The hypothesised variable of social support was not found to be a significant predictor of PTSD symptomatology.

Possible reasons for the research findings are discussed together with the implications of the findings for clinical practice and future research.

DECLARATION

No portion of the work referred to in this thesis has been submitted in support of an application for another degree or qualification of this or any other University or other institute of learning.

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STATEMENT OF CANDIDATE'S ACADEMIC TRAINING

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CHAPTER 1: INTRODUCTION

1. Introduction

1.1. Overview of Chapter One

This study aims to investigate the prevalence and predictors of Post-traumatic Stress Disorder (PTSD) following the traumatic medical events of subarachnoid haemorrhage and myocardial infarction. The study focuses on factors that have been indicated as significant in the development and maintenance of PTSD and emotional distress generally following traumatic events. In particular, the study examines the role of illness representations, social support, and thought control strategies in PTSD and emotional distress subsequent to acute medical trauma.

The introduction aims to locate the study within the context of prior research and the wider knowledge base pertaining to illness representations and PTSD. It initially describes key areas in the psychology of physical illness processes, the medical phenomena of subarachnoid haemorrhage and myocardial infarction and research relating to the experience of emotional distress following these medical conditions. It will then proceed to describe the development of the concept of PTSD, the current definition of this condition and clinical areas in which the concept has been applied. The introduction then outlines several models that aim to explain the development and maintenance of PTSD and consequent treatment approaches. Finally, the rationale, aims and hypotheses of the study will be presented.

1.2. Psychological aspects of physical illness and traumatic medical events

Clinical Health Psychology has developed many concepts and theories regarding the role of psychological factors in illness, health behaviour, and recovery processes. In their seminal work in 1959, Friedman and Rosenman demonstrated the effect of psychological factors upon physical illness processes through research into behaviour patterns and risk of myocardial infarction (heart attack). They identified a particular behaviour pattern ('Type A behaviour') that was associated with an increased risk of myocardial infarction. Type A behaviour was defined as:

> 'an action-emotion complex that can be observed in any person who is aggressively involved in a chronic, incessant struggle to achieve more and more in less and less time, and, if required to do so, against the opposing efforts of other things or persons...'
> (Friedman and Rosenman, 1959)

Thus, their research differentiated emotional and behavioural factors and described their impact upon physical disease processes. Although later studies have failed to demonstrate an association between Type A behaviour and clinical coronary disease (for review: Matthews and Haynes (1986)), Friedman and Rosenman's work focussed clinicians' and researchers' attention on the importance of looking beyond the physical disease process in order to understand patients' experience of illness and maximise their chances of recovering.

Since this research, psychological theories have been applied to a range of health related areas including:

- the nature of clinician-patient interactions (Tuckett, Boulton, Olson and Williams, 1985);
- clinical decision making, (Schwartz and Griffin, 1986);
- preparation for surgery and hospitalisation (Volicer, Isenberg and Burns, 1977);
- increasing health behaviours (Millar and Millar, 1995);
- the treatment of unexplained physical symptoms (Sharpe, Peveller and Mayou, 1992) and
- the psychological treatment of physical disease, (Lewin, Robertson, Cay, Irving and Campbell, 1992).

Alongside research into psychological components of clinical, treatment and illness processes, a related field of enquiry has been that of psychological morbidity associated with physical health problems.

Emotional reactions to physical illness have been researched extensively and it is generally accepted that around 20 to 25% of patients with chronic medical problems experience clinically significant psychological symptoms (White, 2001). These may include PTSD, anxiety, and depression. Research has also examined psychological morbidity in acute health problems although this field has received less attention from researchers (Salmon, 2000).

There are many concepts derived from clinical health psychology that inform current knowledge regarding patients' experience of illness and treatment. One central concept throughout research in this field is that of health beliefs, or, illness representations. The concept is used to describe the mental representations and explanations a patient may have regarding a disease or physical illness process. This area of research was initiated in response to the observation of substantial variance in physical, behavioural, and psychosocial responses to the same disease process between individuals. This observation led to enquiry into differences between people's perceptions of the disease process and between their causal attributions regarding their illness. One model used to examine the variation in how patients make sense of and respond to illness has been the self-regulation model of Leventhal, Diefenbach and Leventhal (1992). Following research demonstrating that emotional responses to changes in tumour size following chemotherapy were a function of patients' personal cognitive models of the illness, the self-regulation model was developed to account for variations in individual's responses to disease and their choice of coping strategies. Drawing on the work of other theorists working in the field, the model proposed that individuals construct cognitive models of their illness to include five principle components: beliefs about the aetiology of the illness or causal attributions, (Baumann, Cameron, Zimmerman and Leventhal, 1989), its symptoms and a label (Lau, Bernard and Hartman, 1989), the personal consequences of the illness (Croyle and Jemmott, 1991), how long it will last (Croyle and Jemmott, 1991), and whether it is amenable to cure or control (Lau and Hartman, 1983). Thus, the self-regulation model emphasises the

importance of cognitive models of the bodily changes associated with illness (illness representations) held by the patient for directing behaviour aimed at reducing threats to health. The model proposes that patients develop parallel emotional and cognitive emotional representations of their illness, and, consequently, that patients pursue a problem solving approach to make sense of their illness condition and reduce the perceived gap between actual health state and ideal health state (Pitts and Phillips, 1998). The cognitive and emotional representations held by patients determine their choice and use of coping strategies, and the psychological consequences of the illness may be affected accordingly.

The model also allows for changes within illness representations over time as patients' knowledge and experience of the disease process changes.

In order to operationalise the concept of cognitive models of illness further, Weinman and colleagues devised the Illness Perception Questionnaire (IPQ) (Weinman, Petrie, Moss-Morris and Horne, 1996). This questionnaire sought to provide a means of accessing patients' beliefs about illness in a direct and non-threatening manner. The original questionnaire was structured to provide data regarding the five components of illness representations outlined within the self-regulation model (causal attributions, identity, consequences, timeline, and cure/control). In order to improve the psychometric properties of the IPQ and extend the scope of its application, the IPQ has subsequently been amended to produce the Revised Illness Perception Questionnaire (IPQ-R) (Moss-Morris et al.(2002). This revised

questionnaire incorporated a new subscale for 'emotional representation'. As the self-regulation model had included an emotional representation component, this addition was considered necessary in order to obtain a measure of patients' emotional responses to the illness. A further subscale measuring the coherence of patients' understanding of the illness was also added. In order to address problems of internal consistency in the original questionnaire, the revised questionnaire split the 'cure/control' subscale into 'personal cure/control' and 'treatment cure/control' and the 'timeline' subscale into 'timeline (cyclical)' and 'timeline (acute/chronic)'. Thus, by refining the measurement of illness representations, the concept has become more comprehensive.

Although the self-regulation model has initiated a wealth of research into the relationships between illness representations and responses to illness, its essentially descriptive nature has limited the development of behavioural interventions to affect change in health behaviours. Such deficiencies within the model have instigated the development of further models (known as social cognition models) to explain health behaviours (e.g. Theory of Planned Behaviour, (Ajzen, 1991). However, in the absence of extensive empirical testing of all such models, they may more usefully be considered as useful frameworks to generate hypotheses about an individual's health behaviour. They also highlight the importance of including patients' views and beliefs in clinical decision-making, an increasingly important factor in delivering health care services (Salmon, 2000).

Despite the difficulties inherent within comprehensive models of illness representations and behaviour such as the self-regulation model, the concept of illness representations as cognitive constructs affecting a patient's health and recovery remains an important factor. It has been shown that the illness representation held by the patient can account for variations in emotional reactions to symptoms of the physical disease process (Prohaska, Keller, Leventhal and Leventhal, 1987), (Murphy, Dickens, Creed and Bernstein, 1999) as well as variations in self-care behaviours (Petrie, Weinman, Sharpe and Buckley, 1996).

The concept of illness representations is considered congruent with the core view in the cognitive approach within psychology. The cognitive psychology view that individuals construct internal representations, or schema, based on experience and use them to develop responses to new situations is congruent with the concept of illness representations described above. Thus, it is considered theoretically valid to apply the illness representation concept to the study of psychopathology using cognitive psychology principles (Weinman and Petrie, 1997).

A further concept that has been explored in order to account for individual variation in the outcome of disease processes is that of social support. Pennebaker (1993) found that the expression of negative emotion and construction of a coherent story regarding an illness, led to significant health improvements. This implies that social support, by facilitating emotional

expression and the development of a coherent account of illness, may provide health benefits to patients.

Finally, of theoretical relevance to this study, is research into coping strategies utilised to deal with physical illness. Manyande et al. (1995) compared a group who used relaxation techniques pre-operatively with a control group and found that the relaxation coping strategy group experienced less post-operative pain. This study highlights the importance of examining an individual's coping strategies in order to understand responses to physical illness. Solomon, Mikulincer and Flum (1988) have provided evidence that avoidant coping strategies may be associated with maladaptive responses to traumatic experiences generally and Lowe, Norman and Bennett (2000), in studying the coping strategies of myocardial infarction patients and the incidence of health complaints, found that coping strategies and psychosocial adjustment were concurrently associated.

The above psychological concepts of illness representations, social support, and coping strategies have been applied to a range of medical conditions and patients' health care outcomes. This present study aims to examine the areas of psychological morbidity, illness representations, social support, and cognitive coping strategies, in relation to two acute medical events, myocardial infarction, and subarachnoid haemorrhage. These medical events, together with research into their psychological consequences, will now be described.

1.3. Subarachnoid haemorrhage

1.3.1. What is a subarachnoid haemorrhage?

Nearly all the effects of vascular disease on the brain may be reduced to two basic pathological processes – infarction (death of tissue) due to deprivation of blood supply, and intracranial haemorrhage (Lishman, 1988). Clinically, both processes fall under the clinical descriptor 'cerebrovascular accident', or, more commonly, 'stroke'. The World Health Organisation defines this as:

> *'a rapidly developed clinical sign of a focal disturbance of cerebral function of presumed vascular origin and of more than 24 hours duration'*
> (Marquardsen, 1983).

However, intracranial haemorrhages are often considered to be qualitatively different from strokes in both aetiology and clinical presentation.

Intracranial haemorrhages may be divided into two categories, primary intracerebral haemorrhage, in which blood leaks into the cerebral matter, and subarachnoid haemorrhage.

A subarachnoid haemorrhage is a cerebrovascular accident in which a sudden leak of blood occurs over the surface of the brain. The leak of blood occurs between the pia mater and the arachnoid mater, two membranes that

cover the surface of the brain. The space between these membranes (the subarachnoid space) contains blood vessels supplying blood to the brain and cerebro-spinal fluid. The usual cause of a subarachnoid haemorrhage is rupture of an intracranial aneurysm (Bonita and Thomson, 1985). Such a rupture is most likely to occur at the forks of cerebral arteries, the majority arising close to the Circle of Willis at the base of the brain (Figure 1).

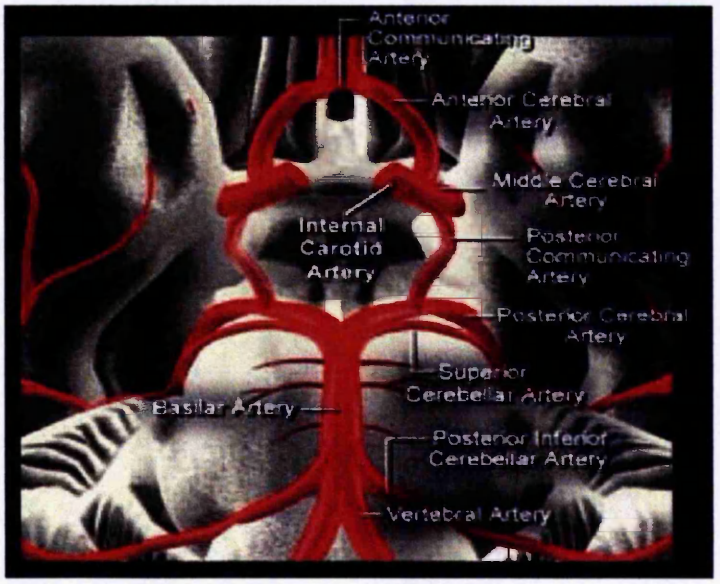


Figure 1: Illustration of the Circle of Willis

Common sites for the rupture of aneurysms are on the anterior cerebral and anterior communicating arteries, at the point of division of the middle cerebral arteries, or where the posterior communicating artery arises from the upper end of the internal carotid artery. Rupture occurs spontaneously with bleeding directly into the subarachnoid space and, occasionally, into adjacent brain substance.

Thus, the onset of the haemorrhage is abrupt and patients often suffer an intense headache. Vomiting, neck pains, photophobia, and seizures may also occur (Berry, 1998). Some individuals lose consciousness or become drowsy and confused, whilst some retain full awareness throughout. Confirmation of a subarachnoid haemorrhage is made by Computerised Tomography (CT) scan through which, the location of the bleeding and aneurysm may be indicated. In the event of a negative CT scan, lumbar puncture (to test for blood in the cerebrospinal fluid) or angiography may be carried out.

1.3.2. Incidence rate, morbidity, and mortality in subarachnoid haemorrhage patients

The incidence of first time cerebrovascular accidents has been estimated at 2 per 1000 population (Bamford et al. (1988). A quarter of patients are under 65 and the risk for males exceeds that for females by 26%. Subarachnoid haemorrhage is considered to constitute approximately 5% of all cerebrovascular accidents and affects a younger age group than other forms of cerebrovascular accident (Lishman, 1988). Aneurysmal subarachnoid haemorrhage has an annual incidence of between 6 and 12 per 100,000 population in most western countries (Linn, 1996). The course of the event is such that approximately 30% of patients will die within 24 hours of the bleed and, without surgical or medical intervention, a further 25 to 30% may die (Alvoord, 1972).

1.3.3. Treatment of subarachnoid haemorrhage

In western countries, those patients who survive the immediate effects of subarachnoid haemorrhage are referred to neurosurgical units. Immediate bed rest is required and the patient is treated with analgesia and sedatives. Early pharmacological treatment with nimodipine, a calcium channel blocker, has been shown to reduce further complications of cerebral ischaemia and subsequent infarction.

Patients may then be treated by one of two interventions. Neurosurgical intervention involves craniotomy and the insertion of a metal clip over the neck of the aneurysm to seal off the site of the haemorrhage (Figure 2).

Figure 2: Surgical clipping of an aneurysm

Outcome from such surgical intervention has been shown to be dependent upon the timing of surgery and the clinical grade of patient on admission. Patients who are stuperose or comatose on admission, have higher mortality rates than those with less impairment of consciousness (Kassell, 1992).Once the aneurysm has been treated surgically, the risk of a further haemorrhage

is the same as for the general population, (Mayberg, Batjer and Dacey, 1994) and patients may be reassured accordingly.

Endovascular treatment has recently been developed as an alternative option for aneurysmal subarachnoid haemorrhage patients. This treatment involves the insertion of metal coiling into the aneurysm in order to prevent blood flow into the aneurysm (Figure 3).

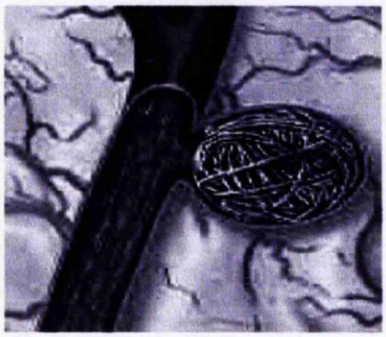


Figure 3: Endovascular coiling of an aneurysm

Although this procedure is usually performed on patients whose aneurysm location has prevented more conventional surgical treatment, the outcome data published so far indicates that the procedure is comparable in post-treatment morbidity and mortality to the surgical procedure (Nichols, 1993). However, comparative data for the two procedures is, at present, insufficient to inform comprehensively clinical and resource decision making, and a randomised controlled trial of the two procedures is currently being undertaken (ISAT, 1994).

1.3.4. Physical, neurological and neuropsychological consequences of subarachnoid haemorrhage

Most of the early research examining the outcomes of subarachnoid haemorrhage has focussed upon physical, neurological and cognitive neuropsychological sequelae (McKenna and Neil-Dwyer, 1993).

During the acute stage, focal ophthalmoplegias frequently occur and, if the haemorrhage has led to arterial vasospasm and further intracerebral bleeding, there may be hemiplegia or aphasia. Persistent coma of more than twenty-four hours is considered a bad prognostic indicator.

In the early stages of recovery, cognitive impairments in the form of severe confusion and akinetic mutism are common. However, these difficulties are often transient. Enduring cognitive sequelae may occur when there has been substantial brain damage.

Research has provided equivocal results regarding the long-term cognitive sequelae of subarachnoid haemorrhage. Early studies found that persistent intellectual deficits occurred in up to 40% of patients (Storey, 1967) with more severe problems being apparent in patients with middle cerebral artery aneurysms. Dysphasic problems were detected in up to 40% of patients with anterior cerebral artery aneurysms (Logue, Durward, Pratt, Piercy and Nixon, 1968) and many of these patients also demonstrated memory deficits. However, later studies utilising more robust cognitive neuropsychological

testing methods have suggested that the level of cognitive impairment suffered by subarachnoid patients may not be as high as previously reported. McKenna, Willison, Lowe and Neil-Dwyer (1989) compared subarachnoid haemorrhage patients with a control group of myocardial infarction patients at discharge, 3 months and one year. In comparing a sample of subarachnoid haemorrhage patients without neurological deficit (i.e. without dysphasia or hemiplegia) with myocardial infarction patients, the study found that there was no significant difference in cognitive testing results. The study concluded that, where the medical and surgical course of the subarachnoid haemorrhage is uncomplicated, patients might recover with no significant or permanent reduction in intellectual ability. Bornstein, Weir, Petruk and Disney (1987), on studying 48 aneurysmal subarachnoid haemorrhage patients, also found that the incidence of neuropsychological deficit was less than in previous reports and suggest that these results may be due to improved bias reduction in sample selection and a broader spectrum of neuropsychological and neurobehavioural functioning measures. However, the study does confirm previous findings that poor cognitive neuropsychological outcome is associated with age and the location of the aneurysm. It also found that neurological outcomes were not associated with the pre-operative grade of haemorrhage whereas neuropsychological outcomes were. In a study of the neurological and psychosocial outcomes of subarachnoid haemorrhage using self-reports of patients 4 to 7 years after the event, Ogden, Utley and Mee (1997) found that 41% of patients reported mild memory problems and a smaller proportion reported sleep disturbance, personality changes, headaches and seizures. However, they conclude that

the majority of patients continue to recover for many years and develop good coping skills and a positive attitude towards their recovery.

1.3.5. Research into the emotional consequences of subarachnoid haemorrhage

The immediate experience of having a subarachnoid haemorrhage is traumatic. The symptoms of sudden onset, intense headaches have been described as being 'agonising' and 'like torture' (McKenna and Neil-Dwyer, 1993) and patients who retain consciousness often give accounts of intense fear and distress at the time of the haemorrhage and may become fearful of the recurrence of the subarachnoid haemorrhage (Berry, 1998).

Early research into the emotional consequences of subarachnoid haemorrhage detected severe and incapacitating anxiety symptoms in 27% of patients (Walton, 1952). Similarly, Storey (1972) found symptoms of anxiety and depression in up to 25% of patients, with severe symptoms reported in up to 14%. In this study, the occurrence of depression was demonstrated to be independent of brain damage subsequent to the haemorrhage. More recent studies, e.g.(Ogden et al., 1997), (McKenna, Willison, Phil, Lowe and Neil-Dwyer, 1989) have explored the psychosocial and quality of life outcomes of subarachnoid haemorrhage. However, there has been relatively little recent research regarding the emotional consequences of subarachnoid haemorrhages. Stegen and Freckmann

(1991) had suggested that one factor affecting patients' anxiety symptoms was their fear of another haemorrhage occurring. Berry (1998) has studied PTSD and anxiety in subarachnoid haemorrhage patients and detected an incidence rate of 32% for PTSD and 50% for anxiety in a sample of patients referred to a clinical psychology service following surgical treatment. This study supported the claim of Stegen and Freckmann (1991) that many patients experienced anxiety regarding the onset of another haemorrhage. However, it is clear that the prevalence rates for psychological disorder as detected in this study sample may be higher than the subarachnoid haemorrhage population as the study sample had been referred for clinical psychology intervention.

1.3.6. Illness representations in subarachnoid haemorrhage patients

To date there has been no research examining the illness representations of people who have suffered a subarachnoid haemorrhage. There is anecdotal data regarding causal attributions for this medical event with patients frequently citing stress or hereditary factors as possible causes for the haemorrhage, although this has not been empirically studied. Berry (1998) comments on the impact of beliefs regarding sudden recurrence of another subarachnoid haemorrhage upon a patient's emotional response and recommends further research into the emotional sequelae of subarachnoid haemorrhage. McKenna and Neil-Dwyer (1993) have discussed the relative ignorance many subarachnoid haemorrhage patients have regarding their

condition and their inability to comprehend information from the neurosurgeon at the time of hospitalisation. This observation also suggests that illness representation factors may have an impact upon outcome and psychological adjustment to subarachnoid haemorrhage.

1.3.7. Rehabilitation and recovery from subarachnoid haemorrhage

Rehabilitation services vary across the country. Some neuroscience centres offer a comprehensive follow-up service involving assessment of psychological, cognitive, and behavioural difficulties subsequent to the haemorrhage, provision of relevant advice and information, rehabilitation, and support for partners and carers, whereas other services will provide a basic medical follow up to the surgical or endovascular procedure. McKenna and Neil-Dwyer (1993), having assessed the provision of aftercare following a subarachnoid haemorrhage, suggest that the majority of anxiety related problems may be alleviated by appropriate and informed counselling regarding their health status and general lifestyle advice. They point out that many primary care clinicians' may be overly protective in their reactions to patients' symptom reporting, thus potentially undermining the patients' confidence in recovery and causing further distress. This study suggests that patients' emotional experience following a subarachnoid haemorrhage and treatment may be affected by an educative approach aimed at changing their illness representations regarding the condition.

1.3.8. Social support and coping strategies for subarachnoid haemorrhage patients

There has been one study examining the role of social support and coping strategies in the subarachnoid haemorrhage population. In their retrospective study of 51 patients, Tomberg et al. (2001) found that patients recovering from a subarachnoid haemorrhage reported using social support strategies less than controls. This study suggests that the role of social support and selective use of coping strategies in psychological outcomes is a valid area for further investigation.

1.3.9. Summary of section 1.3

Having a subarachnoid haemorrhage is likely to be an extremely traumatic event for most sufferers. The sudden onset and threat of serious injury or death make the event fulfil the criteria for PTSD (see section 1.6.2). Research to date has implied links between illness representations and emotional consequences of a subarachnoid haemorrhage and highlighted differences between subarachnoid haemorrhage patients and controls in use of social support as a coping strategy. At present, there is no research looking at the links between illness representation and emotional consequences explicitly and no research examining the role of social support and cognitive coping strategies in determining the psychological outcomes of this condition. Although, there have been studies examining cognitive neuropsychological recovery over time, there have not been any prospective

studies examining changes in illness representations and emotional distress over time.

1.4. Myocardial infarction

1.4.1. What is a myocardial infarction?

A myocardial infarction occurs when a blockage to one of the coronary arteries prevents oxygen from reaching part of the heart (BHFb, 1999). The myocardial infarction, (more commonly known as a 'heart attack') usually causes severe pain in the centre of the chest lasting for upwards of fifteen minutes. The pain may last for several hours and feels like a heaviness or tightness which may also spread to the arms, neck, jaw, face, back or stomach. Frequently, if mild, the symptoms are mistaken for severe indigestion. The onset of myocardial infarction is often sudden and excruciatingly painful and may lead to a cardiac arrest in which the heart stops pumping. Thus, the event is considered life threatening and emergency treatment is required.

A myocardial infarction is often the result of a disease process that has been progressing for many years. The walls of coronary arteries become narrowed through a build up of fatty material (atheroma). These diseased arteries may then crack and platelet blood cells become attracted to the damaged areas. A clot (or thrombus) may form on top of this and block the artery. If the artery

is blocked for a few minutes, the muscle cells in the area of the heart supplied by that artery may become permanently damaged (Figure 4).

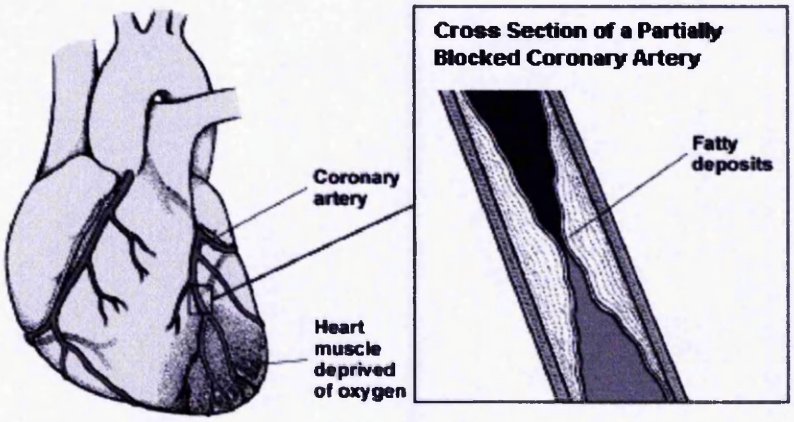


Figure 4: Illustration of atheroma of coronary artery and damage to heart muscle

After a heart attack, the area of the heart muscle that was affected will be replaced with scar tissue. After two to three months, the hearts of many patients are functioning just as well as they were before the attack. However, for some patients, the damage is so severe that the pumping action of the heart is impaired. This may result in symptoms of breathlessness, tiredness, and swelling of the ankles. Some patients will experience chest pain on exercise (angina) due to persisting narrowing of the coronary arteries.

During a heart attack, the heart rhythm may be disturbed. The most serious form of this is called ventricular fibrillation and this occurs when the electrical activity of the heart becomes so chaotic that the heart stops pumping and

quivers or 'fibrillates' instead. This is called a cardiac arrest and, if detected immediately, can be brought under control by administering a large electric shock through the chest wall using a defibrillator. During a cardiac arrest the patient loses consciousness immediately, breathing may stop and a pulse cannot be detected.

There has been extensive research to identify risk factors for myocardial infarction. There are four major risk factors: smoking, high blood pressure, high blood cholesterol, and physical inactivity (BHFb, 1999). Other risk factors, which contribute to the above include, obesity, excessive salt intake, and excessive alcohol intake. Some individuals are genetically predisposed to coronary disease that may lead to a myocardial infarction and people with diabetes are predisposed to coronary heart disease.

1.4.2. Incidence rate, morbidity, and mortality in myocardial infarction patients

There are 300,000 myocardial infarctions per year within the UK and circulatory disease accounted for 44% of all deaths in the UK in 1994 (BHF, 2002).

The prevalence rate for myocardial infarction differs between males and females. According to the Joint Health Surveys Unit, the prevalence rate for

men aged between 65 and 74 is 11.6%, whereas for women it is 5.5%. (JHSU, 1999).

1.4.3. Treatment of myocardial infarction

As soon as myocardial infarction is suspected, arrangements are made for an emergency admission to hospital. The first priority of treatment is pain relief and ambulance staff will use a mixture of oxygen and nitrous oxide through facial mask to relieve the pain. Aspirin may also be given to thin the blood and prevent further clotting. On admission to hospital, patients are injected with a thrombolytic drug such as streptokinase in order to dissolve the clot blocking the coronary artery (a process called 'thrombolysis'). The patient is usually transferred from accident and emergency to a coronary care unit where the heart rhythm is monitored and further tests are carried out to detect the extent of the damage and blood levels.

Once stabilised, the patient will receive treatment aimed at preventing further heart attacks, (e.g. aspirin, beta-blocker or anti arrhythmic drugs) and aimed at relieving breathlessness and heart failure (e.g. diuretics and ACE inhibitors). If tests have shown that one or more of the coronary arteries is severely blocked, then the patient may be advised to have coronary angioplasty (compression of the atheroma in order to widen the coronary artery) or coronary bypass surgery (grafting of blood vessels to bypass the narrowed part of the coronary artery).

1.4.4. Physical consequences of myocardial infarction

Many people who have suffered myocardial infarction may experience fatigue, breathlessness, and swelling of the ankles and feet subsequent to their condition. These symptoms may be have a sudden onset or may develop over months (BHFa, 1999).

1.4.5. Research into emotional consequences of myocardial infarction

Unlike subarachnoid haemorrhage research, there have been several recent studies examining emotional consequences following myocardial infarction. Bennett and Carroll (1997) state that most research into the consequences of myocardial infarction has been concerned with the emotional and psychological aspects of recovery. This may be the result of early research suggesting that the occurrence of depression in myocardial infarction patients has a detrimental effect upon recovery. For example, Mayou, Foster and Williamson (1978) and Carney, Freedland, Eisen, Rich and Jaffe (1995) found that depression, by disengaging patients from adherence to adaptive strategies was associated with poorer outcomes. Similarly, Trelawney-Ross and Russell (1987) found that depression whilst in hospital or 10 days after discharge was associated with the number of symptoms reported at 6 months. Lowe et al. (2000) in a longitudinal study, examined emotional

reactions to myocardial infarction and found that depression and anxiety symptoms remained constant over a 6 month period. Thus, research has indicated the importance of considering the patient's emotional status in the physical recovery process. This concern with the impact of psychological factors upon the recovery and rehabilitation process is demonstrated by the quantity of research examining the efficacy of cardiac rehabilitation programmes.

There have been several studies examining the incidence of PTSD in myocardial infarction patients. In their cross-sectional, retrospective study, Bennett and Brooke (1999) discovered a 10% prevalence of PTSD in myocardial infarction patients. Similarly, Kutz, Shabtai, Solomon, Neumann and David (1994) found a PTSD prevalence rate of 16% in myocardial infarction patients whilst Doerfler, Pbert and DeCosimo (1994) stated an 8% prevalence rate for patients who had a myocardial infarction and patients who had cardiac surgery.

Wiklund, Sanne, Vedin and Wilhelmsson (1984) found that 74% of first time myocardial infarction patients had persistent worries regarding their health. This study of the emotional consequences of myocardial infarction reveals the necessity to consider the role of illness representations.

1.4.6. Illness representations in myocardial infarction patients

The illness representations of myocardial infarction patients have been extensively researched. In formulating the Illness Perception Questionnaire, Weinman and colleagues selected myocardial infarction patients as one of the research groups and, therefore, provided data regarding their illness representations. This study demonstrated that increased timeline and consequences scores were associated with increased fear of recurrence of a myocardial infarction (Weinman et al., 1996). Although the Lowe et al. (2000) study had detected relative stability in emotional outcome over time, the Weinman et al. (1996) study, found a low test-retest correlation for IPQ factors over a 6 month period for myocardial infarction patients. They suggest that this result may be due to the fact that patients will have developed a very different attitude to their illness since the acute event.

In a further study into causal attributions and myocardial infarction, Weinman et al. (2000) found that specific patterns of causal attributions in first time myocardial infarction patients were associated with changes in health related behaviour after 6 months. This finding demonstrates the importance in considering the illness representations of patients when examining clinical outcomes and changes in health related behaviour. However, to date there are no studies exploring links between the full range of illness representations specified within the self-regulation model (and measured by the IPQ-R) and a broad range of emotional distress.

1.4.7. Rehabilitation and recovery after a myocardial infarction

Myocardial infarction patients benefit from extensive research directed towards improving outcomes for their recovery process. As it has been ascertained that the four main risk factors for myocardial infarction are smoking, physical inactivity, high blood pressure and high blood cholesterol, rehabilitation programmes have been structured to support health behaviour change in these areas. Most rehabilitation programmes will offer support in building up exercise routines, dietary and smoking cessation advice, and access to patient support groups. Mayou et al. (2002) have evaluated the effectiveness of a educational behavioural programme for acute myocardial infarction patients and found that the intervention led to a reduction in further treatment needs, increased confidence in returning to activities one month after the myocardial infarction. The establishment of the British Heart Foundation has served to promote and disseminate good practice in cardiac rehabilitation across the country.

The development of Cardiac Rehabilitation Programmes nationally is increasingly informed by standards set out in the National Service Framework for Coronary Heart Disease which states:-

> *NHS Trusts should put in place agreed protocols/systems of care so that, prior to leaving hospital, people admitted to hospital suffering from coronary heart disease have been invited to participate in a*

multidisciplinary programme of secondary prevention and cardiac rehabilitation. The aim of the programme will be to reduce risk of subsequent cardiac problems and to promote their return to a full and normal life. (DOH, 2000).

1.4.8. Social support and coping strategies for myocardial infarction patients

The Bennett and Brooke (1999) study has explored the role of social support and coping strategies in myocardial infarction patients and found that social support was predictive of negative affect. Lowe et al (2000), in examining the role of coping strategies in recovery from myocardial infarction, found that problem focused coping was adaptive. There have not been any studies exploring the predictive capability of cognitive coping strategies and perceived levels of social support for PTSD following myocardial infarction.

1.4.9. Summary of section 1.4

Having a myocardial infarction is likely to be an extremely traumatic event for most sufferers. The sudden onset and threat of serious injury or death enable the event partially to fulfil the criteria for PTSD (see section 1.6.2). There has been considerably more research into the psychological aspects of recovery and health behaviour change for myocardial infarction than for subarachnoid haemorrhage. Research to date has explored links between

specific illness representations (causal attributions), coping strategies, and emotional consequences of a myocardial infarction. At present, there is no research examining the roles of the full range of illness representations, social support and coping strategies together in determining the emotional outcomes of this condition. There have been studies examining illness representation changes and recovery over time, although these studies remain limited in scope.

1.5. Defining acute medical trauma

The disease processes of subarachnoid haemorrhage and myocardial infarction are very different. However, the above accounts of the emotional consequences of these two traumatic medical events reveal similarities in the patients' experiences. Both conditions involve sudden onset, life threatening, and painful experiences that may lead to a slow recovery period with anxiety, depression, and PTSD symptoms. McKenna and Neil-Dwyer (1993) in their study of cognitive outcome and quality of life one year post event compared a subarachnoid haemorrhage group with a myocardial infarction control group. The rationale for this choice of control group was that both groups of patients had suffered a sudden, life-threatening illness. The results of the study reveal many similarities in cognitive and quality of life outcomes for both groups and, thus, validate the argument for considering the two groups as similar in terms of their experience of acute medical trauma. Thus, both groups appear to have shared a similar experience and may be usefully studied together as patients who have undergone an acute medical trauma. Both groups, through having had such experiences may also be considered as fulfilling part of the criteria for PTSD that will be discussed in section 1.6.2. Due to the shared nature of their experiences, both groups may also have illness representations in common which may influence their experience of PTSD and other emotional distress.

1.6. Post- Traumatic Stress Disorder (PTSD)

1.6.1. Development of the concept of PTSD

Throughout history, human responses to traumatic experiences have been recorded and noted to include certain characteristics. For example, Samuel Pepys recorded his emotional reactions to the trauma of the great fire of London in 1666 and his attempts to cope with them (Daly, 1983).

Early theorising regarding the phenomena of psychological consequences of trauma came with the advent of mass transport in the nineteenth century. This period gave rise to a range of concepts, such as 'spinal concussion' and 'railway spine', to explain the hypothetical link between psychological consequences for victims of transport accidents and damage to the central nervous system (Erichsen, 1866). Freud (1894), through his observations of stress responses to trauma, developed the concept of 'anxiety neurosis' to explain the relationship between the traumatic event and psychological and behavioural symptoms.

Perhaps the most significant development in clinical awareness of the psychological consequences of acute stress occurred in the twentieth century following the trauma inflicted on thousands of soldiers and civilians in combat. The highly visible distress of those returning from combat, coupled with the development of scientific techniques for researching such distress, led to an increasingly rich theoretical understanding of the psychological

symptoms of trauma. During World War 1, 'shell shock' was the term most frequently used to describe the syndrome observed in soldiers who had served on the battlefield and, in keeping with previous beliefs regarding the link between the symptoms and central nervous system injury, this was considered to be related to physical injuries to the nervous system caused by explosions. By World War 2, mental health professionals had become much more involved in the care of military personnel. Kardiner (1941) systematised his observations of symptoms of irritability, aggressive outbursts, exaggerated startle responses, and fixation on the traumatic event and developed the concept of a syndrome he termed 'post-trauma syndrome'. Further refining of the concept occurred in the aftermath of the Vietnam War. The diagnostic term 'Post-Traumatic Stress Disorder' was first used after this conflict in order to systematise the diagnosis of clinical phenomena and facilitate the treatment and compensation process for the many traumatised military personnel returning home (Figley, 1978). As well as describing the symptomatology and course of the disorder, research from this time demonstrated the potentially long-term nature of the condition and its social and personal consequences.

Subsequent to the Vietnam War, many mental health professionals began to note similar clinical presentations in civilians who had experienced severe trauma. Horowitz (1975) sought to explain PTSD utilising psychodynamic and information processing concepts and conceptualised the individual's presentation as the result of being overwhelmed by intrusive and emotionally disturbing memories. Horowitz described the process by which the individual

then sought to defend against the distressing intrusions by using avoidant strategies. This work led to the codification of PTSD within the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (APA, 1980). Since this time, there have been two further revisions of the diagnostic criteria for PTSD in 1987 and 1994. A current definition of PTSD is found in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (APA, 1994).

1.6.2. Current definition of PTSD

1.6.2.1. Symptomatology and diagnosis of PTSD

DSM IV (APA, 1994) provides diagnostic criteria for PTSD according to four criteria, the first of which (Criterion A) relates to the nature of the traumatic event itself:

Criterion A - The person must have been exposed to a traumatic event in which both the following were present:

1. The person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of the self or others.
2. The person's response involved intense fear, helplessness, or horror.

The remaining diagnostic criteria relate to the symptomatic responses of re-experiencing phenomena, avoidance, and hyperarousal in the individuals experiencing the trauma. These are referred to as Criteria B, C, and D.

Criterion B – The traumatic event is persistently re-experienced in one (or more) of the following ways:

1. Recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions.
2. Recurrent distressing dreams of the event.
3. Acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations and dissociative flashback episodes, including those that occur on awakening or when intoxicated).
4. Intense psychological distress at exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.
5. Physiological reactivity on exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.

Criterion C – Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:

1. Efforts to avoid thoughts, feelings, or conversations associated with the trauma.
2. Efforts to avoid activities, places or people that arouse recollections of the trauma.
3. Inability to recall an important aspect of the trauma.
4. Markedly diminished interest or participation in significant activities.
5. Feeling of detachment or estrangement from others.

6. Restricted range of affect (e.g. unable to have loving feelings).
7. Sense of a foreshortened future (e.g. does not expect to have a career, marriage, children, or a normal life span).

Criterion D – Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:

1. Difficulty falling asleep or staying asleep.
2. Irritability or outbursts of anger.
3. Difficulty concentrating.
4. Hypervigilance.
5. Exaggerated startle response.

For a diagnosis of PTSD, the symptoms must exceed 1 month. A diagnosis of Acute Stress Disorder (ASD) may be considered for the presentation of the above symptoms within this time scale. In cases of delayed onset PTSD, symptoms may develop more than 6 months after the trauma. A significant disruption in social, occupational, or other important areas of functioning is also required for diagnosis.

1.6.2.2. Time course of PTSD

A large proportion of people exposed to severe trauma frequently experience the above symptoms and they often disappear within a few weeks. However, for a significant number of people, the symptoms persist for months or even

years. Holen (1991) studied the survivors of an oilrig collapse and found that some were severely affected 8 years later. Seventeen years after being sexually assaulted, 17% of women were found to still have PTSD (Kilpatrick, Saunders, Veronen, Best and Von, 1987). A National Comorbidity survey undertaken by Kessler, Sonnega, Bromet, Hughes and Nelson (1995) undertook a survival analysis which revealed that more than one third of people with an index episode of PTSD fail to recover, even after many years (the lifetime prevalence for PTSD being 8%). In a more detailed study of the time course of psychological distress following trauma, McFarlane (1988) assessed the psychiatric morbidity of fire fighters after a bush fire in Australia in which colleagues were killed. Assessments at 4, 11 and 29 months after the event revealed that 50% never reached caseness (i.e. a level of clinical significance) for a psychiatric disorder, 9% formed an acute group with caseness at 4 months but not thereafter and 10% developed a chronic and persistent set of disorders. Interestingly, the study also found that 5% reached caseness at 4 and 29 months, but were well at 11 months. Although this study addressed the broad concept of psychiatric disorder rather than PTSD in particular, it raises the possibility that the time course of PTSD reactions may not always be linear and adaptation to trauma may involve remitting and recurring psychological morbidity. Studies of people suffering burn injuries also indicates that PTSD may occur after discharge from hospital and thus the condition may involve a delayed onset (Tarrier, 1995).

1.6.3. Co-morbidity

In addition to the symptoms of PTSD as detailed in DSM IV, survivors of trauma often suffer a range of other psychological and social consequences. Horowitz (1986) noted that many survivors undergo personality changes as well as high levels of anxiety and depression. The McFarlane and Papay (1992) study of Australian fire fighters found that 77% of those diagnosed as having PTSD had at least one other psychiatric diagnosis. Many other studies report, in particular, high levels of depression following a traumatic event. (North, Smith and Spitznagel, 1994). Other areas affected by trauma include cognitive functioning (McNally, Lasko, Macklin and Pitman, 1995), substance use (Kulka et al. (1990), physical health (Price, 1978) and social relationships (Goenjian, 1993). Thus, it is considered useful to address other manifestations of psychological distress when addressing PTSD in order to obtain a fuller picture of a person's clinical presentation (Yule, Williams and Joseph, 1999).

1.6.4. Areas investigated in PTSD research

1.6.4.1. War

Section 1.6.1 has highlighted the fact that much of our current knowledge of PTSD arose from a need to understand the experiences of military personnel returning from the battlefield. The experience of combat meets Criterion A for

diagnosis of PTSD according to DSM IV (APA, 1994) in that the military personnel experience situations that are threatening to life or to the physical integrity of the self or others.

Recent studies have shown high rates of PTSD symptomatology amongst combatants. Orner, Lynch and Seed (1993) found that 32 out of a sample of 53 Falklands war veterans manifested sufficient numbers of symptoms to meet DSM III criteria for a diagnosis of PTSD. A prospective study of a large cohort of Gulf War combatants showed that PTSD rates increased over time after return from conflict (Wolfe, Erickson, Sharkansky, King and King, 1999). In light of the high prevalence rates of PTSD within this population group, this group has been used in order to determine the possible predictors and course of PTSD. However, questions have been raised about the suitability of generalising findings from this population to civilian groups (Brewin, Andrews and Valentine, 2000).

1.6.4.2. Disasters

Following the increased media attention given to the impact of disasters upon civilians, research into this area has developed. Yule et al. (1999) studied the experiences of survivors of the 'Jupiter' ferry disaster and found that 50% of survivors developed some symptoms of PTSD. More generally, findings from studies into psychological consequences of disasters has

demonstrated a 'dose-response' relationship between stressors and symptoms (e.g. Weisaeth, 1983).

1.6.4.3. Road traffic accidents

Given the relative frequency of road traffic accidents (RTAs) in civilian life compared to war or disaster experience, this has proved to be an interesting and relevant area for enquiry into responses to trauma. In 1998, over 320,000 people were injured in RTAs and were admitted to Accident and Emergency departments for treatment (DETR, 1998) and it has been claimed that RTAs are the main cause of death in people under 30 years of age.

Prospective studies of RTA sufferers indicate that approximately 25 % of people experience PTSD symptoms within the first 3 to 6 months (Ehlers, Mayou and Bryant, 1998), and approximately 15% go on to develop PTSD over the following 12 months. Bryant and Harvey (1995) found that between 78% and 82% of RTA survivors who have PTSD symptoms within the first month of their accident develop PTSD by 6 months.

In a prospective study of survivors of RTAs, Holeva, Tarrier and Wells (2001) examined the role of perceived loss, social support and thought control strategies in predicting the development of PTSD. This study is described in more detail in section 1.6.8.

1.6.4.4. Physical illness and injury

Although most PTSD research has been directed at traumatic experiences involving external stressors, more recently, attention has been drawn to the experience of PTSD symptoms following internal medical events. Amongst increasing numbers of researchers, van Driel and Op den Velde (1995) and Bennett and Brooke (1999) have argued that the experience of an acute medical event such as myocardial infarction may meet the criteria for PTSD according to DSM IV in that such an experience is sudden, unexpected, and carries with it the potential threat of death. Accordingly, Bennett and Brooke (1999) have studied a range of predictor variables for PTSD including alexithymia, age, social support, and awareness of the myocardial infarction at the time of its occurrence. Their retrospective study found that each of the above variables was associated with PTSD. Berry (1998) has studied PTSD rates in subarachnoid haemorrhage patients. Similarly, Ballard, Stanley and Brockington (1995) have reported PTSD symptoms in women in childbirth. Thus, the application of PTSD concepts and psychological treatment of PTSD is increasingly being considered in the field of acute medical trauma such as myocardial infarction and subarachnoid haemorrhage.

1.6.5. Implications for research into PTSD

PTSD symptoms have been observed and noted by clinicians working with patients who have experienced acute medical trauma. These symptoms may

have been subsumed within the concepts of anxiety or depression by medical and nursing clinicians seeking to understand the emotional reaction to a life-threatening event. Patients' emotional responses have been normalised as part of the recovery process that, with its emphasis upon achieving medical stability, may have paid less attention to the emotional experience of the patient. As many patients present with only some symptoms of PTSD, consideration of diagnosing PTSD may also have been overlooked. Increasingly, recent research has indicated the appropriateness and usefulness of utilising the PTSD concept within acute medical trauma populations in order to understand their emotional experience and recovery. However, many of the studies in this area have been retrospective studies and have addressed a limited range of predictor variables in order to assess PTSD. In particular, the roles of illness representations, which have been shown to have an effect within recovery and adjustment to illness, have not been fully examined in respect of PTSD. Further study of illness representations, social support, and cognitive coping strategy predictor variables may yield further information that could usefully inform screening and rehabilitation programmes for both subarachnoid haemorrhage and myocardial infarction populations.

1.6.6. Models and theories of PTSD

With the acknowledgment that the development and maintenance of PTSD was not solely dependent upon physical damage to the central nervous

system or upon the nature of the precipitating traumatic event, it became clear that PTSD was the product of a more complex array of factors. A range of psychological theories arose to describe possible mechanisms for the phenomenon of PTSD as it became necessary to account for the wide range of symptoms as well as the variety of predictors of caseness following trauma.

1.6.6.1. Two factor learning theory

One of the initial theories regarding PTSD development and maintenance was the two factor learning theory of Mowrer (1960). This theory drew upon both classical and operant conditioning in explaining the fear response and avoidant behaviour of those affected by PTSD. The first factor involved a neutral stimulus (NS) becoming associated with an unconditional stimulus (UCS) that immediately evoked fear. Thus, the NS would become a conditioned stimulus (CS) that elicited a fear reaction. The subsequent pairing of the initial conditioned stimulus (CS1) with other neutral stimuli would then lead to the development of further conditioned stimuli (CS2, CS3 etc.). This process of generalisation may lead to a range of stimuli, including events, people, environments, words, thoughts, and images, acquiring the same fear provoking potential as the initial conditioned stimuli CS1. Factor 2 involved the development of learned responses that decreased or terminated the fear arising from the presence of a range of conditioned stimuli. The most common response employed to reduce fear is avoidance, thus the individual

learns to avoid a range of conditioned stimuli, and the fear reaction is consequently reduced or terminated.

Whilst the above theory provides a useful account of how individuals develop avoidant behaviour in response to a range of stimuli beyond the initial trauma, it does not account for other phenomena observed in PTSD. For example, it fails to explain the occurrence of intrusive thoughts and images, and the startle response often observed in PTSD. Foa, Steketee and Rothbaum (1989) also note that the theory does not account for why individuals go on to avoid non-dangerous situations and why this avoidance persists. A number of studies suggest that premorbid personality factors and individual perceptions of threat may also contribute to the experience of PTSD as well as the exposure to and the magnitude of the stressor (McFarlane, 1989) (Feinstein and Dolan, 1991).

Such criticisms of the two factor learning theory led to further theorising regarding PTSD.

1.6.6.2. Cognitive processing

Cognitive processing models of PTSD have stressed the role of cognitions and beliefs in developing and maintaining PTSD. Such models suggest that the development of PTSD is due to trauma related information not being processed into existing cognitive structures and to repeated avoidance of

intrusions and emotions associated with the trauma (Litz and Keane, 1989). Harvey and Weary (1985) suggest that individuals have a desire to predict the future, to control events, and to operate on a day-to-day basis through beliefs that the world is controllable and predictable. Schematic representations of such beliefs are likely to be defended by an individual in order to maintain the personal sense of predictability and controllability necessary for daily functioning. When an individual encounters information that challenges the existing schema there are two options: the individual may seek to assimilate the information to fit in with the existing schema or the existing schema can be accommodated to fit with the new information. Extremely traumatic events may prevent either of these processes from occurring as the information is too extreme to assimilate and too threatening to be accommodated by the existing schema. Janoff-Bulman (1985) suggests that material that is not processed via assimilation or accommodation may continue to cause emotional distress through intrusive thoughts and images, flashbacks and nightmares. Over time, a process of inhibition is adopted in order to regulate such distress. An individual may also avoid processing such information as it presents such a threat to the existing schema. This model, therefore, provides an explanation for the phenomena of intrusions and behavioural and cognitive avoidance in PTSD. However, it is unable to account for individual variations in the onset of PTSD according to personal and social risk factors.

1.6.6.3. Self-regulatory executive function model (S-REF)

The cognitive model has been criticised for utilising too narrow a focus on the content of cognition (including schema) and not paying enough attention to broader aspects of cognitive processing such as thought control strategies, attentional processes, and meta-cognition (Wells and Matthews, 1994). In order to address these issues and variations in the cognitive processing styles of individuals and the impact upon emotional disorders, Wells and Matthews (1994) have developed a model distinguishing three levels of processing. The three levels of processing proposed within the model include an automatic processing level dealing with immediate internal and external stimuli, a controlled processing level that deals with the regulation of thoughts and behaviour and a schema level comprising of a store of beliefs that direct the controlled processing. The S-REF model states that self-regulation is achieved by low-level stimuli (sensory and affective data) interacting with high-level beliefs through consciously controlled cognitive processes. This self-regulatory process permits the individual to deal with distressing information from internal and external sources, and discrepancies with current schema by selecting certain behavioural and cognitive coping strategies.

The model suggests that individuals develop and maintain emotional distress by utilising attentional strategies characterised by threat monitoring, self-focussed attention, and cognitive strategies of rumination and worry. Worry (which is conceptualised as a thought control strategy rather than a symptom

of anxiety (Wells, 2000) and rumination strategies result in low-level threat detection processes being continuously primed and more adaptive self-regulation processes are consequently deprived of cognitive processing resources. Similarly, perseverative self-focussed attention diverts attentional resources away from processing new information that is incompatible with existing beliefs. The model, as applied to PTSD, suggests that while the cognitive-attentional system utilises maladaptive thought control strategies such as rumination and worry, it is unable to process trauma related information and adapt beliefs at a higher level to cope with such information.

This model, therefore, allows scope for consideration of schematic representations of trauma, idiosyncratic cognitive styles, and both internal and external traumatic stimuli as factors affecting the development and maintenance of PTSD for acute medical trauma information. However, it does not include social factors that may affect the experience of PTSD.

1.6.6.4. Psychosocial model of PTSD

The psychosocial model of PTSD (Williams and Joseph, 1999) offers an integration of cognitive and behavioural factors addressed in previous models together with an acknowledgment of social and environmental factors affecting the development and maintenance of PTSD. The model states that an individual's emotional state is a product of interactions between event cognitions (conscious and non-conscious representations of the event), appraisal mechanisms (e.g. rumination and attributional processes), coping

strategies (cognitive, avoidant and active strategies for dealing with distress), personality factors (pre-existing schema) and the environmental and social context (social support and environmental triggers). Thus, this model integrates cognitive, behavioural, social, and event factors in order to facilitate a fuller understanding of the experience of PTSD and provides a broad conceptual framework for examining the question of why some individuals are more prone to develop PTSD than others. In particular, the model proposes that a more complex understanding of PTSD may be informed by consideration of the nature of the representations of the trauma, the schematic representation of the trauma and its consequences, and coping strategy (both cognitive and behavioural) and social support factors. A diagrammatic representation of the model is shown below in Figure 5.

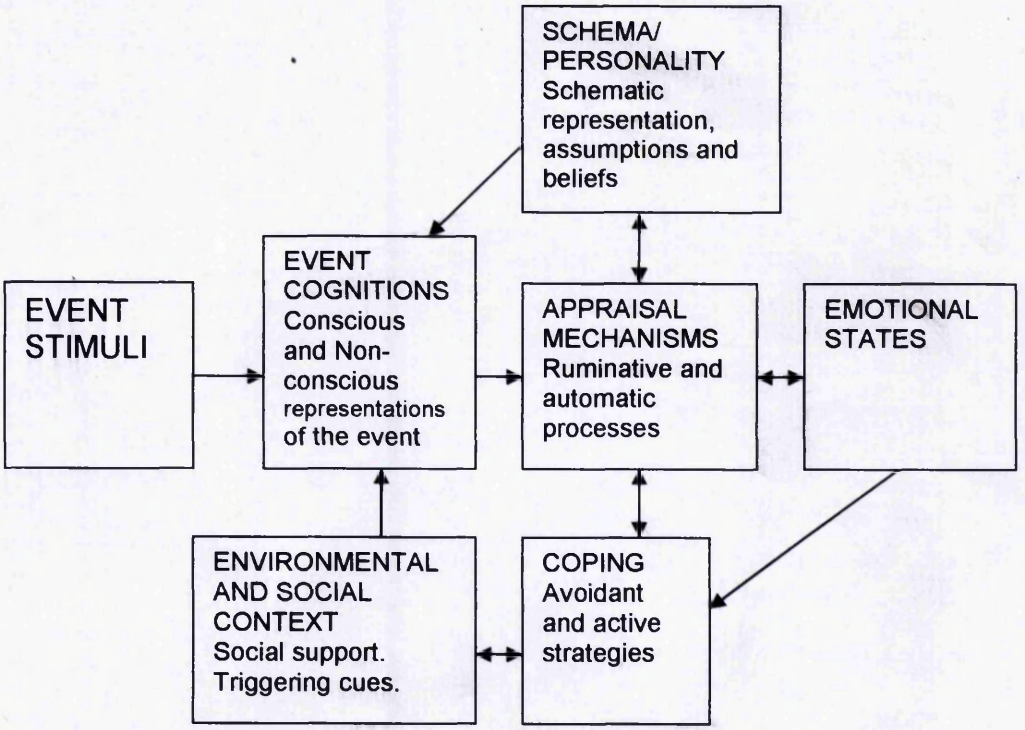


Figure 5: Integrative model for adaptation to traumatic stress (Williams and Joseph, 1999).

1.6.6.5. Predictors of PTSD according to the psychosocial model

The psychosocial model of PTSD (Williams and Joseph, 1999) specifies several factors that impact upon the experience of emotional consequences following an traumatic event. As the current study aims to explore a number of these factors, they will now be examined in turn.

1.6.6.5.1. Event cognition factors

The occurrence of a traumatic event leads to the cognitive representation of the event stimuli within the survivor's memory. The memory trace is considered to be particularly strong due to the traumatic event's personal salience and the difficulty such a memory presents for assimilation or accommodation within existing representations. Williams and Joseph (1999) propose that event cognitions may take two forms, information that is not available for conscious inspection and information that intentionally retrievable and may be impacted upon by aspects of the individual's past experiences, personality and beliefs. Thus, cognitions regarding the traumatic event are affected by both bottom-up (event related information) and top-down (schema related information) processes. Within PTSD, the event cognitions may be experienced as intrusive cognitions, either in verbal or imagery form. The intrusions are considered an intrinsic component of PTSD according to DSM IV criteria and are, by nature of them being

unwanted, difficult to control. Thus, the individual is prompted to act to eradicate or reduce the impact of the intrusion using thought control, emotional expression or other behavioural methods (de-Silva and Marks, 1999).

1.6.6.5.2. Schema factors

As mentioned above, the psycho-social model states that event cognitions may be affected by 'top-down' schematic representations held by the individual. Thus, schematic beliefs and assumptions regarding the traumatic event and regarding the individuals themselves are likely to impact upon the nature of the event cognition itself, and, in turn, upon the experience of further PTSD symptoms. The psychosocial model also proposes that schematic representations may be modified by the appraisal of the event cognitions through assimilation and accommodation. This aspect of the model is informed by the work of Horowitz (1986) who noted a 'completion tendency' in individuals wishing to make sense of their trauma and, thus, modify their pre-existing schematic representations. This tendency is hypothesised to preserve self-esteem as the individual seeks to understand causation of the trauma. In order to obtain data regarding the schematic representation of acute medical trauma such as myocardial infarction and subarachnoid haemorrhage, it is possible to explore a patient's illness representations which are likely to be informed by both event related and previous illness schema related information (Weinman and Petrie, 1997). Schema information regarding acute medical trauma will include causal attributions as well as illness representations concerning duration,

consequences, identity, and cure/control according to the self-regulation model.

Suls and Fletcher (1985) found that the search for causal attributions soon after the trauma experience might lead to higher levels of emotional distress but become more adaptive later. In a further comment on changes in schematic representation of illness over time, Leventhal (1997) noted that illness representations are likely to change over time as an individual's knowledge and direct experience of the disease process change. These findings, coupled with research detailing variances in the time course of PTSD (McFarlane, 1988), emphasises the importance of examining cognitive and emotional phenomena over time in order to understand factors predictive of PTSD.

The above account has stressed the importance of illness representations in exploring the psychological consequences of myocardial infarction and subarachnoid haemorrhage. Leventhal has commented that there is a lack of research into the relationship between illness representations and psychological morbidity. Furthermore, Leventhal has pointed out the weakness in many illness representation studies in that most are cross-sectional, lab based and short-term.

1.6.6.5.3. Environment and social context factors

The psychosocial model exceeds the majority of behavioural and cognitive models of PTSD in specifying the impact of social and environmental factors within the development and maintenance of PTSD. In particular, social support has been specified as a predictive factor of PTSD.

A lack of social support has been found to be associated with high levels of PTSD in war veterans (Green, Grace, Lindy, Gleser and Leonard, 1990). Similarly, Pruzinsky, Rice, Himel, Morgan and Edlich (1992) found that social support is a significant variable affecting an individual's psychological adjustment to burn injuries. In a study of expressed emotion within families of people with a diagnosis of schizophrenia, Tarrier, Barrowclough, Porceddu and Watts (1988) detected an association between high levels of expressed emotion and high levels of autonomic arousal. This finding has implications for people with PTSD as arousal levels may be instrumental in triggering PTSD symptoms (Holeva et al., 2001). A further mechanism whereby a negative psychosocial environment may impact upon PTSD is the inhibition of the cognitive processing of the trauma and consequent maintenance of maladaptive coping strategies (Tarrier, 1996).

Research into social support and PTSD has typically focussed upon positive social interactions (Holeva et al., 2001) and it is commonly believed that talking through the event is a beneficial process allowing the individual opportunity to make their own sense of what has happened to them. This

belief is held so widely that Allen (1995) has commented that 'talking about it to someone' is considered the 'universal prescription' for dealing with trauma. However, subsequent research has refined the concept of social support by highlighting the need to consider the perception of the individual regarding the adequacy of their social support and differentiating between practical and emotional support. In order to operationalise a more refined concept of social support, Holeva (1998) modified the Perceived Criticism Scale (Hooley and Teasdale, 1989) and developed the Social Support Inventory. This self-report questionnaire measures perceived social support, critical interactions within the social environment and satisfaction with emotional and practical support following a traumatic event. The subjective nature of this measure is considered an important feature as previous studies have examined the relationship of subjective rather than objective social support in PTSD (Solomon, Mikulincer, Fried and Wosner, 1987).

With respect to acute medical trauma, Bennett and Brooke (1999) examined the relationship between social support and PTSD in a myocardial infarction sample retrospectively. They found that nearly 10% of the variation in PTSD scores was accounted for by social support variance. The role of social support in predicting PTSD within a subarachnoid haemorrhage population has not been explored to date.

Whilst it is appealing to think that social support may be of equal benefit for all individuals, individual variation in the usefulness and protectiveness of social support must be considered. Holeva et al. (2001) in studying survivors

of RTAs found that perceived social support was only significant in its impact upon PTSD when associated with preferential choice of social control as a thought control strategy for dealing with emotional distress. Thus, when exploring the role of social support in medical trauma events, it is important to consider individuals' chosen thought control strategies to understand the impact of social support upon their clinical presentation.

1.6.6.5.4. Appraisal mechanism factors

Within the psychosocial model, appraisal factors are conceptualised as ruminative and automatic thought processes. Many studies have drawn attention to the phenomena of searching for causal explanations of the trauma as being a common feature of adaptation to the trauma. Thus, many traumatised individuals may adopt a ruminative appraisal style as they search for meaning and causal explanations for their experience. The literature on blame highlights the tendency for individuals to preserve their self-esteem during the early period of adjustment by attributing cause to external sources. However, to preserve a sense of personal control, individuals are also motivated to attribute trauma to their own behaviour. These tendencies may result in complex attributional processes involving multiple and conflicting sources of causation (Weinberg, 1994). It is possible that a ruminative appraisal style for the individual to make sense of such complexity and tension arises out of such complexity and confusion.

Consideration of an individual's appraisal style also suggests that an individual's attributional style may affect their experience of PTSD (Mikulincer and Solomon, 1988) although much of the evidence for this remains correlational. The analysis of an individual's appraisal style may be informed by the hopelessness theory of Abramson, Metalsky and Alloy (1989). This theory suggests that the expectation of recurrence of negative events and perceived inability to respond is mediated by stable and global causal attributions about the event. However, the role of such an attributional model within PTSD theories remains unclear. A further theoretical field for the conceptualisation of appraisal mechanisms may be that of metacognitive theory and the exploration of beliefs about particular cognitive coping strategies.

1.6.6.5.5. Coping/thought control factors

Coping refers to complex processes aimed at reducing distress and frustration in difficult situations. Lazarus and Folkman (1984) define coping as a collection of cognitive and behavioural efforts to manage external or internal demands that are appraised as difficult or exceeding the resources of the person. Three stages of appraisal have been suggested: a primary appraisal in which an individual appraises a situation to be neutral, positive and benign or threatening. Secondary appraisal follows by evaluating the resources and strategies available to deal with the situation and their likely effectiveness in dealing with the difficulty. A final appraisal occurs once

change has taken place and takes into account subsequent changes in the environment. Thus, coping may be seen primarily as a cognitive process geared towards selecting and applying particular strategies to reduce distress.

Endler and Parker (1990) have proposed a three dimensional model of coping in which strategies are classified into three patterns: task/problem focussed coping, emotion focussed coping, and avoidance. Problem focussed coping refers to active efforts to deal with environmental stressors and requires the execution of an action plan. According to Wells and Matthews (1994), problem focussed coping is the most demanding of cognitive resources. Emotion focussed coping refers to attempts to modify the cognitive and emotional reactions to an event without directly dealing with external reality. An example of such a strategy may be accepting blame for an event. Avoidance strategies are utilised to prevent thoughts about the event from occurring. Wells and Matthews (1994) claim that this strategy is the least resource demanding. However, avoidant strategies are also associated with higher levels of distress (Bryant and Harvey, 1995). McFarlane (1988) found that fire-fighters who reported attempting to not think about the trauma of the bush fire experienced more psychiatric morbidity than those who did not attempt to suppress their thoughts about the event.

Wells and Matthews refined the concept of coping in order to account for limitations in the Lazarus and Folkman model. They consider the Lazarus and Folkman theory vague in its account of the information processing that

supports appraisal and coping. Similarly, Leventhal has pointed out the restrictions in limiting coping strategies to broad categories such as emotional and problem focussed coping.

Wells and Matthews (1994) have suggested that cognitive coping strategies may be re-conceptualised as 'thought control strategies'. This term applies to the choice of strategies available to an individual seeking to deal with distressing cognitive intrusions. The S-REF model of attentional processing (Wells and Matthews, 1994) posits emotional processing to be the product of cognitive-attentional processing whereby distressing information is attended to at length. Thought control strategies of rumination and threat monitoring maintain attention on distressing information and intrusions. The thought control strategies of rumination, worry, and threat monitoring may prevent new information from entering the information processing system and, therefore, the cognitive fear structure remains unchanged.

In order to operationalise the concept of Thought Control Strategies, (Wells and Davies, 1994) developed the Thought Control Questionnaire (TCQ) in order to obtain measures of different strategies used by individuals. The questionnaire measures individuals' use of strategies of social control, worry, distraction, punishment and re-appraisal. Research using the TCQ has demonstrated that individuals using worry as a thought control strategy have increased levels of emotional vulnerability. Reynolds and Wells (1999) found that people recovering from PTSD and/or depression were more likely to use

re-appraisal and distraction thought control strategies than self-punishment and worry.

Thus, the work of Wells and Matthews has refined the concept of 'coping' to include more cognitive coping strategies whilst developing the theoretical basis for consideration of the effect of 'coping' in the development and maintenance of emotional disorders. The role of thought control strategies in mediating emotional distress in acute medical trauma and their relation to social support factors have, to date, not been explored.

1.6.6.6. Review of PTSD models

The account of the models of PTSD above suggests that the psychosocial model represents an integration of previous models. In particular, it draws upon cognitive theories whilst allowing for the coping strategy, social and environmental factors that some studies have indicated to be of particular importance in PTSD. The model may be applied to the area of acute medical trauma by considering the patient's illness representations as part of their schema network alongside their coping strategies and social support factors in recovery from the illness.

However, the psychosocial model may be enhanced by integrating the thought control concepts from the S-REF model of Wells and Matthews. This integration would provide a more detailed description regarding the thought control and appraisal processes involved in maintaining PTSD and emotional

distress, and thus, describe the cognitive processes that mediate between schematic beliefs and emotional distress in more detail.

1.6.7. Treatment of PTSD

Many of the above theories have provided theoretical underpinnings for treatment approaches for PTSD patients. Often, there is not a clear delineation between theory and treatment approach as many treatments draw upon elements of different theories. However, current treatment strategies may be separated into three broad groups.

1.6.7.1. Exposure based treatments

Exposure based treatments for PTSD include systematic desensitisation (Wolpe, 1958),(Muse, 1986), flooding (Keane, Fairbank, Caddell and Zimmering, 1989), image habituation training (Vaughan and Tarrier, 1992) and prolonged exposure (Foa, Rothbaum, Riggs and Murdock, 1991). Most of these approaches involve exposure to the feared stimuli that triggered the PTSD reaction and aim to give the patient an opportunity to decrease the fear reaction to the feared stimuli. Such a reduction in fear reaction occurs when an individual recounts details of their personal trauma in a manner that permits re-experiencing of the affect associated with the trauma. As the re-experiencing occurs within a safer environment, new information regarding

their current more relaxed and safe situation, is assimilated into their memory of the traumatic event. Frequently, exposure is likely to be imaginal as the feared stimuli are of a dangerous and traumatic nature in real life.

1.6.7.2. Coping skills based treatments

Coping skills treatments aim to reduce psychological distress associated with memories of the trauma by teaching the individual new skills in reducing and managing their distress levels. Such skills include relaxation skills (to increase a sense of self-control), and basic cognitive skills such as distraction. Veronen and Kilpatrick (1983) developed a stress inoculation protocol for rape victims in which participants undertook training in three phases, an educational phase, a skills acquisition phase, and a skills application phase. Foa et al. (1991) have developed a programme (Stress Inoculation Training) that includes techniques such as progressive relaxation, thought stopping, breathing retraining and problem solving. The programme uses education and role-play to teach such skills and has been found to be successful in reducing symptomatology in rape victims.

1.6.7.3. Cognitive therapy

The main impetus behind cognitive therapy approaches for PTSD has come from the work of Beck on anxiety disorders (Beck, 1976), (Beck, Emery and

Greenberg, 1985). The foundation for Beck's theory of emotional distress is the concept that it is the individual's interpretation of events that causes psychological distress and not the event itself. Cognitive therapy, therefore, aims to restructure an individual's interpretations of distressing events in order to change the individual's emotional state.

Studies comparing behavioural approaches using exposure and cognitive therapy have not found any consistent superiority of one approach over the other (Marks, Lovell, Noshirvani, Livanou and Thrasher, 1988; Tarrier et al., 1999).

More recently, the work of Wells and Matthews (1994) has suggested that clinical attention should be broadened to examine cognitive processes as well as cognition content. They have developed a strategy, 'detached mindfulness', in which patients are encouraged to disengage rumination and active worry from intrusions. Wells (1990) has also developed an attentional training approach for the treatment of panic disorder. These techniques have not yet been researched in specific application with a PTSD population, although there is a theoretical rationale for their use.

1.6.7.4. Eye movement desensitization and reprocessing (EMDR)

Interest in EMDR has grown rapidly in recent years. The technique, discovered accidentally rather than theoretically derived (Shapiro, 1995),

involves a dual task procedure in which an individual is encouraged to recall traumatic memories whilst performing invoked eye movements. The protocol for delivering EMDR has evolved considerable since its discovery and there have been several studies attesting to the effectiveness of the treatment method (Vaughn et al., 1994; Wilson, Becker and Tinker, 1995). However, the underlying process by which the dual task procedure is believed to effect neurophysiological change remains imprecise and further research is required into its effectiveness and the psychological mechanism of change.

1.7. Review of the background to the study

1.7.1. Rationale for present study

The study aims to address some of the gaps in the current body of research into PTSD in an acute medical trauma population.

The above account of the psychosocial model of PTSD has shown that several variables are considered important in the development and maintenance of PTSD and emotional distress. The model is open to considerable interpretation regarding the operationalisation of several of the factors included, and lacks the specificity of more theoretically rigorous models (e.g. S-REF). Consideration of all the variables suggested by the psychosocial model within one study would require extensive interviewing or administration of questionnaires, a process that may be excessively demanding of recently traumatised participants and the current research

methodology. Therefore, the present study focuses upon the variables of social support, thought control and illness representations to ascertain whether these particular factors are predictive of PTSD symptoms in people who have suffered the traumatic medical events of myocardial infarction and subarachnoid haemorrhage. These factors have been chosen, as there is a body of research examining possible relationships between these factors in PTSD, emotional distress generally and health behaviour change.

Event cognition, appraisal, behavioural coping strategy, personality and environmental trigger factors are not addressed within this study.

Therefore, this study will address the recommendation from Berry (1998) to explore the risk factors for PTSD within a subarachnoid haemorrhage population, and supplement the work of Bennett and Brooke (1999) in exploring risk factors in addition to those of alexithymia, age and awareness for PTSD within a myocardial infarction sample. The work of Weinman et al. (2000) has explored the role of causal attributions within a myocardial infarction population, but the predictive role of the other factors of illness perceptions regarding PTSD symptomatology has not been examined to date.

As a previous study by McKenna, Willison et al. (1989) demonstrated that age and sex are also significant variables in outcome following a subarachnoid haemorrhage, this study will also examine these variables in determining predictors of PTSD in an acute medical trauma population.

As the majority of studies of predictor variables for PTSD in myocardial infarction have been retrospective, the study employs a prospective design to trace changes over time and overcome retrospective bias in the sample.

Greater understanding of risk factors for PTSD within an acute medical trauma population may serve to inform screening processes and rehabilitation services seeking to support those suffering emotional distress.

A further gap in current research is the study of changes in PTSD symptomatology and illness representations over time. Leventhal (1997) has alluded to the necessity to examine changes in illness representations over time and previous PTSD studies have shown that clinical presentation following trauma may change dynamically. As Weinman et al. (1996) have pointed out that the test-retest reliability was greater for some of the components of the IPQ (the consequences and cure/control factors) compared to others the timeline and identity factors), there is a need to explore these changes over time in greater detail. Therefore, this study seeks to address the issue of changes in PTSD symptomatology over time and changes in illness representations over time for acute medical trauma. This is of particular importance in the immediate aftermath of an acute medical trauma when clinical staff may be most concerned about a patient's emotional state. Rehabilitation services may obtain greater insight into the likely time course of a person's emotional recovery following an acute medical trauma.

Although the work of McKenna et al. (1989) has supported the case for considering the experience of the two populations of subarachnoid haemorrhage and myocardial infarction patients to be homogenous, it is considered necessary to explore the similarities of these two groups more thoroughly in respect of the illness representations they hold. Therefore, this study aims to compare the illness representation profiles of the two groups to address this issue of homogeneity under the label 'acute medical trauma'.

In order to supplement previous studies, further data regarding the incidence of PTSD specifically, and emotional distress generally, within the two samples will also be obtained.

1.7.2. Aims of present study

The study aims are:-

- To examine the relationship between illness representations and PTSD in an acute medical trauma population.
- To examine changes in PTSD symptomatology and illness representations in this population over time.
- To determine whether basic demographic factors, illness representations, social support, and thought control strategies are predictor variables for PTSD within an acute medical trauma population.
- To determine the levels of PTSD, anxiety and depression in an acute medical trauma population.

1.8. Hypotheses

1.8.1. Primary hypothesis

There will be an association between PTSD symptomatology and illness representations for both myocardial infarction and subarachnoid haemorrhage samples.

1.8.2. Subsidiary hypotheses

1.8.2.1. The level of PTSD symptomatology will change over time for both myocardial infarction and subarachnoid haemorrhage samples.

1.8.2.2. Illness representations will change over time for both myocardial infarction and subarachnoid haemorrhage samples

1.8.2.3. There will be a similar profile of illness representations for the myocardial infarction and subarachnoid haemorrhage samples.

1.8.2.4. Illness representations will be a predictor of PTSD.

1.8.2.5. Perceived levels of social support will be a predictor of PTSD.

1.8.2.6. Worry and punishment thought control strategies will be predictors of PTSD.

1.8.2.7. Levels of distress generally and PTSD specifically will be found in myocardial infarction and subarachnoid haemorrhage samples.

CHAPTER 2: METHOD

2. Method

2.1. Ethical approval

The current study was carried out at two sites. Myocardial infarction patients were recruited from hospital Trust one (Trust 1) and subarachnoid haemorrhage patients from hospital Trust two (Trust 2). Ethical approval for the research was granted by both Salford and Trafford and North Manchester Local Research Ethics Committees (see 6.1). The process of acquiring ethical approval involved the development of guidelines on dealing with emotional distress in participants. The Research and Development managers of both Trusts gave approval for the study to be conducted at their hospitals (see 6.2). Consent to approach myocardial infarction patients at Trust 1 was given by consultant physicians and consultant cardiologists through an opt in consent form (see 6.3). At Trust 2, the clinical director for neurosciences gave consent on behalf of all the consultant neurosurgeons and consultant neuroradiologists for the researcher to approach subarachnoid haemorrhage patients.

2.2. Preliminary investigations and preparatory work

2.2.1. Meetings with Trust 1 and Trust 2 staff

Before data collection began, meetings were held with ward managers from the coronary care and cardiology wards at Trust 1, and the ward managers

from the neurosurgery wards at Trust 2. The research was explained to the ward managers. Advice was sought regarding potential numbers of patients for recruitment and the means of recruiting participants from the wards. Confirmation and modification of initial ideas regarding inclusion and exclusion criteria was also discussed. Ward information packs were developed for each of the wards involved in the study, one for Trust 1 (6.4) and one for Trust 2 (6.5).

Ward managers suggested that up to 40 patients from each site may be recruited within the time frame for the study given the agreed inclusion and exclusion criteria.

It was agreed that the researcher would contact each of the relevant wards by telephone or by visit twice per week, and discuss potential participants with the nurse in charge.

2.3. Design

A longitudinal cohort design was employed. Data collected at time one (T1) was compared to data collected at time two (T2) and time three (T3).

T1 was specified as being within two weeks of the patient's hospital admission and whilst the patient was still an inpatient. T2 was specified as being between five and seven weeks after the admission and T3 was specified as being between eleven and fourteen weeks after the admission.

2.4. Inclusion and exclusion criteria

All participants were adults recruited from two hospitals in the North of England. The participants were inpatients receiving care for either a subarachnoid haemorrhage or a myocardial infarction.

To be included within the study patients had to meet the following inclusion criteria:

1. They must have a clinical diagnosis of subarachnoid haemorrhage or myocardial infarction.
2. They must be medically stable.
3. They must speak and understand English.
4. They must be fully orientated to the ward.
5. Nursing staff must consider the patient competent to give consent, even if there is mild cognitive disturbance.
6. They must be over 18.

Individuals were excluded from the study if they met one or more of the following exclusion criteria:

1. They are currently receiving intervention by a psychiatrist or psychologist for mental health problems.
2. They have a previous history of severe mental health problems (e.g. psychotic disorder).
3. Neurological deficits of dysphasia and/or hemiplegia are present.

4. They have had multiple (more than two) myocardial infarctions or severe premorbid cardiac disease.

Exclusion criteria 1 and 2 were included to minimise the impact of the potentially confounding variable of previous psychological morbidity in measuring change in participants' outcome during the study. Exclusion criterion 3 was included to ensure that subarachnoid haemorrhage participants were competent to consent to participate in the study, provide consistent responses during interview and tolerate the interview procedure whilst an inpatient. Finally, exclusion criterion 4 was included to reduce the risk of including myocardial infarction participants with cognitive deficits subsequent to multi-infarct dementia.

2.5. Sample size

As the study potentially involved a large number of factors for the multiple regression analysis, it was decided that approximately 60 participants would be required in order to produce an adequate model. This estimated sample size was confirmed by a power calculation for correlational analyses in which it was found that a sample size of 60 would be required to achieve a correlation of 0.25. It was unlikely that 60 participants would be recruited solely from either the myocardial infarction group or the subarachnoid haemorrhage group within the period of the study, therefore, it was considered necessary to include both groups simultaneously. The two groups were considered to be similar through the shared experience of a life

threatening, internal, medical event. It was acknowledged that there was substantial risk of a high drop out rate for postal questionnaire returns for participants experiencing trauma (e.g. Holeva et al, 2001). Therefore, attempts were made to maximise the sample size by contacting the relevant wards twice per week.

2.6. Data collection

2.6.1. Demographic information

Details of the patients' sex and age were recorded. For the subarachnoid haemorrhage patients, details regarding the site of the aneurysm and procedure undertaken were recorded. For the myocardial infarction patients, details of the procedure undertaken were recorded. Participants were asked if they were receiving treatment from a psychologist or psychiatrist for mental health problems or had received mental health services in the past.

2.6.2. Measures at T1, T2, and T3

The following measures were administered at all three time points within the study to measure PTSD, and illness representation variables.

2.6.2.1. Illness Perception Questionnaire – Revised (IPQ-R)

The Illness Perception Questionnaire – Revised (IPQ-R) (Moss-Morris et al., 2002) is a scale used to measure key components of patients' representations of their illness. The original Illness Perception Questionnaire (IPQ) (Weinman et al., 1996) was designed as a quantitative measure of the five components of illness representation in Leventhal's self-regulatory model (causal attributions, identity, consequences, timeline, and cure/control). The revised version arose from a need to address problems with the psychometric properties of two factors (timeline, and cure/control) and to include additional factors (emotional representation and illness coherence). Factor analysis of the 38 general questionnaire items within the IPQ – R has verified a structure including the following factors: identity, timeline (acute/chronic), timeline (cyclical), consequences, personal control, treatment control, emotional representations and illness coherence. For the purposes of this study, these factors will be referred to collectively as IPQ-R General Factors. Further factor analysis of the 15 causal attribution questionnaire items has supported a structure for causal attributions according to the following factors: psychological attributions, risk factor attributions, immunity attributions and chance attributions. For the purposes of this study, these factors will be referred to collectively as IPQ-R Causal Attribution Factors. The validation of the IPQ – R involved patients from a range of medical conditions including rheumatoid arthritis, myocardial infarction, chronic and acute pain, asthma and HIV patients.

All 12 factors of the IPQ - R were used at T2 as predictor variables for PTSD at T3 within a regression model. All 12 factors were also used in correlational analyses to explore associations with measures of PTSD symptomatology at T1, T2, and T3, and in analyses of change over time across all three time points. A copy of the IPQ – R can be found in Appendix 6.6.

2.6.2.2. Davidson Trauma Scale (DTS)

The Davidson Trauma Scale (DTS) (Davidson, 1996) is a self-rated symptom scale for PTSD. The scale consists of 17 items rated for frequency and severity. These items are computed into sub-scale scores for three symptom clusters: intrusion, avoidance/numbing and hyperarousal. The scale measures symptoms that are diagnostic of PTSD according to DSM IV (APA, 1994). High scores on each of the sub-scales are indicative of high levels of symptomatology. An aggregate Total PTSD score is also computed.

Reliability for the DTS has been reported by Zlotnick, Davidson, Shea and Pearlstein (1996). (Cronbach's ά for the DTS Total scale = 0.93, for the intrusive, avoidant and arousal sub-scales 0.85, 0.83, and 0.87 respectively). The test-retest reliability was established by Davidson (1996) to be 0.86.

The DTS was adapted to specify the trauma (i.e. myocardial infarction or subarachnoid haemorrhage) at the top of the questionnaire. The cut-off score for caseness was taken to be 40, as specified by Davidson (1996).

The DTS was used at T1, T2, and T3 as an outcome measure. The DTS may not be used as a diagnostic measure at T1 because a diagnosis of PTSD within one month of the trauma is not permitted (1.6.2.1). Thus, the use of the DTS at this time is simply as a measure of symptomatology.

2.6.3. Measures at T2, and T3

The following measures were administered at T2 and T3 to measure anxiety, depression outcomes, and thought control strategies and perceived social support predictor variables.

2.6.3.1. Hospital Anxiety and Depression Scale (HADS)

The Hospital and Anxiety Scale (HADS) (Zigmond and Snaith, 1983), is a 14-item scale used to measure psychological morbidity in terms of anxiety and depression. The scale was designed specifically for use in non-psychiatric hospital departments and aims to exclude references to physical symptoms which might be attributable to physical illness (e.g. headaches and dizziness). The scores on both the anxiety and depression subscales range from 0 to 21, and the higher the score, the more psychological morbidity experienced. Patients scoring more than 11 on either the anxiety or the depression subscale may be regarded as moderate cases (Snaith and Zigmond, 1994).

The reliability of the HADS has recently been established by Crawford, Henry, Crombie and Taylor (2001). Their study reported reliabilities (Cronbach's ά) for the anxiety, depression and combined scales of 0.82, 0.77 and 0.76 respectively. The correlation between the two subscales of anxiety and depression was found to be highly significant ($p<0.001$).

The HADS was used at both T2 and T3 as an outcome measure. A copy of the HADS can be found in Appendix 6.7.

2.6.3.2. Thought Control Questionnaire (TCQ)

The Thought Control Questionnaire (TCQ) (Wells and Davies, 1994), is a 30 item self-report questionnaire used to measure the extent to which individuals use cognitive thought control strategies for dealing with distressing intrusive thoughts. The questionnaire comprises of five sub-scales measuring cognitive thought control strategies of distraction, social control, worry, punishment, and re-appraisal. Scores for each sub-scale range from 6 to 24.

Reliability for the TCQ has been reported by Wells and Davies (1994). (Cronbach's ά for the sub-scales range 0.64 – 0.79, test-retest range 0.68 – 0.83).

The sub-scales of the TCQ were used at T2 as predictor variables for PTSD at T3 in multiple regression calculations. A copy of the TCQ can be found in Appendix 6.8.

2.6.3.3. Social Support Inventory (SSI)

The Social Support Inventory (SSI) (Holeva, 1998) was designed to measure participants' perceptions of support within their psychosocial environment. It is derived from the Perceived Criticism Scale (PCS) (Hooley and Teasdale, 1989). The scale includes five items relating to perceived criticism towards and from a key significant other, satisfaction with practical and emotional support and prediction of future support.

Factor analysis of the SSI demonstrated that the 2 perceived criticism items and the 3 social support items constitute 2 separate factors (correlation of -0.157 between the two factors), accounting for 79% of the total variance. The scale was demonstrated to have acceptable internal consistency (Cronbach's ά: social support 0.83; perceived criticism 0.67) and test-retest reliability (social support 0.55; perceived criticism 0.79; on a sample of 45 participants tested 4 weeks apart).

The 5 items of the SSI were used at T2 as a predictor variables for PTSD at T3 in multiple regression calculations. A copy of the SSI can be found in Appendix 6.9.

2.7. Procedure

Potential participants were recruited by the researcher contacting identified wards at both Trust 1 and Trust 2 twice weekly. Nursing staff who were familiar with the inclusion/exclusion criteria for the study were requested to approach identified patients on the researcher's behalf to request their participation within the study and confirm their opinion of the patient's competence to consent.

When patients fulfilling the inclusion/exclusion criteria were identified by the nursing staff and notified to the researcher, the patient was approached and the research study was explained to them in detail. Patients were given an information sheet to read (see 6.10) and given opportunities to ask questions. Patients were also asked if they wished to delay giving a decision regarding participation until they had spoken with either family members or friends. Those patients who consented to participate within the study immediately completed a consent form (see 6.11) and the researcher then administered the questionnaires for T1. Patients who requested a delay before consenting were approached within two days. If they agreed to participate, they completed a consent form and the researcher administered the questionnaires for T1.

Relevant demographic and medical information was obtained from the medical notes after the T1 assessment.

A letter was sent to the participant's GP informing them of their inclusion within the study and an entry was made in the medical notes.

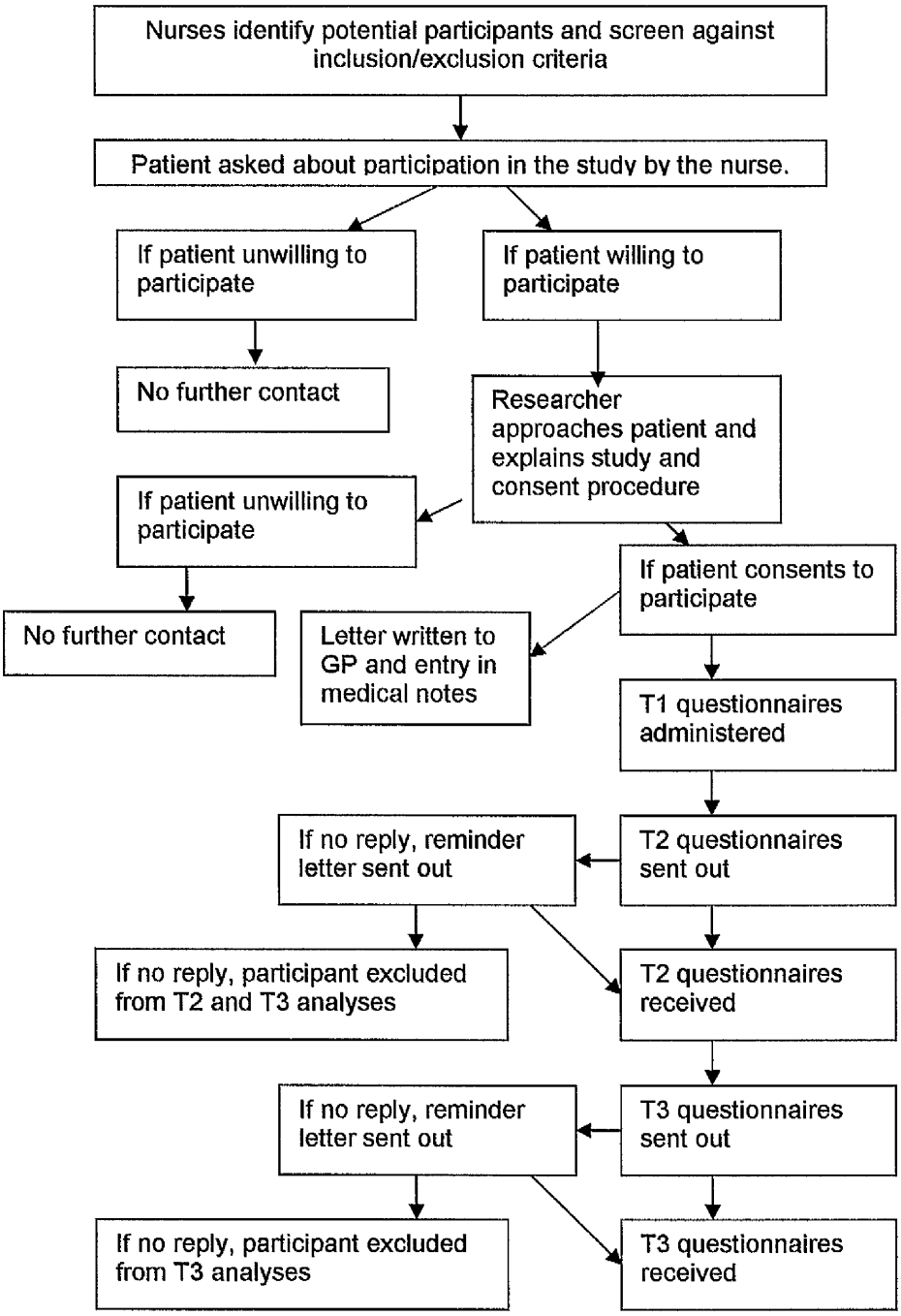
The five T2 assessment questionnaires were sent out by post together with a covering letter (see 6.12), a further copy of the information sheet and the patient consent form. Participants who failed to return the questionnaires within two weeks of posting the questionnaires were sent a reminder letter (see 6.13).

The five T3 assessment questionnaires were sent out by post together with a covering letter (see 6.14). Participants who failed to return the questionnaires within two weeks of posting the questionnaires were sent a reminder letter (see 6.15).

Where possible, incomplete data for T2 and T3 questionnaires was completed via a telephone call to the participant.

The procedure for the study is represented diagrammatically in Figure 6.

Figure 6: Study procedure



2.8. Statistical Analysis

Statistical analyses were carried out using SPSS version 11 for Windows.

As the IPQ-R had not been used with a subarachnoid haemorrhage population before, a factor analysis was carried out on the IPQ-R data from T1, T2 and T3 for the subarachnoid haemorrhage sample and the myocardial infarction sample together to confirm that the proposed factorial structure applied to the combined acute medical trauma sample.

Correlational analyses, ANOVA and Friedman tests, T-tests and Mann-Whitney U Tests were carried out to inform the research hypotheses in 1.8.

Finally, the data were analysed using three preliminary multiple regression analyses for the PTSD outcome measure at T3 (DTS3) for each of the predictor variable questionnaires (IPQ – R, TCQ and SSI) at T2. In the TCQ and SSI multiple regression analyses, the potentially confounding variables of age and sex were entered as control variables in the initial step of the analyses. A final multiple regression model was then analysed using predictor variables that had been found to be significant in the three preliminary multiple regressions.

Descriptive statistics for psychological morbidity at each of the three time points were also generated.

CHAPTER 3: RESULTS

3. Results

3.1. Overview of Chapter Three

This chapter consists of four parts. The first part (3.2) will provide information on the number of individuals recruited to the study and summarises the attrition process by which the initial recruitment sample was reduced at the two follow-up data collection time points (T2 and T3). For the purposes of summarising the recruitment data at this stage, data will be presented for the two medical condition samples separately, and for the combined acute medical trauma sample.

The second part of this chapter (3.3) will provide basic demographic and psychological morbidity information regarding the two medical condition samples at the recruitment stage (T1). The basic demographic and psychological morbidity data will be analysed to explore potential differences between those participants who remained in the study and those who did not.

The third part of this chapter (3.4) will present the results of a factor analysis of the IPQ-R for the combined acute medical trauma sample at T1. Although the IPQ-R has been used with a myocardial infarction sample previously (Petrie et al., 1996; Weinman et al., 1996; Weinman et al., 2000; Moss-Morris et al., 2002), it has not been used with a subarachnoid haemorrhage sample. Therefore, the factor analysis was undertaken in order to assess the appropriateness of using the IPQ-R for the two samples of subarachnoid

haemorrhage and myocardial infarction patients that were combined as an acute medical trauma sample within this study.

Finally, the fourth part of the chapter (3.5) will present the results of statistical analyses undertaken to test the research hypotheses specified in 1.8. Statistical advice regarding these analyses was sought from medical statisticians and researchers within the field of PTSD and Clinical Health Psychology. The statistical analyses was also informed by previous research that utilised similar, prospective designs to the current study (e.g. (Holeva et al., 2001; Lemon, 2001). The statistical procedure will be outlined for each analysis.

3.2. Results Part one

3.2.1. Sample size: subarachnoid haemorrhage

The subarachnoid haemorrhage sample was recruited using an opportunistic sampling method. Using the recruitment procedure outlined in 2.7, 41 patients were identified for inclusion within the study from Trust 2. Following an introduction to the study and discussion of the consent procedure with the potential participants, a subarachnoid haemorrhage sample size of 38 was recruited to the study at T1 (3 patients declined to participate). Of the 38 participants, 7 (18%) did not return questionnaires at T2, 4 (11%) returned questionnaires at T2 and did not return questionnaires at T3 and 27 (71%) completed all the questionnaires at T2 and T3.

3.2.2. Sample size: myocardial infarction

The myocardial infarction sample was also recruited using an opportunistic sampling method. Using the recruitment procedure outlined in 2.7, 32 patients were identified for inclusion within the study. Following an introduction to the study and discussion of the consent procedure with the potential participants, a sample size of 30 was recruited to the study at T1 (2 patients declined to participate). Of the 30 participants, 9 (30%) did not return questionnaires at T2, 4 (13%) returned questionnaires at T2 and did not return questionnaire at T3 and 17 (57%) completed all the questionnaires at T2 and T3.

3.2.3. Total sample size

The total acute medical trauma sample recruited at T1 was 68. Of these, 38 (56%) were subarachnoid haemorrhage patients and 30 (44%) were myocardial infarction patients. Of the 68 participants, 16 (23%) did not return questionnaires at T2, 8 (12%) completed questionnaires at T2 but not at T3, and 44 (65%) completed questionnaires at both T2 and T3.

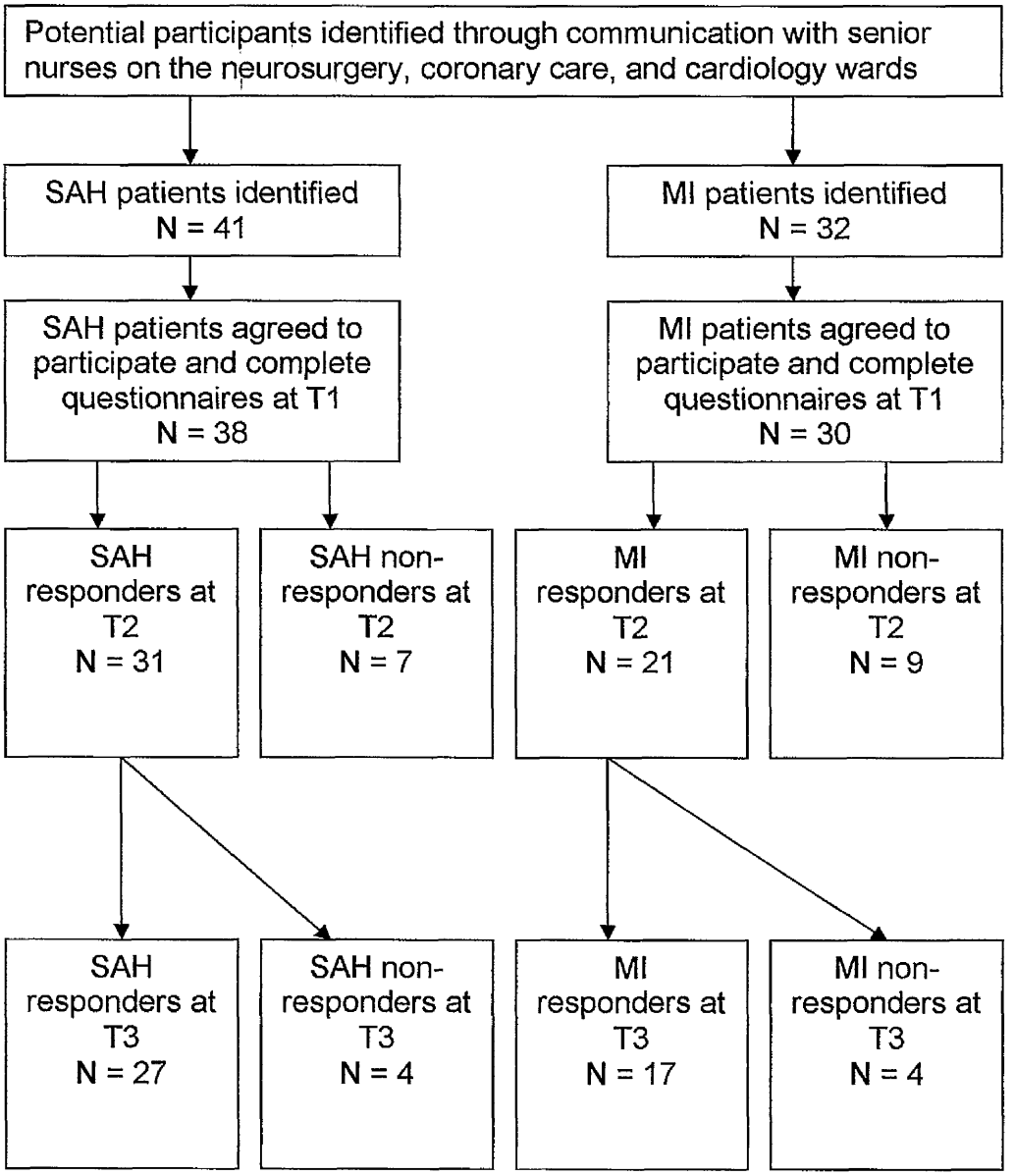
The acute medical trauma sample statistics are shown below in Table 1.

Table 1: Sample statistics by number of questionnaires completed

		Condition		Total
		MI	SAH	
Questionnaires completed at T1 alone	N	9	7	16
	% within Condition	30.0%	18.4%	23.5%
Questionnaires completed at T1 and T2 alone	N	4	4	8
	% within Condition	13.3%	10.5%	11.8%
Questionnaires completed at T1, T2 and T3	N	17	27	44
	% within Condition	56.7%	71.1%	64.7%
Total	N	30	38	68
	% within Condition	100.0%	100.0%	100.0%

Figure 7 demonstrates the recruitment and attrition rates for the study.

Figure 7: Flow diagram demonstrating recruitment and attrition rates for the study



SAH = subarachnoid haemorrhage.

MI = myocardial infarction.

3.3. Results Part two

3.3.1. Demographic information

Sixty-eight (68) participants were included in the study at T1. Of the total sample, 41 (60%) were male and 27 (40%) female and the mean age was 54.21 (standard deviation, sd = 12.3). Research literature detailing the demographic profiles of the two medical conditions indicated that the gender and age profiles were likely to be different for the two samples. Therefore, analysis of the demographic information for the two sample groups was conducted to detect any significant differences between them.

Of the subarachnoid haemorrhage sample, 17 (45%) were male and 21 (55%) female and the mean age was 48.2 years (standard deviation, sd = 10.76). Ages of the subarachnoid haemorrhage participants ranged from 32 to 66 years and the mean +/- 2sd computes a range of 27 to 70 indicating that the data was not skewed. The result of the One-sample Kolmogorov-Smirnov test for the age distribution of the subarachnoid haemorrhage sample was not significant (z = 0.926; p = 0.371) confirming that the age data is normally distributed and, therefore, a parametric test for this sample would be appropriate. The myocardial infarction sample presented with a different demographic profile. Of this sample, 24 (80%) were male and 6 (20%) were female and the mean age was 61.8 years (s.d. = 9.72). Ages of the myocardial infarction participants ranged from 44 to 83 years and the mean +/- 2sd computes a range of 81 to 42 indicating that the data was not

skewed. The result of the One-sample Kolmogorov-Smirnov test for the age distribution of the myocardial infarction sample was also not significant (z = 0.483; p = 0.974) confirming that the age data is normally distributed and, therefore, a parametric test for this sample would be also be appropriate.

The result of the One-sample Kolmogorov-Smirnov test for the age distribution of the total acute medical trauma sample was not significant (z = 0.753; p = 0.623) confirming that the age data is normally distributed and, therefore a parametric test would be appropriate for the total acute medical trauma sample.

3.3.2. Analysis of demographic differences between the two medical condition samples

The results of the t test conducted to assess age differences between the two medical condition sample groups was significant (t (66) = 5.415; p < 0.001). Fisher's Exact test (two sided) analysis of the gender differences between the two samples was also found to be significant (Fisher's Exact; p = 0.006). Thus, the two sample groups differed significantly in respect of the gender composition and mean age. The results of the statistical tests demonstrating significant differences between the two groups in respect of gender and age are shown in Table 2.

Table 2: Analysis of demographic differences between the two medical condition samples

Demographic variables	SAH Participants (N = 38)	MI Participants (N = 30)	t test/Chi-square test
Mean age (s.d.)	48.2 (10.76)	61.8 (9.72)	t = 5.415 p<0.001***
Sex: **Male** **female**	 17 (45%) 21 (55%)	 24 (80%) 6 (20%)	 Fisher's Exact test p = .006**

3.3.3. Demographic differences between responders and non-responders at T2

As 24% of the total acute medical trauma sample did not respond at T2, an analysis of the demographic differences between T2 responders and T2 non-responders was conducted. The analysis compared all participants who completed questionnaires at T1 and T2 alone, and at T1, T2 and T3 with participants who completed questionnaires at T1 alone according to the recorded demographic variables of gender and age.

To explore gender differences between responders and non-responders at T2 a Fisher's Exact Test (two-sided) was conducted. The result was not significant (Fisher's Exact; p = 0.389). 50% of non-responders at T2 were male and 63.5% of responders at T2 were male.

To explore age differences between responders and non-responders at T2, a t-test was conducted (the normal distribution of the age variable data had been demonstrated in section 3.3.1). The result was not significant ($t(66) = -1.343$; $p = 0.184$). The mean age of non-responders at T2 was 57.81 and the mean age of responders at T2 was 53.11.

Therefore, the results of these analyses indicate that there were no significant differences between the responders and non-responders at T2 in terms of gender and age.

3.3.4. Demographic differences between responders and non-responders at T3

35% of the total acute medical trauma sample did not respond at T3, and to explore this, an analysis of the demographic differences between T3 responders and T3 non-responders was conducted. The analysis compared all participants who completed questionnaires at T1, T2 and T3, with participants who completed questionnaires at T1 alone, and T1 and T2 alone according to the recorded demographic variables of gender and age.

To explore gender differences between responders and non-responders at T3 a Fisher's Exact Test (two-sided) was conducted. The result was not significant (Fisher's Exact; $p = 0.605$). 54.2% of non-responders at T3 were male and 63.6% of responders at T3 were male.

To explore age differences between responders and non-responders at T3, a t-test was conducted. The result was not significant (t(66) = -1.726; p = 0.089). The mean age of non-responders at T3 was 57.65 and the mean age of responders at T3 was 52.34.

Therefore, the results of these analyses indicate that there were no significant differences between the responders and non-responders at T3 in terms of gender and age.

3.3.5. Psychological morbidity differences between responders and non-responders at T2

Further analysis was conducted in order to examine possible bias introduced by differences in psychological morbidity between responders and non-responders at T2. The analysis compared the DTS scores at T1 (DTS1) of participants who completed questionnaires both at T1 and T2 alone and at T1, T2, and T3, with the DTS1 scores of participants who completed questionnaires at T1 alone. The analysis was carried out on the total acute medical trauma sample.

The result of the One-sample Kolmogorov-Smirnov test for the DTS1 scores of the total acute medical trauma sample was not significant (z = 0.968; p = 0.306) confirming that the DTS1 data is normally distributed and, therefore, a parametric test for this variable would be appropriate for the whole sample.

To investigate DTS1 score differences between responders and non-responders at T2, a t-test was conducted. The result was not significant ($t(66) = 0.420$; $p = 0.676$). The mean DTS1 score of non-responders at T2 was 20.81 and the mean DTS1 score of responders at T2 was 22.98.

Therefore, the results of these analyses indicate that there were no significant differences between the responders and non-responders at T2 in terms of the levels of their PTSD symptomatology.

3.3.6. Psychological morbidity differences between responders and non-responders at T3

A final analysis of psychological morbidity differences between responders and non-responders compared all participants who completed questionnaires at T1, T2, and T3 with participants who completed questionnaires at both T1 and T2 alone, according to the recorded psychological morbidity variables of PTSD as measured by the DTS at T2 (DTS2) and anxiety and depression as measured by the HADS at T2 (HADSAnx2 and HADSDep2). The analysis was carried out on the total acute medical trauma sample.

The results of the One-sample Kolmogorov-Smirnov tests for the DTS2, HADSAnx2 and HADSDep2 scores for the 52 participants from both sample groups who completed T2 questionaires failed to reach significance ($z = 0.933$; $p = 0.349$, $z = 0.991$; $p = 0.280$ and $z = 0.908$; $p = 0.382$). These

results confirm that the psychological morbidity variables of DTS2, HADSAnx2, and HASDSDep2 are normally distributed and, therefore parametric tests for these variables would be appropriate for the whole sample.

To investigate DTS2 score differences between responders and non-responders at T3, a t-test was conducted. The result was significant ($t(50) = -2.065$; $p = 0.044$). The mean DTS2 score of non-responders at T3 was 51.63 and the mean DTS2 score of responders at T3 was 30.34.

T-tests were also conducted for the HADSAnx2 and HADSDep2 variables. The results were not significant ($t(50) = -1.310$; $p = 0.196$ and $t(50) = -1.252$; $p = 0.216$) respectively. The mean HADSAnx2 score of non-responders at T3 was 9.75 and the mean HADSAnx2 score of responders at T3 was 7.16. The mean HADSDep2 score of non-responders at T3 was 9.25 and the mean HADSDep2 score of responders at T3 was 7.59.

Therefore, the results of these analyses indicate that there were significant differences between the responders and non-responders at T3 in terms of the levels of their PTSD symptomatology at T2. Non-responders had significantly higher levels of PTSD at T2 than responders did. However, significant differences in anxiety and depression levels between the responders and non-responders at T3 were not detected.

3.3.7. Summary of section 3.3

The above analyses have detected differences in age and gender profiles between the two sample groups. The subarachnoid haemorrhage group has a significantly lower mean age and lower male: female ratio than the myocardial infarction sample. Consequent to the analysis of the demographic and psychological morbidity variables between responders and non-responders at T2 and T3, it appears that there are no significant differences between the two groups of responders and non-responders at T2. The only significant difference between responders and non-responders at T3 is that of level of PTSD recorded at T2.

3.4. Results Part three

3.4.1. Factor analysis of the IPQ-R at T1

A myocardial infarction sample had been used in a study to develop and refine the IPQ-R (Moss-Morris et al., 2002). However, this measure has not been used previously with a subarachnoid haemorrhage sample. Therefore, it was considered necessary for the purposes of this study, to carry out factor analyses on the IPQ-R for both sample groups together in order to assess the validity of its use with the combined acute medical trauma sample. The use of heterogenous samples in exploratory factor analysis has been advocated by Kline (1994). It is assumed that, if similar factor profiles to the original IPQ-R validation study are detected for the combined sample within this study, then the IPQ-R may be considered a valid measure to use for this study. The factor analysis was carried out purely to provide support for the use of the IPQ-R for the total acute medical trauma sample. The sample size (N = 68) is considered inadequate for carrying out a robust factor analysis of the IPQ-R data.

According to the validation study of Moss-Morris et al. (2002), the IPQ-R is structured according to two groups of factors, IPQ-R General factors (including identity, timeline (acute/chronic), timeline (cyclical), consequences, personal control, treatment control, illness coherence and emotional representation factors) and IPQ-R Causal Attribution factors (including psychological attributions, risk factor attributions, immunity

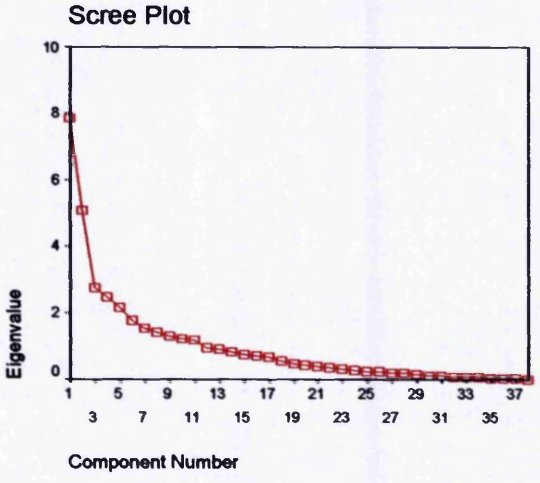
attributions and chance attributions factors). Therefore, in order to assess the validity of the IPQ-R for the acute medical trauma sample used in this study, two factor analyses were conducted.

The factor analyses were carried out on IPQ-R data at T1 for the 68 participants from the acute medical trauma sample.

The first factor analysis explored the IPQ-R General factor structure as specified by Moss-Morris et al. (2002). The Moss-Morris et al.(2002) study used a principal components analysis with varimax rotation selecting items with correlations greater than 0.45 for loading on to one factor. The result identified a 7-factor structure for the IPQ-R General questionnaire items. (The identity factor was excluded from this analysis as it was measured using a separate scale). Therefore, a similar method was used in this study. However, as the purpose of this analysis was to assess whether a factor structure for the acute medical trauma sample similar to that of the IPQ-R validation study would be obtained, the analysis specified a priori that 7 factors were to be extracted. The current analysis also differed in selecting items with correlations greater than 0.4 for loading on to one factor. This is considered a generally accepted cut-off (Everitt, 1994) and accommodates the low number of participants used within this factor analysis. This correlation value means that, as a minimum, only 16% of one variable would be explained by the other and, therefore, smaller correlations may be discounted. This statistical procedure allows all factors to be rotated to the most appropriate structure that allocates individual items to separate factors.

As the specification of 7 factors was determined by the prior findings of the Moss-Morris et al. (2002) study, an initial examination of the scree plot was carried out to ensure that such an assumption was valid (see Figure 8).

Figure 8: Scree plot for the principal components analysis of the IPQ-R General questionnaire items



The scree plot indicates that setting the cut-off point for the number of factors required at 7 was acceptable as the eigen values for subsequent factors were shown to reduce at a lesser rate.

The Kaiser-Meyer-Olin measure of sampling adequacy (KMO) was acceptable at 0.582 although this result is mediocre and suggests the possibility that correlations between variables may not be explained by other variables. Bartlett's test of Sphericity was calculated and found to be

significant (Approx Chi-Square (703) = 1813.645; $p < 0.001$), thus increasing the likelihood that a meaningful number of factors representing relationships between sets of inter-related factors may be extracted.

Closer examination of the extracted 7 factor solution demonstrated that the first factor accounted for 12.80% of the variance, the second factor accounted for 11.63% and the third factor contributed a further 9.59%. The final four factors contributed between 8.54% and 5.24% of the variance each, a total of 28.25% of the variance. Thus, the 7-factor model accounted for a total of 62.27% of the variance amongst the IPQ-R General variables. Table 3 shows the results of the principal components analysis with varimax rotation for the specified seven factors.

Table 3: Results of principal components analysis with varimax rotation for IPQ-R General questionnaire items

Component	Eigen Value	% Variance	Cumulative %
1	4.865	12.802	12.802
2	4.418	11.627	24.428
3	3.644	9.590	34.018
4	3.245	8.538	42.556
5	2.833	7.455	50.011
6	2.667	7.019	57.030
7	1.989	5.235	62.265

Table 4 shows the correlations between the questionnaire items and the extracted factors above 0.40. In order to compare these results with the factor analysis conducted by Moss-Morris et al. (2002), the questionnaire items that match the factor identification obtained in the validation study are highlighted in red.

Table 4: Rotated component matrix for IPQ-R General Questionnaire

Items

	Component						
	1	2	3	4	5	6	7
IPQ1X1	.843						
IPQ1.2	.704						
IPQ1.3	.885						
IPQ1X4	.849						
IPQ1.5	.574						
IPQ1.6							
IPQ1.7							.529
IPQ1X9	.595						
IPQ1.10							.562
IPQ1.11							
IPQ1.13							.631
IPQ1.17				.522			
IPQ1.18				.743			
IPQ1.20				.536			
IPQ1X21				.591			
IPQ1.22				.785			
IPQ1X23				.748			
IPQ1X26						-.504	
IPQ1X27						.650	
IPQ1.28							
IPQ1.29						.656	
IPQ1.30							
IPQ1X31						.668	
IPQ1X32		.820					
IPQ1X33		.848					
IPQ1X34		.893					
IPQ1X35		.867					
IPQ1.36		.672					
IPQ1.37							
IPQ1.38					.675		
IPQ1.39					.782		
IPQ1.41					.692		
IPQ1.44			.670				
IPQ1.45			.744				
IPQ1.46			.672				
IPQ1X47							
IPQ1.48			.759				
IPQ1.50			.788				

Extraction Method: Principal Component Analysis.
a Rotation converged in 11 iterations.

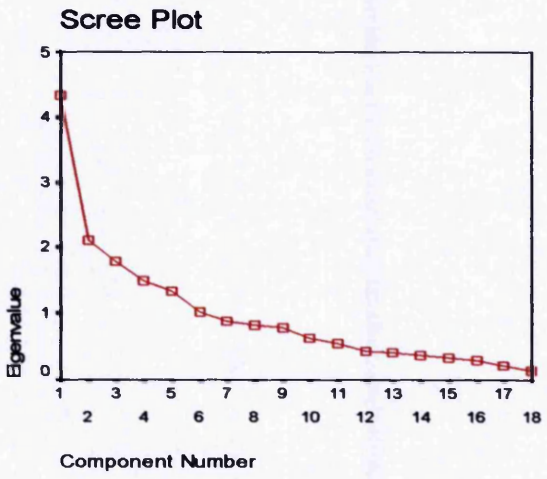
From this analysis, it can be seen that 30 out of the 38 IPQ-R General questionnaire items correspond to the factors identified in the Moss-Morris et al. (2002) study. Six (6) questionnaire items did not enter the factor structure as their correlation statistics were less than 0.40 and 2 questionnaire items were loaded onto factors other than the ones demonstrated in the validation study.

From this exploratory analysis, it may be concluded that the results of the factor analysis of the IPQ-R General questionnaire items for this sample approximate those of the initial validation study and, therefore, that the measure may be considered valid for this sample.

The second factor analysis explored the IPQ-R causal attributions factor structure as specified by Moss-Morris et al. (2002). The validation study had used the same statistical method for the analysis of this group of questionnaire items. The result was the identification of a 4-factor structure for the IPQ-R causal attribution questionnaire items. Therefore, for the purposes of this study, the analysis specified a priori that 4 factors were to be extracted and items would be selected if they had correlations greater than 0.40 for loading on to one factor.

As the specification of 4 factors was determined by the prior findings of the Moss-Morris et al. (2002) study, an initial examination of the scree plot was carried out to ensure that such an assumption was valid (see Figure 9).

Figure 9: Scree plot for the principal components analysis of the IPQ-R Causal Attribution questionnaire items.



The scree plot indicates that choosing the cut-off point for the number of factors required at 4 was acceptable as the eigen values for subsequent factors were shown to reduce at a lesser rate. However, the rate of reduction was greater than for the General IPQ-R factors analysis.

The Kaiser-Meyer-Olin Measure of sampling adequacy (KMO) was acceptable at 0.649 although, again, this result is mediocre and suggests the possibility that correlations between variables may not be explained by other variables. Bartlett's test of Sphericity was calculated and found to be significant (Approximate Chi Square (153) = 394.676; $p < 0.001$), thus increasing the likelihood that a meaningful number of factors representing relationships between sets of inter-related factors may be extracted.

Having extracted the 4-factor solution, closer examination demonstrated that the first factor accounted for 20.35% of the variance, the second factor accounted for 12.40%, the third factor contributed a further 11.05% and the

final factor contributed 10.31%. Thus, the four-factor model accounted for a total of 54.10% of the variance amongst the IPQ-R causal attribution variables. Table 5 shows the results of the principal components analysis with varimax rotation for the specified four factors.

Table 5: Results of principal components analysis with varimax rotation for IPQ-R Causal Attribution questionnaire items

Component	Eigen Value	% Variance	Cumulative %
1	3.662	20.345	20.345
2	2.231	12.395	32.740
3	1.989	11.048	43.788
4	1.856	10.312	54.100

Table 6 shows the correlations between the variables and the extracted factors above 0.40. In order to compare these results with the factor analysis conducted by Moss-Morris et al. (2002), the questionnaire items that correspond to the factor identification of the validation study are highlighted in red.

Table 6: Rotated component matrix for IPQ-R Causal Attribution questionnaire items

	Component			
	1	2	3	4
IPQ1.C1				-.501
IPQ1.C2				.555
IPQ1.C3		.588		
IPQ1.C4			.543	
IPQ1.C5			-.771	
IPQ1.C6	.736			
IPQ1.C7	.492			
IPQ1.C8			.742	
IPQ1.C9	.768			
IPQ1.C10	.725			
IPQ1.C11		.710		-.452
IPQ1.C12	.813			
IPQ1.C13				.748
IPQ1.C14		.597		
IPQ1.C15			.614	
IPQ1.C16		.514		.529
IPQ1.C17		.645		
IPQ1.C18	.798			

Extraction Method: Principal Component Analysis.
a Rotation converged in 5 iterations.

From this analysis it can be seen that only 8 out of the 18 IPQ-R causal attribution questionnaire items correspond to the factors identified in the Moss-Morris et al. (2002) study. Two (2) questionnaire items did not enter the factor structure their correlation statistics were less than 0.45 and 8 questionnaire items were loaded onto factors other than the ones demonstrated in the Moss-Morris et al. (2002) validation study.

From this exploratory analysis, it may be concluded that the results of the factor analysis of the IPQ-R causal attribution questionnaire items for this sample do not approximate those of the initial validation study as closely as

the factor analysis of the IPQ-R General variables. Therefore, the use of the IPQ-R causal attribution factors for this sample has reduced validity.

3.5. Results Part four

3.5.1. Analysis of association between illness representations and PTSD symptomatology

3.5.1.1. Exploratory data analysis for IPQ-R variables

In order to analyse the association between the 12 IPQ-R factors (General and Causal Attribution factors) and the DTS scores at T1, T2 and T3 it was necessary to assess the distribution of the scores for the IPQ-R factors at all three time points.

Through exploring the IPQ-R data at T1 and conducting One-sample Kolmogorov-Smirnov tests for each of the factors, the following factors were found to be normally distributed: identity, timeline (acute/chronic), consequences, personal control, timeline (cyclical), emotional representation, psychological attributions, and risk factor attributions. The treatment control, illness coherence, immunity attribution and chance attribution factors were not found to be normally distributed. Therefore, the latter, non-normally distributed factors were transformed into log variables in order to permit parametric Pearson's correlational analysis for the detection of linear relationships between the variables. However, despite the log transformation, the One-sample Komogorov-Smirnov test results for these variables remained

significant and, therefore, a normal distribution could not be assumed for these variables. Consequently, a non-parametric correlational analysis was undertaken for these variables (Spearmans).

Through exploring the IPQ-R data at T2 and conducting one-sample Kolmogorov-Smirnov tests for each of the factors, the following factors were found to be normally distributed: identity, timeline (acute/chronic), consequences, personal control, treatment control, timeline (cyclical), emotional representation, psychological attributions, risk factor attributions, immunity attributions and chance attributions factors. The Illness coherence factor was not found to be normally distributed. Therefore, the latter, non-normally distributed factor was transformed into log variables in order to permit parametric Pearson's correlational analysis for the detection of linear relationships between the variables. Despite the log transformation, the one-sample Komogorov-Smirnov test results for this variable remained significant and, therefore, a normal distribution could not be assumed for these variables. Consequently, a non-parametric correlational analysis was undertaken for this variable (Spearmans).

Finally, through exploring the IPQ-R data at T3 and conducting one-sample kolmogorov-smirnov tests for each of the factors, all of the IPQ-R factors were found to be normally distributed. Therefore, Pearson's correlational analysis was conducted on all the IPQ-R T3 variables in order to explore linear relationships between the variables.

As multiple correlational analyses were conducted for the IPQ-R variables at all 3 time points, this procedure carried a risk of increasing the chance of detecting false positives (Type 1 error). To reduce the possibility of Type 1 error, the statistical significance level was set at $p < 0.01$ for all correlational analyses.

3.5.1.2. Correlational analysis between IPQ-R and DTS scores at T1

Pearson's correlations were carried out between the identity, timeline (acute/chronic), consequences, personal control, timeline (cyclical), emotional representation, psychological attributions, risk factor attributions factors and DTS1. Spearman's correlations were carried out on the treatment control, illness coherence, immunity attributions, chance attributions factors and DTS1.

Significant correlations were detected between DTS1 scores and identity, timeline (acute/chronic), consequences, timeline (cyclical) and emotional representation scores at T1 ($p<0.01$) (Table 7).

Table 7: Correlations between DTS1 and IPQ-R scores at T1

		DTS1 Total
IPQ1 Identity	Pearson Correlation	.369(**)
	Sig. (2-tailed)	.002
	N	68
IPQ1 Timeline (acute/chronic)	Pearson Correlation	.362(**)
	Sig. (2-tailed)	.002
	N	68
IPQ1 Consequences	Pearson Correlation	.325(**)
	Sig. (2-tailed)	.007
	N	68
IPQ1 Personal Control	Pearson Correlation	-.013
	Sig. (2-tailed)	.917
	N	68
IPQ1 Timeline (cyclical)	Pearson Correlation	.415(**)
	Sig. (2-tailed)	.000
	N	68
IPQ1 Emotional	Pearson Correlation	.726(**)
	Sig. (2-tailed)	.000
Representation	N	68
IPQ1 Psychological	Pearson Correlation	.168
	Sig. (2-tailed)	.172
Attributions	N	68
IPQ1 Risk Factor Attributions	Pearson Correlation	.037
	Sig. (2-tailed)	.767
	N	68
IPQ1 Treatment Control	Spearman's Correlation	-.157
	Sig. (2-tailed)	.200
	N	68
IPQ1 Illness Coherence	Spearman's Correlation	-.305(*)
	Sig. (2-tailed)	.011
	N	68
IPQ1 Immunity Attributions	Spearman's Correlation	.303(*)
	Sig. (2-tailed)	.012
	N	68
IPQ1 Chance Attributions	Spearman's Correlation	.225
	Sig. (2-tailed)	.066
	N	68

** Correlation is significant at the 0.01 level (2-tailed)

* Correlation is significant at the 0.05 level (2-tailed)

3.5.1.3. Correlational analysis between IPQ-R and DTS scores at T2

Pearson's Correlations were carried out between the identity, timeline (acute/chronic), consequences, personal control, treatment control, timeline (cyclical), emotional representation, psychological attributions, risk factor attributions, immunity attributions and chance attributions factors and DTS2. Spearman's correlations were carried out on the illness coherence factor and DTS2.

Significant correlations were detected between DTS2 scores and identity, timeline (acute/chronic), consequences, emotional representation and psychological attributions scores at T2 ($p<0.01$) (Table 8).

Table 8: Correlations between DTS2 and IPQ-R scores at T2

		DTS2 Total
IPQ2 Identity	Pearson Correlation	.523(**)
	Sig. (2-tailed)	.000
	N	52
IPQ2 Timeline (acute/chronic)	Pearson Correlation	.367(**)
	Sig. (2-tailed)	.007
	N	52
IPQ2 Consequences	Pearson Correlation	.418(**)
	Sig. (2-tailed)	.002
	N	52
IPQ2 Personal Control	Pearson Correlation	.027
	Sig. (2-tailed)	.847
	N	52
IPQ2 Treatment Control	Pearson Correlation	-.007
	Sig. (2-tailed)	.960
	N	52
IPQ2 Timeline (cyclical)	Pearson Correlation	.314(*)
	Sig. (2-tailed)	.023
	N	52
IPQ2 Emotional Representation	Pearson Correlation	.687(**)
	Sig. (2-tailed)	.000
	N	52
IPQ2 Psychological	Pearson Correlation	.478(**)
	Sig. (2-tailed)	.000
Attributions	N	52
IPQ2 Risk Factor Attributions	Pearson Correlation	.086
	Sig. (2-tailed)	.546
	N	52
IPQ2 Immunity Attributions	Pearson Correlation	.227
	Sig. (2-tailed)	.106
	N	52
IPQ2 Chance Attributions	Pearson Correlation	.173
	Sig. (2-tailed)	.221
	N	52
IPQ2 Illness Coherence	Spearman's Correlation	-.073
	Sig. (2-tailed)	.608
	N	52

** Correlation is significant at the 0.01 level (2-tailed)

* Correlation is significant at the 0.05 level (2-tailed)

3.5.1.4. Correlational analysis between IPQ-R and DTS scores at T3

Pearson's Correlations were carried out on all of the IPQ-R factors at T3 and DTS3.

Significant correlations were detected between DTS3 scores and identity, timeline (acute/chronic), consequences, timeline (cyclical) and emotional representation scores at T3 ($p<0.01$) (Table 9).

Table 9: Correlations between DTS3 and IPQ-R scores at T3

		DTS3 Total Score
IPQ3 Identity	Pearson Correlation	.648(**)
	Sig. (2-tailed)	.000
	N	44
IPQ3 Timeline (acute/chronic)	Pearson Correlation	.401(**)
	Sig. (2-tailed)	.007
	N	44
IPQ3 Consequences	Pearson Correlation	.603(**)
	Sig. (2-tailed)	.000
	N	44
IPQ3 Personal Control	Pearson Correlation	-.127
	Sig. (2-tailed)	.413
	N	44
IPQ3 Treatment Control	Pearson Correlation	-.244
	Sig. (2-tailed)	.110
	N	44
IPQ3 Illness Coherence	Pearson Correlation	-.381(*)
	Sig. (2-tailed)	.011
	N	44
IPQ3 Timeline (cyclical)	Pearson Correlation	.479(**)
	Sig. (2-tailed)	.001
	N	44
IPQ3 Emotional Representation	Pearson Correlation	.734(**)
	Sig. (2-tailed)	.000
	N	44
IPQ3 Psychological Attributions	Pearson Correlation	.381(*)
	Sig. (2-tailed)	.011
	N	44
IPQ3 Risk Factor Attributions	Pearson Correlation	-.038
	Sig. (2-tailed)	.805
	N	44
IPQ3 Immunity Attributions	Pearson Correlation	.136
	Sig. (2-tailed)	.380
	N	44
IPQ3 Chance Attributions	Pearson Correlation	-.095
	Sig. (2-tailed)	.541
	N	44

** Correlation is significant at the 0.01 level (2-tailed)
* Correlation is significant at the 0.05 level (2-tailed)

3.5.2. Summary of section 3.5

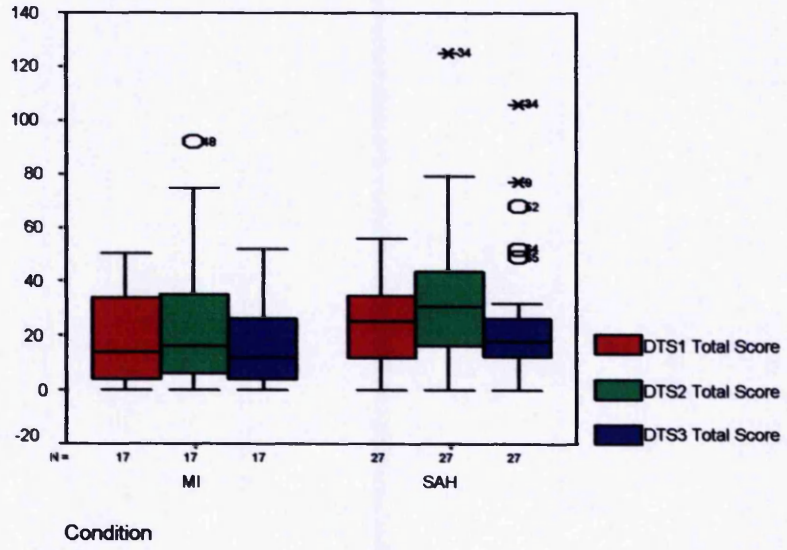
The above correlational analyses reveal a consistency in the association between the 4 IPQ-R factors of identity, timeline (acute/chronic), consequences, emotional representation with DTS scores across the 3 time points. The timeline (cyclical) factor was associated with DTS scores at T1 and T3.

3.6. Analysis of changes in PTSD symptomatology over time

3.6.1. Exploratory data analysis

Exploration of the database by boxplot graphs (Figure 10) revealed similar profiles of DTS scores across the three time points within the study (DTS1, DTS2 and DTS3 scores) for both samples. One extreme case was identified at T2 for the subarachnoid haemorrhage sample and two extreme cases were detected for this sample at T3. However, these cases were included in the analysis as there were no theoretical reasons for their exclusion. These cases were not considered unrepresentative within the sample. Examination of the boxplots' median bars suggests that the data is not skewed. Evidence for the normal distribution of the DTS1, DTS2, and DTS3 scores is gained from the respective non-significant results of One-sample Kolmogorov-Smirnov tests.

Figure 10: Boxplot graphs of DTS scores at T1, T2 and T3 by medical condition samples



3.6.2. Within-subjects ANOVA analysis of changes in PTSD symptomatology over time

Having decided to include all the cases from both groups and confirmed the validity of carrying out a parametric repeated measures analysis of DTS scores across the three time points, the analysis proceeded to examine differences in DTS scores and possible interactions between changes in scores over time and the condition (the 2 medical group samples). Thus, a two factor, mixed factorial ANOVA was utilised. The within-subjects variables were the DTS1, DTS2, and DTS3 scores and the between subjects variable was the medical condition (subarachnoid haemorrhage or myocardial infarction). The analysis was carried out on 44 subjects who had completed all three sets of questionnaires.

The descriptive statistics for the analysis are shown in Table 10.

Table 10: Descriptive statistics for DTS Scores at T1, T2, and T3

	Condition	Mean	Std. Deviation	N
DTS1 Total Score	**MI**	17.94	16.407	17
	SAH	24.89	16.258	27
	Total	22.20	16.483	44
DTS2 Total Score	**MI**	23.82	26.202	17
	SAH	34.44	28.032	27
	Total	30.34	27.531	44
DTS3 Total Score	**MI**	16.47	16.318	17
	SAH	25.41	25.071	27
	Total	21.95	22.327	44

The result of Mauchly's test of Sphericity was significant (Mauchly's W = 0.664, $p < 0.001$) indicating that there is heterogeneity of covariance within the sample. Therefore, a more conservative F test (Greenhouse-Geisser) was used for the ANOVA calculation. The results of the within subjects test are shown in Table 11.

Table 11: Test of Within-subjects effects for DTS Scores at T1, T2 and T3 by medical condition group

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
TIME	Greenhouse-Geisser	1765.992	1.497	1179.623	4.994	.017
TIME*CONDITION	Greenhouse-Geisser	70.537	1.497	47.116	.199	.755
Error(TIME)	Greenhouse-Geisser	14850.751	62.877	236.186		

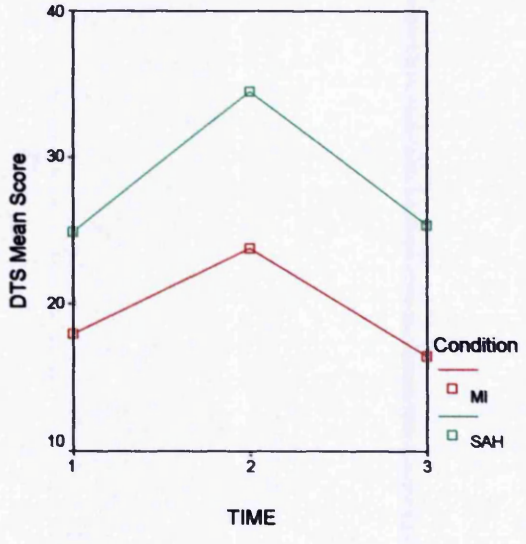
This demonstrates that the factor time is significant at the 0.05 level ($F(1.497, 62.877) = 4.994$; $p = 0.017$) and that the interaction between time and condition is not significant ($F(1.497, 62.877) = 0.199$; $p = 0.755$).

Further pairwise comparisons of the DTS1, DTS2 and DTS3 scores using Bonferroni adjustment for multiple comparisons indicates that there is a significant decrease in scores between DTS2 and DTS3 scores ($p = 0.003$). Differences between DTS1 and DTS2, and DTS1 and DTS3 were not significant.

Test of between-subjects effects show that there are no significant differences between the subarachnoid haemorrhage and the myocardial infarction groups ($F(1,42) = 2.122$; $p = 0.153$).

The differences between the two groups in DTS scores over the three time points may be demonstrated graphically as in Figure 11.

Figure 11: Line graph of DTS scores at T1, T2 and T3 by medical condition samples



3.6.3. Summary of section 3.6

These results demonstrate that there are significant differences in PTSD symptomatology over time after the first three months of recovery. They also confirm the homogeneity of change over time for both sample groups.

3.7. Comparison of profiles of illness representations for the two sample groups

3.7.1. Exploratory data analysis

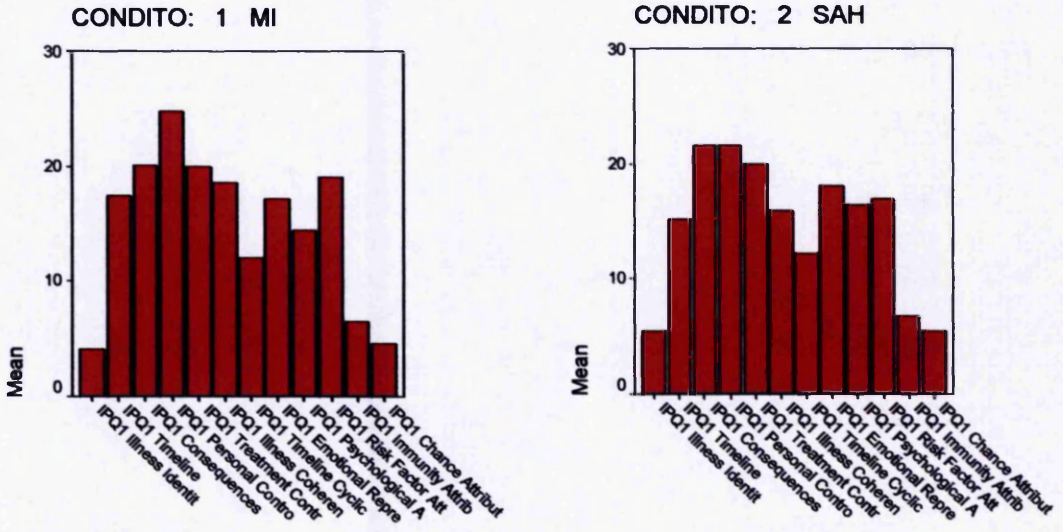
Exploratory analysis revealed that a considerable number of variables were affected by the presence of extreme cases, and several of the IPQ-R variables were non-normally distributed. Therefore, it was decided to conduct non-parametric tests (Mann-Whitney U tests) to compare the mean ranks of the two samples for the IPQ-R variables. As in other analyses, multiple testing was required to examine the full range of IPQ-R variables, and, therefore, the level of significance was set at $p < 0.01$ to reduce the possibility of type 1 errors occurring.

3.7.2. Comparison of illness representations means between the two sample groups

For IPQ-R variables at T1, three variables showed significant differences between the two sample groups; personal control ($z = 4.21$; $p = 0.000$), risk factor attributions ($z = 2.66$; $p = 0.008$) and chance attributions ($z = 2.602$; $p = 0.009$). Examination of the descriptive statistics indicates that the mean rank scores for the personal control and risk factor attributions variables were significantly higher for the myocardial infarction sample and the mean rank scores for the chance attribution variables were significantly higher for the

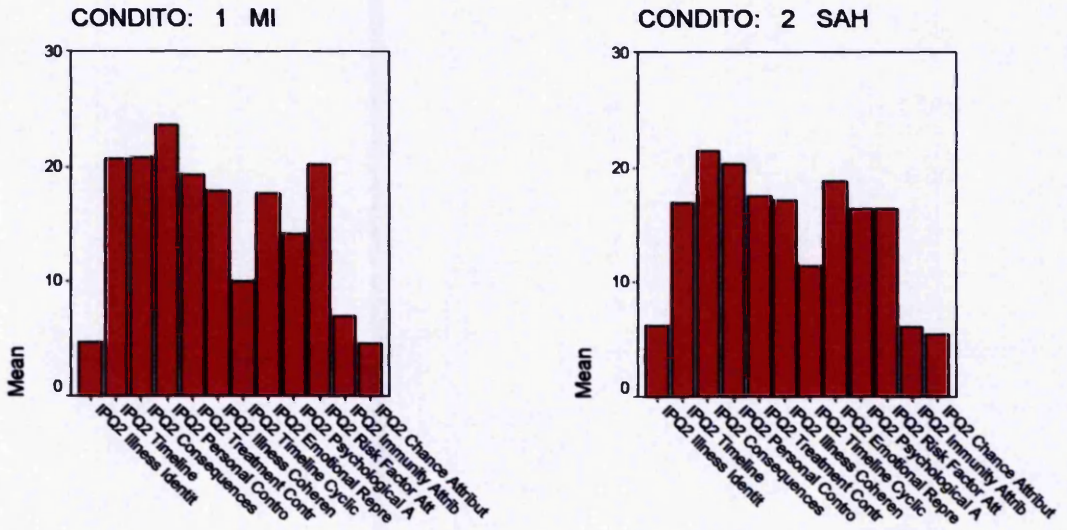
subarachnoid haemorrhage sample. The profiles of the IPQ-R Factor scores for each of the medical condition samples are shown in Figure 12.

Figure 12: Bar charts of mean IPQ-R factor scores at T1 by medical condition samples



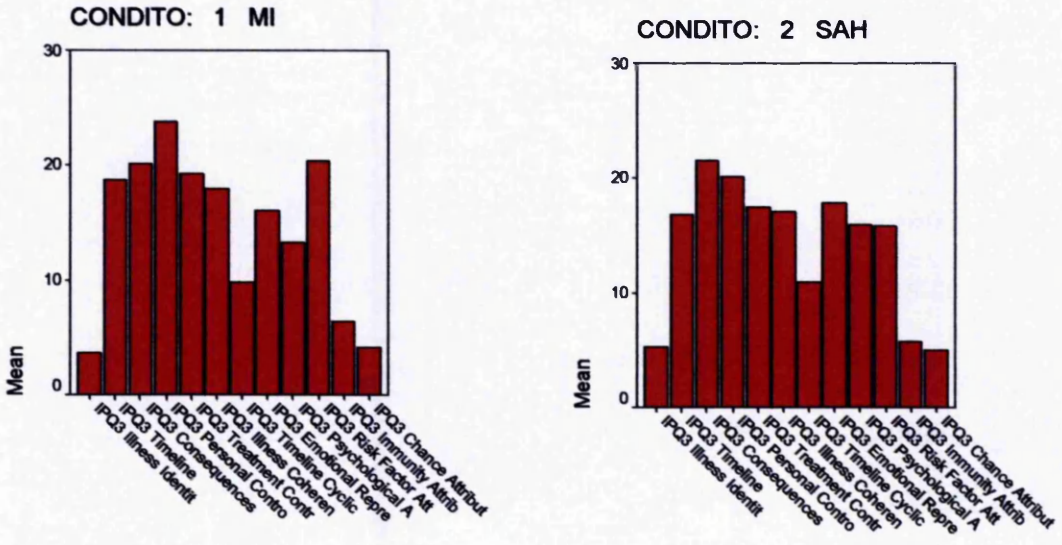
For IPQ-R variables at T2, three variables showed significant differences between the two sample groups; timeline (acute/chronic) ($z = 2.628$; $p = 0.009$), personal control ($z = 3.165$; $p = 0.002$), and risk factor attributions ($z = 3.287$; $p = 0.001$). Examination of the descriptive statistics indicates that the mean rank scores for the timeline (acute/chronic), personal control, and risk factor attributions variables were significantly higher for the myocardial infarction sample. The profiles of the IPQ-R Factor scores for each of the medical condition samples are shown in Figure 13.

Figure 13: Bar charts of mean IPQ-R factor scores at T2 by medical condition samples



For IPQ-R variables at T3, two variables showed significant differences between the two sample groups; personal control ($z = 2.80$; $p = 0.005$), and risk factor attributions ($z = 3.35$; $p = 0.001$). Examination of the descriptive statistics indicates that the mean rank scores for the personal control and risk factor attributions variables were significantly higher for the myocardial infarction sample. The profiles of the IPQ-R Factor scores for each of the medical condition samples are shown in Figure 14.

Figure 14: Bar charts of mean IPQ-R factor scores at T3 by medical condition samples



3.7.3. Summary of section 3.7

In summary, the results of the Mann-Whitney U tests indicate that there is a degree of similarity in the profiles of the illness representations for the two samples at all three time points. At T1 and T2, there were no significant differences between the mean ranks for 9 out of the 12 IPQ-R variables. At T3, there were no significant differences between the mean ranks for 10 out of the 12 IPQ-R variables. However, the myocardial infarction sample had significantly higher mean rank scores in the variables of personal control, and risk factor attributions across all three time points. The myocardial infarction sample also had a significantly higher timeline (acute/chronic) mean score at T2.

3.8. Analysis of changes in illness representations over time

3.8.1. Exploratory data analysis

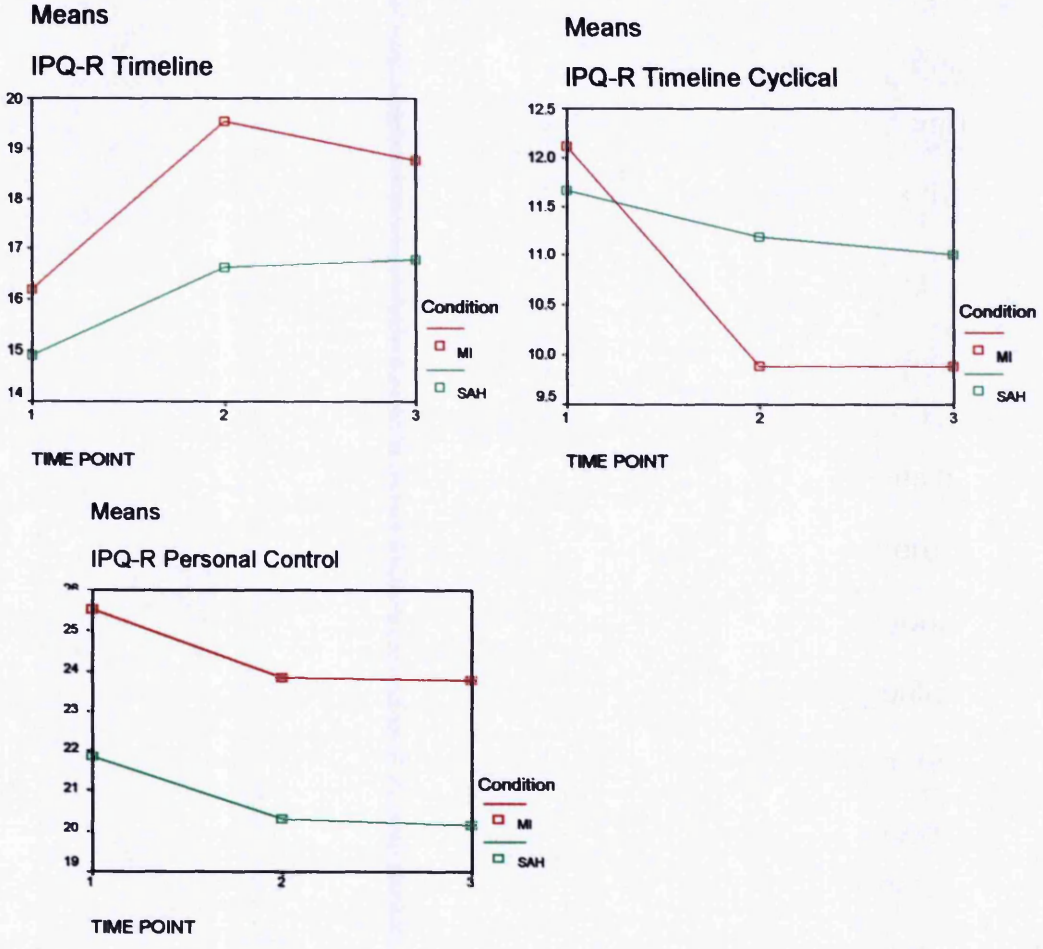
The previous analysis had demonstrated that the IPQ-R profiles for the two sample groups were similar. This finding, lends support to the validity of analysing changes in the IPQ-R variables for both groups across the three time points.

In order to examine changes in the IPQ-R over the three time points, 8 repeated-measure ANOVAs were conducted for the IPQ-R variables that had a normal distribution at all 3 time points. Four Friedman tests were carried out for those variables that had a non-normal distribution at any one of the time points (treatment control, illness coherence, immunity attributions and chance attributions). As this statistical procedure introduced an increased chance of Type 1 error (detecting false positives), the significance level for these analyses was set at $p < 0.01$. For the repeated-measure ANOVAs, the Greenhouse-Geisser analysis was also used to produce a more conservative result. A similar statistical method was utilised to the analysis of PTSD changes over time as outlined above (3.6), with time (scores at T1, T2 and T3) as the within subjects factor and condition (subarachnoid haemorrhage or myocardial infarction) as the between subjects factor.

3.8.2. Two-factor mixed factorial ANOVA analyses

Out of the 8 analyses, significant within-subject differences were detected for the following three IPQ-R variables; timeline (acute/chronic) ($F(1.874, 78.718) = 7.748$; $p = 0.001$), personal control ($F(1.625, 68.239) = 6.726$; $p = 0.004$), and timeline (cyclical) ($F(1.810, 76.008) = 4.996$; $p = 0.011$). There were no significant interactions between the within-subjects factor (time) and the between-subjects factor (medical condition). These results are demonstrated graphically in Figure 15.

Figure 15: Line graphs of IPQ-R factor scores at T1, T2, and T3 by medical condition samples for significant ANOVA analyses

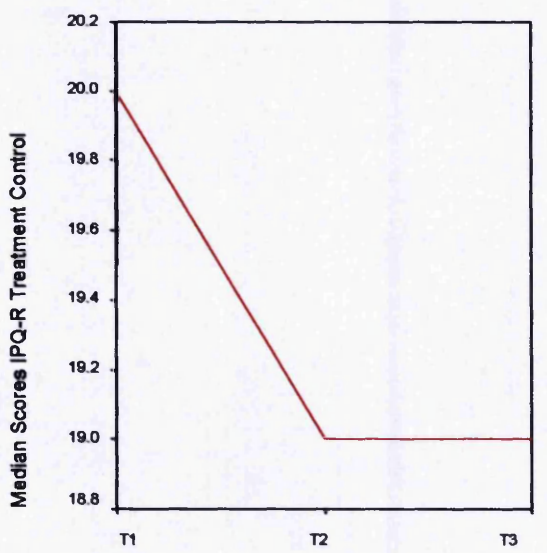


Significant between-subjects differences were found for the following three variables; personal control ($F(1,42) = 16.683$; $p < 0.001$), risk factor attributions ($F(1,42) = 12.077$; $p = 0.001$) and chance attributions $F(1,42) = 6.614$; $p = 0.005$).

3.8.3. Friedman test analyses

Out of the 4 analyses, one significant result was found for the treatment control variable (Chi-Square = 24.352, df = 2; $p < 0.001$). This analysis did not permit any examination of between-subject differences. A plot of the median values of the IPQ-R treatment control scores at T1, T2 and T3 is shown in Figure 16.

Figure 16: Line graph of median IPQ-R treatment control factor scores at T1, T2, and T3 by medical condition samples



3.8.4. Summary of Section 3.8

The above analyses have demonstrated significant changes over time for 4 of the IPQ-R variables. The timeline (acute/chronic) variable is shown to increase over time and the timeline (cyclical), personal control and treatment control variables are shown to decrease over time.

3.9. Analysis of predictor variables for PTSD

The following multiple regression analyses were carried out to examine predictor variables for levels of PTSD symptomatology at T3 from the groups of demographic, social support, thought control and illness representation variables at T2. The regression analyses were conducted upon the 44 cases who completed the questionnaires at all three time points.

3.9.1. Analysis of Social Support Inventory (SSI) predictor variables at T2 for PTSD at T3

3.9.1.1. Multiple regression analysis

This analysis included all 5 variables from the SSI (perceived criticism from significant other, criticism of significant other, satisfaction with emotional support, satisfaction with practical support and expectation of support). The demographic variables of age and gender were also included in this analysis. The results of this regression analysis found that none of the social support

and demographic variables were significantly associated with the outcome variable of DTS score at T3.

3.9.2. Analysis of Thought Control Questionnaire (TCQ) predictor variables at T2 for PTSD at T3

3.9.2.1. Multiple regression analysis

Predictive multiple regressions were conducted to examine the impact of the five Thought Control Questionnaire variables (distraction, reappraisal, worry, punishment and social control) from the TCQ at T2 upon DTS scores at T3. The demographic variables of age and gender were also included in this analysis.

The results of the multiple regression analysis for Thought Control Questionnaire variables and demographic variables at T2 against DTS scores at T3 demonstrated that the variables of worry ($B = 3.883$; $p < 0.001$), Age ($B = -0.52$; $p = 0.022$) and distraction ($B = -1.335$; $p = 0.048$) at T2 are significant predictors of PTSD at T3 ($F = 8.861$; $p < 0.001$). The adjusted R^2 value for the model including the above three variables at T2 is 0.354 indicating that approximately 35% of the variance in DTS3 scores may be accounted for by this model.

3.9.3. Analysis of IPQ-R predictor variables for PTSD

3.9.3.1. Multiple regression analyses

Predictive multiple regressions were conducted to examine the impact of Illness Perception scores at T2 upon DTS scores at T3. The demographic variables of age and gender were not included in these analyses as they had been examined previously in the multiple regression analyses. Age had already been found to have a predictive association with DTS scores when analysed together with the Thought Control Questionnaire variables. The exclusion of these variables also increased the ratio of subjects to predictor variables within the equation, thus increasing the power of the regression.

The results of the multiple regression analysis for IPQ-R General variables at T2 against DTS scores at T3 demonstrated that the variables of emotional representation (B = 2.228; p <0.001), illness coherence (B = -1.538; p = 0.015), consequences (B = 1.978; p = 0.003) and treatment control (B = -1.902; p = 0.038) at T2 are significant predictors of PTSD at T3 (F = 18.434; p = 0.000). The adjusted R^2 value for the model including the above 4 variables at T2 is 0.619 indicating that approximately 62% of the variance in DTS3 scores may be accounted for by this model.

The results of the multiple regression analysis for IPQ-R Causal Attribution variables at T2 against DTS scores at T3 demonstrated that the variable of psychological attributions (B = 1.997; p = 0.006) at T2 is a significant predictor of DTS scores at T3 (F = 8.479; p = 0.006). The adjusted R^2 value

for the model including the above variable at T2 is 0.148 indicating that approximately 15% of the variance in DTS3 scores may be accounted for by this model.

3.9.4. Final regression analysis of significant predictor variables for PTSD

Having identified significant predictor variables from a range of separate preliminary multiple regression analyses, a final analysis was carried out in which all of the variables found to be significant in the previous multiple regression analyses were entered into the final multiple regression equation. Once again, extreme cases were included in order to achieve the maximum number of cases within the analysis. Thus, the regression included the following variables from T2; worry, distraction, emotional representation, illness coherence, consequences, treatment control and psychological attributions and age.

The results of the final multiple regression analysis for the individual predictor variables from T2 deemed to be significant from previous analyses against DTS scores at T3 demonstrated the following. The variables of emotional representation ($B = 1.541$; $p = 0.008$), illness coherence ($B = -1.216$; $p = 0.047$), consequences ($B = 2.264$; $p = 0.001$), treatment control ($B = -2.392$; $p = 0.009$) and worry ($B = 1.769$; $p = 0.029$), at T2 are significant predictors of PTSD at T3 ($F = 17.364$; $p < 0.001$). The adjusted R^2 value for the model including the above five variables at T2 is 0.656 indicating that approximately

66% of the variance in DTS3 scores may be accounted for by this model. The model summary and ANOVA statistics for the final regression are shown below in Tables 12 and 13.

Table 12: Model summary for final regression analysis

Model Summary(f)									
	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
Model					R Square Change	F Change	df1	df2	Sig. F Change
	.834(e)	.696	.656	13.105	.041	5.180	1	38	.029

Predictors: (Constant), IPQ2 Emotional Representation, IPQ2 Illness Coherence, IPQ2 Consequences, IPQ2 Treatment Control, TCQ2 Worry Score

Dependent Variable: DTS3 Total Score

Table 13: ANOVA statistics for final regression analysis

ANOVA(f)					
Model	Sum of Squares	df	Mean Square	F	Sig.
Regression	14909.953	5	2981.991	17.364	.000(e)
Residual	6525.956	38	171.736		
Total	21435.909	43			

Predictors: (Constant), IPQ2 Emotional Representation, IPQ2 Illness Coherence, IPQ2 Consequences, IPQ2 Treatment Control, TCQ2 Worry Score

Dependent Variable: DTS3 Total Score

3.10. Descriptive statistics for psychological morbidity rates at T1, T2 and T3

3.10.1. PTSD caseness descriptive statistics

Levels of PTSD symptomatology were measured at all three time points using the DTS. The changes in symptomatology have been analysed in section 3.5.2. The DTS manual (Davidson, 1996) suggests a cut-off score of 40 to achieve maximum sensitivity and specificity for this scale.

Using this cut-off criterion for PTSD caseness, 11 (16%) of the total acute medical trauma sample may be considered to have clinical levels of PTSD at T1 (4 (13%) myocardial infarction participants and 7 (18%) subarachnoid haemorrhage participants reach caseness for PTSD at T1). Table 14 demonstrates the frequency and percentage of caseness for PTSD at T1 by medical condition sample.

Table 14: Descriptive Statistics for participants reaching PTSD caseness at T1 by medical condition sample.

		PTSD Caseness at T1	
Condition		**Frequency**	**%**
MI	**Non-Case**	26	86.7
	Case	4	13.3
SAH	**Non-Case**	31	81.6
	Case	7	18.4

At T2, 16 (31%) of the total acute medical trauma sample may be considered to have clinical levels of PTSD (5 (24%) myocardial infarction participants

and 11 (36%) subarachnoid haemorrhage participants reach caseness for PTSD). Table 15 demonstrates the frequency and percentage of caseness for PTSD at T2 by medical condition sample.

Table 15: Descriptive statistics for participants reaching PTSD caseness at T2 by medical condition sample

		PTSD Caseness at T2	
Condition		Frequency	%
MI	Non-Case	16	76.2
	Case	5	23.8
SAH	Non-Case	20	64.5
	Case	11	35.5

Finally, at T3, 7 (16%) of the total acute medical trauma sample may be considered to have clinical levels of PTSD (2 (12%) myocardial infarction participants and 5 (19%) subarachnoid haemorrhage participants reach caseness for PTSD). Table 16 demonstrates the frequency and percentage of caseness for PTSD at T3 by medical condition sample.

Table 16: Descriptive statistics for participants reaching PTSD caseness at T3 by medical condition sample

		PTSD caseness at T3	
Condition		Frequency	%
MI	Non-Case	15	88.2
	Case	2	11.8
SAH	Non-Case	22	81.5
	Case	5	18.5

Of the participants who completed questionnaires at both T2 and T3, 6 of the 11 reaching caseness for PTSD at T2 reached caseness for PTSD at T3.

This suggests that approximately 55% of those reaching caseness for PTSD at T2 go on to develop PTSD at T3.

3.10.2. Anxiety and depression caseness descriptive statistics

In their review of normative data for the HADS, Crawford et al. (2001) suggest using a cut-off of 10/11 for caseness for both anxiety and depression. In determining the proportions of participants' cases reaching caseness within this study, the higher cut-off value of 11 has been employed in order to maximise the specificity of the HADS scales.

A total of 14 (27%) participants reached caseness criteria for anxiety at T2. 4 (19%) of the myocardial infarction sample and 10 (32%) of the subarachnoid haemorrhage sample reached caseness. The descriptive statistics for participants reaching anxiety caseness at T2 are given in Table 17.

Table 17: Descriptive statistics for participants reaching anxiety caseness at T2 by medical condition sample

		Anxiety Caseness at T2	
Condition		**Frequency**	**%**
MI	**Non-Case**	17	81.0
	Case	4	19.0
SAH	**Non-Case**	21	67.7
	Case	10	32.3

A total of 11 (21%) participants reached caseness criteria for depression at T2. 4 (19%) of the myocardial infarction sample and 7 (23%)of the

subarachnoid haemorrhage sample reached caseness. The descriptive statistics for participants reaching anxiety caseness at T2 are given in Table 18.

Table 18: Descriptive statistics for participants reaching depression caseness at T2 by medical condition sample

		Depression caseness at T2	
Condition		**Frequency**	**%**
MI	**Non-Case**	17	81.0
	Case	4	19.0
SAH	**Non-Case**	24	77.4
	Case	7	22.6

A total of 10 (23%) participants reached caseness criteria for anxiety at T3. 3 (18%) of the myocardial infarction sample and 7 (26%) of the subarachnoid haemorrhage sample reached caseness. The descriptive statistics for participants reaching anxiety caseness at T3 are given in Table 19.

Table 19: Descriptive statistics for participants reaching anxiety caseness at T3 by medical condition sample

		Anxiety caseness at T3	
Condition		**Frequency**	**%**
MI	**Non-Case**	14	82.4
	Case	3	17.6
SAH	**Non-Case**	20	74.1
	Case	7	25.9

A total of 6 (14%) participants reached caseness criteria for depression at T3. 2 (12%) of the myocardial infarction sample and 4 (15%)of the subarachnoid haemorrhage sample reached caseness. The descriptive

statistics for participants reaching depression caseness at T3 are given in Table 20.

Table 20: Descriptive statistics for participants reaching depression caseness at T3 by medical condition sample

		Depression caseness at T3	
Condition		**Frequency**	**Valid %**
MI	**Non-Case**	15	88.2
	Case	2	11.8
SAH	**Non-Case**	23	85.2
	Case	4	14.8

3.10.3. Summary of section 3.10

The above analyses have demonstrated that the subarachnoid haemorrhage and myocardial infarction samples have clinically important levels of PTSD, anxiety and depression across all three time points. The percentage of participants reaching caseness for PTSD increased between T1 and T2 and then decreased again at T3. The percentage of participants reaching caseness for anxiety and depression decreased between T2 and T3.

CHAPTER 4: DISCUSSION

4. Discussion

4.1. Overview of Chapter Four

This chapter is divided into five parts. Initially the results of the study will be described with reference to the aims and research hypotheses as specified in Chapter 1 (see 1.7.2 and 1.8). In part two, these findings will be discussed in the context of the wider research literature. Methodological issues are discussed in the third part and, the implications for clinical practice and future research will be outlined in part four.

4.2. Research findings

The present study specified several aims. It aimed to investigate the relationship between illness representations and PTSD symptomatology, and changes in illness representations and PTSD over time. It also sought to explore the predictive capability of illness representations, social support and thought control variables for PTSD, and explore levels of PTSD anxiety and depression in an acute medical trauma population.

The above aims have been achieved. However, reflection on the research process has highlighted limitations within the research and further ways in which the variables could be explored. These will be discussed in sections 4.4 and 4.5.

The results of the study will now be examined with regard to each research hypothesis.

4.2.1. Primary hypothesis

There will be an association between PTSD symptomatology and illness representations for both myocardial infarction and subarachnoid haemorrhage samples.

This hypothesis was tested by conducting correlational analyses between the 12 illness representation variables as measured by the IPQ-R and PTSD scores as measured by the DTS at all three time points, T1 (within two weeks of admission), T2 (5 to 7 weeks after admission) and T3 (11 to 14 weeks after admission).

Only partial support for the hypothesis was provided by the result that 4 of the 12 IPQ-R factors (identity, timeline (acute/chronic), consequences and emotional representation) were strongly correlated with DTS scores at all three time points. One further IPQ-R factor (timeline (cyclical)) was significantly correlated at time points T1 and T3, but not at T2.

The finding that some illness representations are associated with PTSD was expected based on the findings from research literature asserting that cognitive schema representations are mediating factors within the

experience of PTSD. However, as only 4 out of 12 IPQ-R factors were significantly associated it must be acknowledged that the association is not strong.

4.2.2. Subsidiary hypothesis one (1.8.2.1)

The level of PTSD symptomatology will change over time for both myocardial infarction and subarachnoid haemorrhage samples.

This hypothesis was tested by conducting a repeated measures ANOVA on the DTS scores at T1, T2 and T3.

The hypothesis was supported by the significant result of the repeat measures ANOVA. As the interaction between time and medical condition was not found to be significant the results are valid for the combined acute medical trauma sample. Further pairwise Bonferroni adjustments identify the significant difference to lie between T2 and T3.

This result was expected based on research demonstrating delayed onset for PTSD and the non-linear time course of psychological morbidity following traumatic events.

4.2.3. Subsidiary hypothesis two (1.8.2.2)

Illness representations will change over time for the myocardial infarction and subarachnoid haemorrhage samples.

This hypothesis was tested by conducting repeat measures ANOVAs and Friedman tests on the IPQ-R scores at T1, T2, and T3.

Of the 12 analyses, 4 significant results were found for the variables of personal control, treatment control, timeline (acute/chronic), and timeline (cyclical). These results provide partial support for the above hypothesis. These results were expected based upon literature asserting the dynamic nature of illness representations. Once again, however, as only 4 out of 12 IPQ-R factors were found to change significantly over time it must be acknowledged that the support for the hypothesis is not robust.

4.2.4. Subsidiary hypothesis three (1.8.2.3)

There will be a similar profile of illness representations for the myocardial infarction and subarachnoid haemorrhage samples.

The examination of the differences in mean IPQ-R scores between the two medical condition groups at all three time points using Mann-Whitney U tests tested this hypothesis.

Partial support for the hypothesis was gained from the results demonstrating that there were no significant differences between mean ranks for 9 out of the 12 IPQ-R variables at T1 and T2 and no significant differences between mean ranks for 10 out of the 12 IPQ-R variables at T3. However, support for the hypothesis may be compromised by the finding that the myocardial infarction sample had significantly higher mean rank scores for the personal control and risk factor attributions scores at all 3 time points.

These results correspond to the consideration that both medical conditions are experientially comparable. However, the two conditions may be seen as significantly different according to the illness representations of personal control and risk factor attributions.

4.2.5. Subsidiary hypothesis four (1.8.2.4)

Illness representations will be a predictor of PTSD.

This hypothesis was tested by conducting a multiple regression of IPQ-R variables at T2 to DTS scores at T3.

The results of the initial multiple regression for IPQ-R variables indicate that the IPQ-R variables of emotional representation, illness coherence, consequences, treatment control and psychological attributions are significant predictors of PTSD. The final regression analysis in which all

significant predictor variables identified in preliminary regressions were considered (see section 3.9.4) retained the IPQ-R variables of emotional representation, illness coherence, consequences, and treatment control as significant and independent predictors of PTSD.

Although there is no research literature examining specific illness representation variables as predictors of PTSD, these results do fit in with research suggesting that schema representation factors are mediating factors within PTSD. In particular, the results correspond with the finding that illness representations of consequences may lead to the fear of recurrence.

4.2.6. Subsidiary hypothesis five (1.8.2.5)

Perceived levels of social support will be a predictor of PTSD.

The multiple regression of demographic and Social Support Inventory factors at T2 to DTS scores at T3 failed to identify any significant predictor variables. Thus, the above hypothesis was not supported.

This finding was unexpected in view of the research highlighting the importance of social support variables in the maintenance of PTSD. Possible reasons for the finding of this study are discussed in 4.3.6 and 4.4.3.3.

4.2.7. Subsidiary hypothesis six (1.8.2.6)

Worry and punishment thought control strategies will be predictors of PTSD.

The preliminary multiple regression of demographic and Thought Control Questionnaire factors at T2 to DTS scores at T3 identified worry, age and distraction as significant predictor variables. The final regression analysis in which all significant predictor variables identified in preliminary regressions were considered (3.5.5.4) retained the TCQ variable of worry as a significant and independent predictor of PTSD.

Thus, the above hypothesis was partially supported. The variable of punishment was not supported as a predictor variable for PTSD.

The research literature reflects the finding for worry as a predictor variable, but does not reflect the finding of the current study that punishment is not a significant predictor variable.

4.2.8. Subsidiary hypothesis seven (1.8.2.7)

Levels of distress generally and PTSD specifically will be found in myocardial infarction and subarachnoid haemorrhage samples.

The descriptive statistics for the two medical condition groups support this hypothesis. Detected levels of PTSD for the separate medical condition samples, indicate that 19% of subarachnoid haemorrhage participants and 12 % of myocardial infarction participants reach PTSD caseness at T3. Similarly, the detected levels of anxiety and depression for the separate medical condition samples indicate levels of distress within the samples (26% of subarachnoid haemorrhage and 18 % of myocardial infarction participants reach caseness for anxiety at T3 and 15% of subarachnoid haemorrhage and 12 % of myocardial infarction participants reach caseness for depression at T3).

4.3. Significance of results to wider knowledge base

4.3.1. The association between illness representations and PTSD

Through addressing the relationship between illness representations and PTSD, this study has demonstrated some consistencies in associations between IPQ-R variables and PTSD symptomatology. The results suggest that the IPQ-R variables of identity, timeline (acute/chronic), consequences and emotional representation, may be of particular interest in understanding patients' psychological responses to the medical conditions of myocardial infarction and subarachnoid haemorrhage. This finding contributes further to the work of Prohaska et al. (1987) by refining the concept that illness representation can account for variations in emotional reactions to symptoms

of physical illness and applying this concept to an acute medical trauma sample.

4.3.2. The time course of PTSD

As the study has demonstrated a significant change in PTSD over the three time points, this finding has corresponded with previous studies. The increase in DTS scores between T1 and T2 and subsequent decrease in DTS scores between T2 and T3 correspond with the research findings suggesting a non-linear time course for emotional disturbance following trauma (McFarlane, 1988). The increase in DTS scores from T1 to T2 may correspond with the delayed onset in PTSD reported by Tarrier (1995). The finding also corresponds with subjective clinician reports of survival curves with respect to PTSD. This phenomenon has been observed in patients who, on experiencing a life-threatening trauma, express relief to have 'come through' the trauma to the relative safety of a hospital. However, on discharge, their emotional condition may deteriorate as the initial euphoric reaction regarding survival dissipates and they are faced with the demands of their previous lives with added medical uncertainty.

4.3.3. The experience of acute medical trauma

Leventhal (1997) suggests that the nature of illness representations will differ according to personal variables, their context, and the illness about which

they are constructed. Thus, different diseases may have different attributional models. Previous studies had already compared the two groups of myocardial infarction and subarachnoid haemorrhage patients (McKenna et al., 1989; McKenna and Neil-Dwyer, 1993) on the basis of their shared experience of a sudden onset, life-threatening medical event. However, Leventhal's proposition regarding distinctions in the two groups' illness representations remained. This study sought to confirm hypothesised similarities in illness representations between the two samples and found that there were many similarities. With regard to this, it is suggested that the experience of the acute medical trauma, as a life threatening and fear inducing experience, may be a shared factor for the two samples and this may contribute to the similarity in their cognitive models of their medical conditions. There may be differences in awareness and recognition of the condition at the time of its occurrence between the two groups as the risk factors for myocardial infarction are more widely known.

4.3.4 Changes in illness representations over time

Leventhal (1997) has also attested to the dynamic nature of illness representations. As the recovery of the patient usually entails many changes in physical ability, affect, social environment, and comprehension of the disease process, it is hypothesised that illness representations may change accordingly. The finding that 4 of the 12 IPQ-R variables had significant results for change over time provides partial support for this hypothesis.

It is of clinical interest to note that participants' beliefs in treatment control and personal control regarding their medical condition appeared to decrease

over time. This finding indicates an increasing uncertainty regarding active control of physical wellbeing and it may be that this uncertainty contributes to the subsequent emotional distress. Similarly, the finding that beliefs in the chronicity of the conditions increased over time may also contribute to changes in emotional distress.

4.3.5. The predictive role of illness representations in PTSD

The results of the Weinman et al. (2000) study indicate the importance of causal attributions as predictor variables for health behaviour change following a myocardial infarction. Their study suggested that patients who attributed their myocardial infarction to poor health habits rather than hereditary factors are more likely to change their health behaviours. Whilst the current study has not addressed health behaviour change, the finding that illness representations may impact upon the emotional consequences of acute medical trauma is broadly in keeping with the results of the Weinman study in attesting to the predictive role of illness representations in future adaptation to acute medical trauma. The finding from the final regression model indicates that the IPQ-R variables of emotional representation, illness coherence, consequences, and treatment control are predictive of PTSD. This suggests that the subsequent experience of PTSD may be mediated by patients' emotional responses to the acute medical trauma, their confidence in their understanding of the illness, their perception of its impact upon their lives and their confidence in subsequent medical treatment.

Of particular relevance is the predictive role of the emotional representation variable for PTSD as this was the most significant predictor of all the IPQ-R variables, accounting for approximately 47% of the variance in DTS scores. In their study of predictors of PTSD for myocardial infarction patients Bennett and Brooke (1999) found that the degree of fear experienced during the event was highly predictive of PTSD 3 months later. As the current study has confirmed the predictive role of the emotional representation of the illness, this result may correspond to Bennett and Brooke's finding by suggesting that the fear representation resulting from the traumatic event may, in part, be measured by the IPQ-R variable of emotional representation.

The finding that no causal attribution variables were predictive of PTSD according to the final regression model requires some consideration. The search for causal attributions following trauma has been noted in previous studies (Turnquist, Harvey and Anderson, 1988) (de-Silva and Marks, 1999) although the work of Joseph (1999) has highlighted the need to consider the process of making causal attributions as a dynamic process. The search for causal explanations is made within a temporal context in which the individual may simultaneously seek to maintain a sense of self-esteem and personal control. Thus, the search for causal attributions may impinge upon either of these processes at different times in the recovery period following trauma. The complexity of this process may, in part, account for the absence of any significant results for this part of the current study. The factor analysis of the IPQ-R causal attribution questionnaire items did not correspond accurately to the findings of the IPQ-R validation study (Moss-Morris et al., 2002). This

may reflect the fact that the data was collected at T1, i.e. within 2 weeks of the acute medical trauma event when the patients' search for causal attributions may not have developed to achieve a degree of consistency.

4.3.6. The predictive role of social support for PTSD

The absence of significant results regarding social support predictor variables for PTSD does not correspond with previous research and this finding must be accounted for. Bennett and Brooke (1999) found that 10% of variance in PTSD scores 6 – 12 months after a myocardial infarction was accounted for by social support variables as measured by the Duke UNC Social Support Questionnaire (Broadhead, Gehlbach, DeGruy and Kaplan, 1988).

The use of the Social Support Inventory in the current study may have affected the results obtained as the researcher noted a response bias within the returned questionnaires. This observation caused the researcher to question the validity and reliability of some of the SSI responses. This problem is considered significant given the small sample size for the regression analysis. Methodological issues concerning the use of the SSI will be discussed in section 4.4.3.3.

4.3.7. The predictive role of thought control strategies for PTSD

Reynolds and Wells (1999) found that individuals recovering from PTSD and/or depression were less likely to use worry and punishment as thought control strategies and the current study provides partial support for this finding. The thought control strategy of worry appeared as a significant predictor variable for PTSD in the preliminary and final multiple regression analyses in the current study.

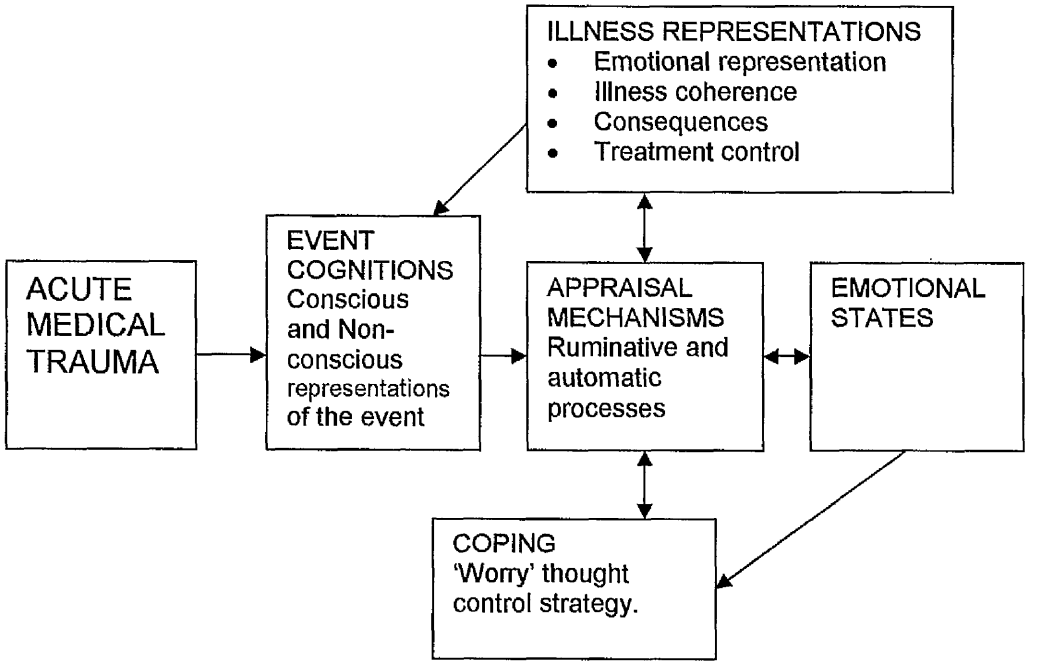
Thus, the use of worry about other issues as an active cognitive distraction technique is likely to increase PTSD symptoms. This finding also corresponds with previous research findings confirming the role of avoidant coping in maintaining PTSD (Bryant and Harvey, 1995; Lemon, 2001). The current research lends support to the theory that cognitive avoidance strategies such as worry may maintain PTSD by preventing the activation and modification of the trauma related schema held by the individual (Litz and Keane, 1989).

The absence of support for the predictive capability of the thought control strategy of punishment may be due to differences between the sample used in this study and the sample used in the Reynolds and Wells (1999) study or to the small sample size within this study.

4.3.8. Psychosocial models of PTSD and acute medical trauma

The current study has confirmed the role of schema representations (illness representations) and coping/thought control factors in the development and maintenance of PTSD. The role of social support variables has not been supported in the current study, (although reasons for this finding are discussed). Thus, the psychosocial model of PTSD of Williams and Joseph (1999) has received partial support from the findings of this study. The correspondence between the findings of the current study and the psychosocial model of PTSD may be represented diagrammatically as in Figure 17.

Figure 17: Correspondence between current research findings and the psychosocial model of PTSD



The role of appraisal mechanisms, event cognitions, and behavioural avoidant coping strategies have not been explored directly. Of particular interest is the concept of appraisal mechanisms; this element of the model may require further refining and utilisation of concepts drawn from the S-REF model (Wells and Matthews, 1994) and hopelessness theory (Abramson et al., 1989). Particular attention may be paid to metacognitive beliefs regarding choice of cognitive coping strategy as this may inform the concept of appraisal mechanisms within the psychosocial model (Wells, 2000).

4.4. Methodological considerations

During the current study, several methodological issues became apparent. Their relevance for the generation of the results obtained and the interpretation of the results will now be considered.

4.4.1. Sampling issues

The first methodological consideration concerns the small sample size recruited to the study. Examination of the attrition rates indicates that, from an initial sample size of 68 participants, the final number of participants at T3 was 44. A basic power calculation for the correlational analyses had indicated that a sample size of approximately 60 participants would be required. However, a comprehensive power calculation was not considered possible due to the absence of any estimations of effect size for the acute

medical trauma sample. Therefore, the study sought to recruit as many participants as possible within the time frame. Without a specific power calculation for this sample, it is not possible to state how significant the influence of unknown variance is.

The small sample size must be acknowledged when interpreting the results of all the statistical analyses. However, it is of particular importance when considering the results of the Factor Analysis of the IPQ-R, and the multiple regressions. The factor analysis in the current study may only be considered as confirmatory of previous, more robust, analyses. The multiple regressions required that identified extreme cases be included in order to maximise the number of participants within the analysis. Even with the extreme cases included, the majority of the regressions were conducted with very small predictor variable to participant ratios (ratios ranged from 1:11 for the IPQ-R causal attribution regression to 2:11 for the General IPQ-R regression). The sample size limitations indicate that all the research findings must be interpreted with caution.

A further sampling issue relates to the inclusion and exclusion criteria used within the study. As these criteria excluded people with significant cognitive problems at T1, it must be acknowledged that a there is a bias towards the inclusion of less severely affected participants within the sample.

4.4.2. Non-responders at T2 and T3

Having analysed the responders and non-responders at T2 and T3, the study detected that there are no demographic or psychological morbidity differences between the two groups at T2. However, the two groups differ at T3 in terms of the level of PTSD as recorded at T2; non-responders at T3 having significantly higher DTS2 scores than responders. This raises the issue of bias entering the study as those participants who experience the most psychological distress at T2 are demonstrated to be more likely to drop out of the study. This may result in the less psychologically distressed participants being included in the regression analyses and, therefore, the results will be biased towards the experience of this reduced sample. The bias introduced through the drop out rate at T2 has not affected the results of the analysis of changes in PTSD and illness representations over time as these analyses were conducted only upon the 44 participants who completed questionnaires at all 3 time points.

A further issue is the limited number of demographic and physical illness variables considered in this analysis. In order to avoid burdening participants excessively, the study did not explore variables such as marital status, occupational status, level of physical disability, severity of illness, and length of stay in hospital. Therefore, differences between responders and non-responders in these variables are not explored.

There is a distinct possibility that many participants chose not to return questionnaires at T2 and T3 a they perceived the task of completing a large number of questionnaires to be arduous. This possibility could not be investigated as feedback on this issue was not requested.

4.4.3. Measures

There are several issues relating specifically to the measures used within the study that require consideration. Each of the measures used will be considered in turn.

4.4.3.1. Davidson Trauma Scale (DTS)

As discussed in 1.6.2.1 and 2.6.2.2, a diagnosis of PTSD may only be made at least one month after the trauma. Up to one month after the trauma, a diagnosis of Acute Stress Disorder (ASD) may be considered. As the symptoms associated with a diagnosis of ASD (e.g. dissociation symptoms) differ slightly from those of PTSD, the use of a PTSD diagnostic measure within one month of the PTSD requires justification. As this study was concerned with measuring PTSD symptomatology consistently over time, the use of the DTS was considered appropriate. Analysis of caseness for PTSD has been limited to T2 and T3 in order to acknowledge this diagnostic issue. However, the design of the study required analysis of PTSD symptomatology levels at all 3 time points.

4.4.3.2. Illness Perception Questionnaire – Revised (IPQ-R)

The IPQ-R has been tested on a wide range of patient samples, including myocardial infarction patients. Through conducting a factor analysis, the current study found that the general IPQ-R General factor structure was appropriate and valid for use with the total acute medical trauma population but that the IPQ-R causal attribution factor structure was not supported by results from the current study. This raises the question of the validity of this measure for the acute medical trauma sample. The uncertainty regarding this measure was also demonstrated by comments made by some of the subarachnoid haemorrhage participants who struggled to complete the questionnaire. Their comments alluded to the fact that they did not see their condition as an illness as such, and believed it to be more of a single event. Such comments lend support to the conceptualisation of subarachnoid haemorrhage as a traumatic event and, thus, vulnerable to symptoms of PTSD. The authors of the IPQ-R acknowledge the difference in perspective between patient groups and state that the measure may be amended for particular illnesses. A further problem with the IPQ-R was the fact that there were two versions in circulation at the time of designing the study. One version included 50 questionnaire items in the general IPQ-R section and the other included only 38. During the course of the study consensus was reached on the validity of the 38 item form. As the study had initially used the 50 item version, this practice was continued (with the 12 redundant items being excluded from the scoring) to ensure continuity and conformity. The use of the 50 item version may have deterred some participants from

completing the questionnaires as it may have appeared too lengthy. Finally, use of the Emotional Representations factor scores within the regression equations may be challenged as, tautologically, it may appear to predict emotional responses to the acute medical trauma from a measure of emotional responses.

4.4.3.3. Social Support Inventory (SSI)

Difficulties with this measure have already been mentioned in 4.3.6. An overview of the responses to the SSI revealed that the scores of several participants indicated that they perceived high levels of criticism from significant others whilst affirming a high level of perceived emotional support. These results appear ambiguous and contradictory. As this pattern of results was obtained on 8 occasions at T2 and 4 occasions at T3, this suggests that the questionnaire may have been completed with a misunderstanding of the direction of effect of some of the questionnaire items. Clearly, this phenomenon may have affected the results through introducing a response bias and may account for the lack of any significant association between social support variables and PTSD. Such a bias would have been compounded by the small sample size within this study.

The use of single question items in the SSI as predictor variables in a regression may also have compromised the validity of this measure. Analysis of the SSI variables on the sample excluding those participants with clearly ambiguous responses was not considered possible, as this would have led to a sample size of 36. This reduced sample size would have reduced the validity of the final regression analysis further.

4.4.3.4. Thought Control Questionnaire (TCQ)

A small number of participants experienced difficulties in completing this questionnaire as they stated that they did not experience any 'unpleasant and/or unwanted thoughts'. This situation was addressed by discussing the participant's tendency to dwell on more positive thoughts and activities and complete the questionnaire after considering these phenomena. However, the participants' comments indicated an assumption within the study that all participants would experience unpleasant and/or unwanted thoughts when some participants considered themselves to be free from such thoughts.

4.4.4. Excluded variables

Although the study was specific in addressing certain variables in the experience of PTSD following an acute medical trauma, other important variables must be acknowledged, as they may constitute uncontrolled variance within the study.

Extra demographic variables such as marital status and occupational status were not recorded and physical illness variables such as clinical grade on admission, severity of condition, length of stay, procedure, uptake of rehabilitation services and, importantly for the subarachnoid haemorrhage sample, location of haemorrhage were not included in any subsequent analysis. Clearly, these variables may have impacted upon a participant's

subsequent emotional response to the acute medical trauma although this study did not address their effect.

Some extra variables have already been demonstrated as significant in experience of PTSD but are not addressed within this study. For example, awareness of the myocardial infarction at the time of the event and the severity of the condition was shown to be predictive of PTSD (Bennett & Brooke 99).

The role and perceptions of spouses and significant others have also been excluded from this study. Weinman et al. (2000) found that the causal attributions of spouses had a significant role in predicting health behaviour change within myocardial infarction patients, and this finding suggests that examination of spouses attributions may be an interesting area of research in this field.

4.4.5. Statistical considerations

As many of the statistical analyses required multiple analyses of several variables over 3 time points, the study needed to set higher levels for statistical significance for many of the individual analyses. Similarly, more conservative tests were conducted for some analyses in order to ensure that Type 1 error was reduced as much as possible. However, the small sample size involved in some of the analyses calls into question the robustness of the findings.

A further statistical issue concerns the regression analyses in which the DTS2 score (DTS at T2) was excluded as a predictor variable within the analyses. This decision was taken as the study had not specified an a priori hypothesis that PTSD at T2 would be predictive of PTSD at T3. However, it is acknowledged that the inclusion of DTS2 scores as a variable within the regression is likely to have affected the results considerably.

Finally, the study utilised predictive regression methods with scores at T2 being analysed for predictive capability of PTSD at T3. However, Lowe, Norman et al. (2000) detected a distinction between concurrent and predictive associations between coping, emotion and perceived health outcomes. Thus, concurrent regression analyses of independent variable measures at T3 to outcome variables at T3 may not correspond to the findings of the predictive regression analyses utilised in the current study.

Finally, whilst discussing the predictive capability of variables within the regression models, it is acknowledged that the statistical results of the regressions do not necessarily indicate causal relationships between the predictor variables and the dependent variable. Although results are discussed in terms of predictive relationships, the inference of causality must be considered as a theoretical interpretation of the results rather than a statistical fact.

4.4.6. Suggestions to enhance methodology

In order to improve on the reliability and validity of the current study, several changes may be made to the methodology.

Firstly, the sample size should be increased in order to make more robust claims about the results and increase the power of the statistical analyses.

Secondly, several changes may be made to the measures within the study. The IPQ-R may be modified to reflect the particular experiences of the subarachnoid haemorrhage and myocardial infarction samples. With respect to the Social Support Inventory, this also may be modified to counter the response bias mentioned in 4.4.3.2. Alternatively, another measure of social support such as the DUKE-UNC Functional Social Support questionnaire (Broadhead et al., 1988) may be used. The use of the DTS as the outcome measure for PTSD permitted more detailed analysis of the sub-scales of avoidant behaviour, hyperarousal and intrusions. The research hypotheses had not required analysis of these sub-scales although such analysis may yield interesting results to facilitate more detailed explanation of the phenomenology of PTSD.

Thirdly, consideration must be given to the number of questionnaires utilised within the study. Whilst the initial interview could be amended to incorporate additional basic demographic information quickly, the inclusion of so many measures within one study for a sample who may already be overwhelmed

with the tasks of restoring their normal daily functioning. This consideration confirms the requirement of specifying a priori hypotheses for research rather than exploring potential relationships in an unstructured manner.

4.5. Implications of the study

Having addressed some of the methodological limitations within the study, the implications of the findings for both clinical practice and future research will now be discussed.

4.5.1. Implications of study for clinical practice

The clinical implications for the findings of this study may be addressed under three topics; patient information requirements, early interventions for PTSD, and the assessment of and intervention for PTSD and cognitive avoidance behaviours.

4.5.1.1. Patient information requirements

Both myocardial infarction and subarachnoid haemorrhage patients may be given a large amount of information regarding their condition, the procedures undertaken, and the likely physical course of their recovery. However, issues remain regarding the timing of the provision of such information and the

provision of more detailed information regarding the emotional consequences of their conditions. Clearly, there are large variations in the provision of information across hospitals, but the findings of this study would imply that all hospitals and community services treating these patient groups may benefit from examining information booklets and clinical support procedures more closely. The finding that PTSD symptomatology is likely to increase after discharge and then decrease over the next two months may serve to reduce patients' concerns about this phenomenon. The finding that the illness representations of emotional representation, illness coherence, consequences, and treatment control are predictive of PTSD may encourage relevant clinicians to place emphasis upon these aspects of their information provision. This may be undertaken in order to normalise emotional responses, increase confidence in their knowledge of their condition, generate realistic expectations of the consequences of their condition, and reassure patients of the efficacy of treatment. Consideration may also be given to providing this information directly to GPs in discharge letters routinely. This may facilitate consistency in dealing with patients concerns about symptoms, and thus developing a more coherent understanding of the patient's condition and more confidence in the provision of treatment.

4.5.1.2. Early interventions for PTSD

The finding that 55% of those reaching caseness for PTSD at T2 go on to develop PTSD at T3 suggests that an early intervention for PTSD may be desirable. Whilst the provision of counselling support or psychological

debriefing for all survivors of trauma is a controversial area (Canterbury and Yule, 1999), there is a body of research to suggest that a focussed early intervention for PTSD using cognitive and behavioural techniques may benefit certain individuals (Litz, Gray, Bryant and Adler, 2002).

4.5.1.3. Assessment and interventions regarding cognitive avoidance behaviours in PTSD

Where individuals require more long-term treatment for enduring symptoms of PTSD, the results of the study that indicate the role of the thought control strategy of worry suggest that meta-cognitive techniques for modifying this strategy may be useful. Thus, clinical interventions directed towards modifying beliefs about the cognitive strategies utilised by individuals may be considered (Wells, 2000). The clinical techniques of detached mindfulness and attentional training may also be used with this sample in order to reduce the tendency to use worry as a cognitive avoidance strategy. Awareness of cognitive avoidance behaviours may also be useful in routine assessment for PTSD and in exposure based treatments.

As the current study has indicated that particular illness representations may have a role in the development and maintenance of PTSD symptoms in an acute medical trauma population, the use of a measure of illness representations (such as a modified IPQ-R) as a screening tool may also be recommended. Information obtained from this screening tool may be used to address patients' beliefs about their condition clinically.

4.5.2. Implications of study for future research

Methodological enhancements for improving the current study have already been considered (4.4.6). However, in order to extend knowledge regarding some of the mechanisms involved in predicting PTSD in an acute medical trauma sample, further research is suggested based on the findings and omissions of this study.

Firstly, a study of the predictive capability of the attributional style of participants for PTSD with an acute medical trauma sample is required. The psychosocial model of Williams and Joseph (1999) posits the role of appraisal style in maintaining PTSD. Whilst the current study has detected an effect for worry as a cognitive avoidance strategy in maintaining PTSD, the study has not enquired into the effect of attributional styles and metacognitive beliefs of individuals.

The study has not detected any effect for social support variables in the prediction of PTSD for this sample and this result is addressed in 4.3.6 and 4.4.3.3. Further research may examine this area using a more robust and valid method. To obtain a more comprehensive and accurate account of the role of social support, further research may examine the role of expressed emotion (a measure used to assess the quality of the relationship between an individual and their key relative (Leff and Vaughn, 1985)) and the perception of significant others alongside a questionnaire assessment of perceived social support.

Finally, as the current study has addressed the phenomenon of PTSD without addressing the individual components of intrusions, avoidant behaviour and hyperarousal, future research may examine the relationships between illness representations, social support, and thought control strategies for these separate sub-categories within PTSD. This may shed more light on the mechanisms by which PTSD is developed and maintained for an acute medical trauma sample.

CHAPTER 5: CONCLUSION

5. Conclusion

The present study aimed to investigate the relationship between illness representations and PTSD symptomatology, and changes in illness representations and PTSD over time. It also sought to explore the predictive capability of illness representations, social support and thought control variables for PTSD, and explore levels of PTSD anxiety and depression in an acute medical trauma sample composed of subarachnoid haemorrhage and myocardial infarction participants.

The results of the study partially support the research literature in suggesting that particular illness representations and cognitive coping strategies are associated with PTSD and that PTSD and particular illness representations change over time. Within this study, however, social support variables were not found to be significantly associated with PTSD and this result does not correspond with previous research literature. The results, therefore, provide support for the schema representation and coping strategy components of the psychosocial model of PTSD, but not for the environmental and social context component.

The results may have been affected by several methodological factors, including sample size and recruitment bias, response bias for the social support measure and the exclusion of certain important variables from the statistical analyses.

Future research in this area may be directed towards:

1. overcoming the methodological problems highlighted above;
2. exploration of the attributional style and metacognitive beliefs of myocardial infarction and subarachnoid haemorrhage patients and
3. investigation of the relationship between illness representation, social support, and cognitive coping strategy predictor variables and specific PTSD symptoms of intrusions, avoidance and numbing, and hyperarousal.

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APPENDICES

6. Appendices

6.1. Extracts from LREC approval letter.

LOCAL RESEARCH ETHICS COMMITTEE

Mr R Sheldrick
Trainee Clinical Psychologist
Academic Division of Clinical Psychology

29 June 2001

Dear Mr Sheldrick,

Predictors of emotional distress following myocardial infarction and subarachnoid haemorrhage: illness perceptions, social support, and thought control strategies.
Local Ethics Committee Reference 01/xxx.

Thank you for your letter of 6 June together with supporting papers. I have considered the amendments and /or documentation submitted in response to the Ethics Committee's earlier review of your application on 17 May 2001. Acting on behalf of the Committee I am now able to confirm final ethical approval for the study. The study should be started within three years of the date on which LREC approval is given.

The following items have been reviewed in connection with the study: ethical application form; revised information sheet; and consent form.

.....

Yours sincerely,

Xxxx xxxx

Chairman.

6.2. Extracts from R & D approval letters from Trust 1 and Trust 2.

Trust 1.

Mr R Sheldrick
Trainee Clinical Psychologist
Academic Division of Clinical Psychology

30 June 2001

Dear Mr Sheldrick,

Re: Predictors of emotional distress following myocardial infarction and subarachnoid haemorrhage: illness perceptions, social support and thought control strategies
Ethics Committee Reference 01/xxx

Thank you for your letter of 6 June and for sending me the LREC documentation relating to the above study as requested.

The research project has now been added to the xxxx R & D database and has been given R & D Management approval.
.....

Yours sincerely,

Xxxx xxxx
Head of Research and Development.

Trust 2.

6 July 2001

Dear Mr Sheldrick,

Re: Trust approval for Study

I am writing to confirm trust management approval for the research project "emotional distress in people with subarachnoid haemorrhage and myocardial infarction"
.....

Yours sincerely,

Xxxx xxxx
R & D Manager

6.3. Opt-In consent form for Consultant Cardiologists and Consultant Physicians at Trust 1.

Dear Dr ,

Re: Research into emotional distress following myocardial infarction and subarachnoid haemorrhage.

I am currently studying a postgraduate course in Clinical Psychology (Clin. Psy. D) at the University of Manchester.

The purpose of this letter is to request your support for some research I am intending to carry out to examine the relationship between emotional distress (particularly post-traumatic stress disorder) and health beliefs in myocardial infarction (MI) and subarachnoid haemorrhage (SAH) patient groups. The research has clinical utility in that a greater understanding of the relationship between emotional distress and the beliefs held by patients about their medical events may facilitate the emotional adjustment of patients in both groups. This research will also be in partial fulfilment of my Clin.Psy.D qualification.

Subject to approval from the Ethics Committee in May 2001, I hope to begin recruiting subjects into the study in June 2001 and finish recruitment in January 2002. The research would involve administering brief questionnaires to patients within two weeks of their MI or SAH and again 6 weeks and 3 months later. The SAH patients will be recruited from xxx. The MI patients are being recruited via the xxx, hence this letter to you as a Consultant Cardiologist or Consultant Physician responsible for the care of patients whom the study might wish to approach.

Ward staff would be asked to inform the researcher regarding the clinical suitability and safety of each potential patient being asked to undertake the questionnaire assessment. Involvement in the study would, of course, be entirely optional for patients. Completion of the questionnaires would be estimated to take approximately 40 minutes on average. Details concerning the questionnaires are available from myself. These will need to have been approved by the Ethics Committee before data collection can begin. A procedure will also be drawn up and agreed with the Ethics Committee for dealing with significant levels of emotional distress identified through the research assessment.

I would be grateful if you could indicate whether you agree on the above basis to your myocardial infarction patients being approached in the context of this research by completing the consent form below and returning it to me at the above address in the enclosed addressed envelope.

If you have any further questions regarding this letter, please do not hesitate to contact me on the above telephone number.

Yours faithfully,

Mr Russell Sheldrick.

Trainee Clinical Psychologist under supervision from Dr John Kincey

6.4. Information pack for Wards at Trust 1.

MYOCARDIAL INFARCTION STUDY

PURPOSE OF STUDY

This study is designed to explore the links between what people think about their illness (their Health Beliefs) and their emotional reactions to their illness over time.

It is hoped that the study will inform health professionals about the emotional experience of people who have suffered myocardial infarction and how this might affect the rehabilitation process. A comparative study looking at subarachnoid haemorrhage patients is being undertaken at the xxxx Hospital as part of this research.

OUTLINE OF STUDY AT xxxx

Myocardial Infarction patients will be asked to complete questionnaires concerning their health beliefs, depression and anxiety and post-traumatic stress. Data will be collected at three different times.

Once they are medically stabilised and just before discharge from hospital (interview and questionnaire completed with the researcher, Russell Sheldrick).
6 weeks after discharge (postal questionnaire) and
3 months after discharge (postal questionnaire).

CRITERIA FOR SELECTING PARTICIPANTS

For inclusion within the study patients must fulfil the following criteria:-

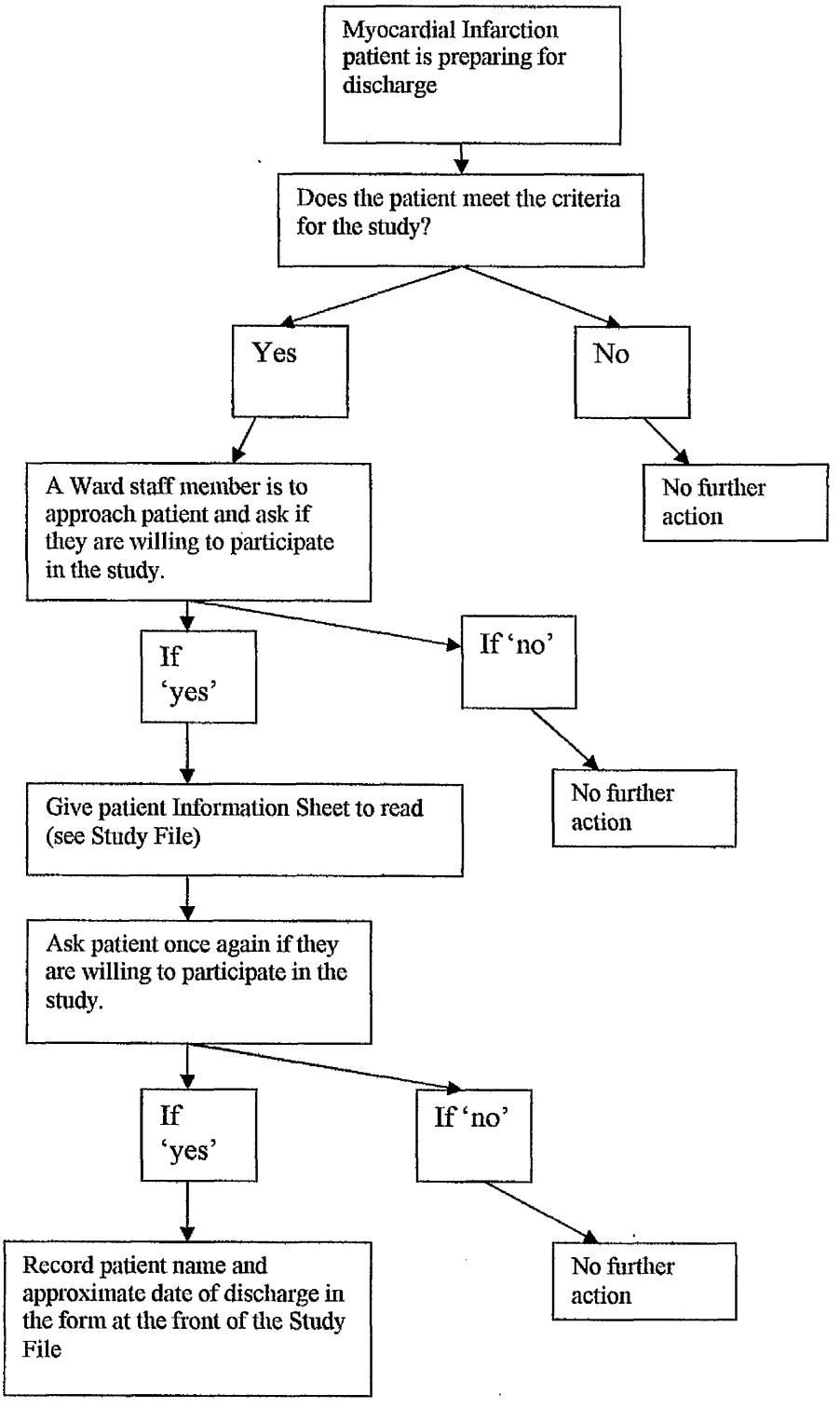
They must have had a myocardial infarction
They must be medically stable
They must speak and understand English
They must be fully orientated to the ward
They must be judged competent to give consent even if there may be mild cognitive disturbance.
They must not have severe dysphasia or hemiplegia.
They must be over 18.

The recruitment protocol is shown overleaf.

RECRUITMENT PROTOCOL

Ward staff are to consider the suitability of each myocardial infarction patient for participation within the study as they approach discharge from the hospital.

The following flow chart outlines how patients may be recruited into the study. The researcher, Russell Sheldrick, will ring the ward twice a week to find out which patients have been recruited.



CONTACTS

RESEARCHER:

Russell Sheldrick

Trainee Clinical Psychologist
Academic Division of Clinical Psychology

SUPERVISOR:

Dr John Kincey

6.5. Information pack for Wards at Trust 2.

SUBARACHNOID HAEMORRHAGE STUDY

PURPOSE OF STUDY

This study is designed to explore the links between what people think about their illness (their Health Beliefs) and their emotional reactions to their illness over time.

It is hoped that the study will inform health professionals about the emotional experience of people who have suffered a subarachnoid haemorrhage and how this might affect the rehabilitation process. A comparative study looking at myocardial infarction patients is being undertaken at the xxx as part of this research.

OUTLINE OF STUDY AT xxxx

Subarachnoid haemorrhage patients will be asked to complete questionnaires concerning their health beliefs, depression and anxiety and post-traumatic stress. Data will be collected at three different times.

Once they are medically stabilised and just before discharge from hospital (interview and questionnaire completed with the researcher, Russell Sheldrick).
6 weeks after discharge (postal questionnaire) and
3 months after discharge (postal questionnaire).

CRITERIA FOR SELECTING PARTICIPANTS

For inclusion within the study patients must fulfil the following criteria:-

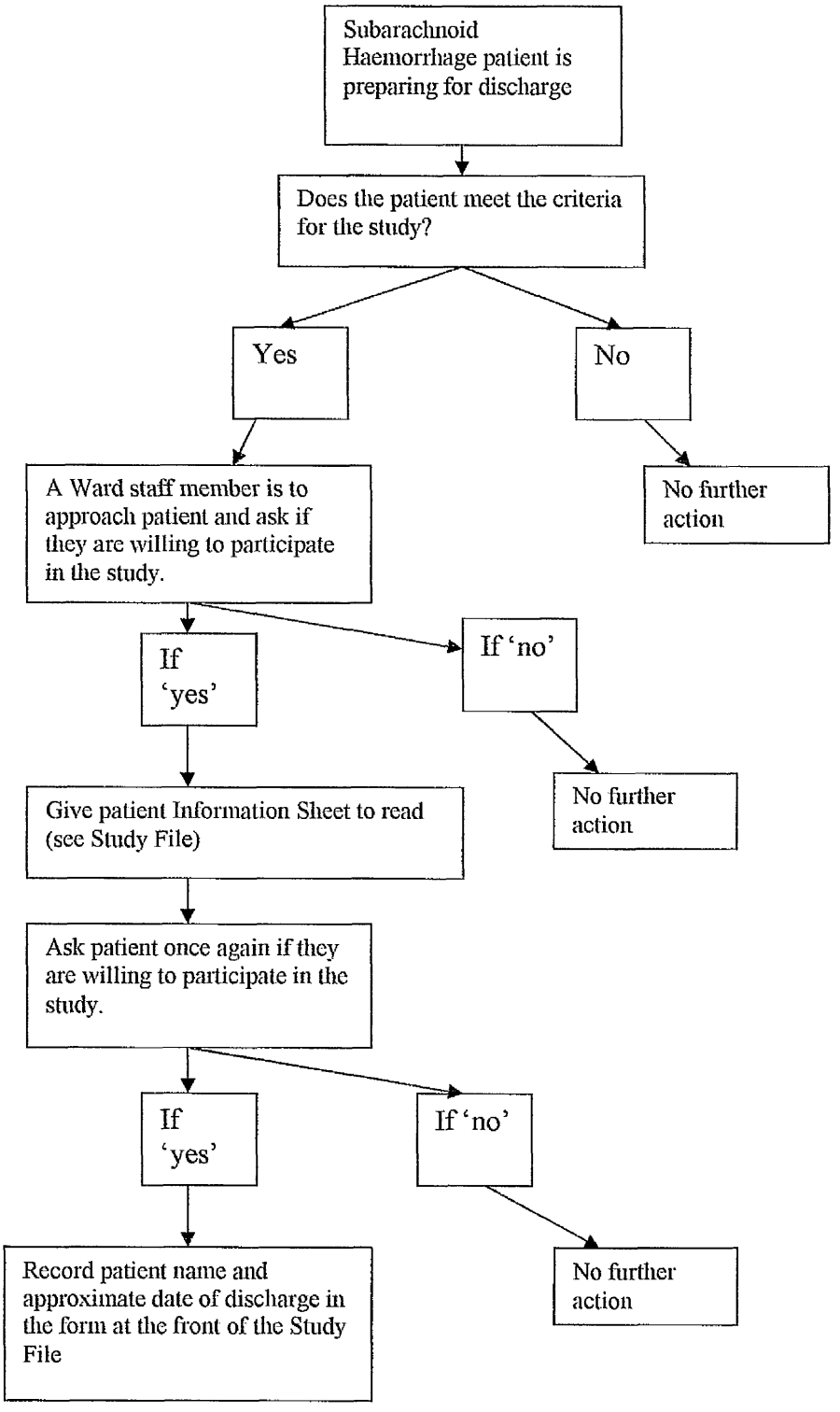
They must have had a subarachnoid haemorrhage
They must be medically stable
They must speak and understand English
They must be fully orientated to the ward
They must be judged competent to give consent even if there may be mild cognitive disturbance.
They must not have severe dysphasia or hemiplegia.
They must be over 18.

The recruitment protocol is shown overleaf.

RECRUITMENT PROTOCOL

Ward staff are to consider the suitability of each subarachnoid haemorrhage patient for participation within the study as they approach discharge from the hospital.

The following flow chart outlines how patients may be recruited into the study. The researcher, Russell Sheldrick, will ring the ward twice a week to find out which patients have been recruited.



CONTACTS

RESEARCHER:

Russell Sheldrick

Trainee Clinical Psychologist
Academic Division of Clinical Psychology

SUPERVISOR:

Elizabeth Berry

6.6. IPQ-R.

ILLNESS PERCEPTION QUESTIONNAIRE

YOUR VIEWS ABOUT YOUR ILLNESS

Listed below are a number of symptoms that you may or may not have experienced since your illness. Please indicate by circling 'YES' or 'NO' whether you have experienced any of these symptoms since your illness, and whether you believe that these symptoms are related to your illness.

	I have experienced this symptom since my illness		This symptom is related to my illness	
Pain	YES	NO	YES	NO
Sore Throat	YES	NO	YES	NO
Nausea	YES	NO	YES	NO
Breathlessness	YES	NO	YES	NO
Weight Loss	YES	NO	YES	NO
Fatigue	YES	NO	YES	NO
Stiff joints	YES	NO	YES	NO
Sore Eyes	YES	NO	YES	NO
Wheeziness	YES	NO	YES	NO
Headaches	YES	NO	YES	NO
Upset Stomach	YES	NO	YES	NO
Sleep Difficulties	YES	NO	YES	NO
Dizziness	YES	NO	YES	NO
Loss of strength	YES	NO	YES	NO

We are interested in your own personal views of how you now see your current illness. Please indicate how much you agree or disagree with the following statements about your illness by ticking the appropriate box.

	Views about your illness	STRONGLY DISAGREE	DISAGREE	NEITHER AGREE NOR DISAGREE	AGREE	STRONGLY AGREE
IP1	My illness will last a short time.					
IP2	My illness is likely to be permanent rather than temporary.					
IP3	My illness will last for a long time.					
IP4	This illness will pass quickly.					
IP5	I expect to have this illness for the rest of my life.					
IP6	My illness is a serious condition.					
IP7	My illness has major consequences on my life					
IP8	My illness is easy to live with.					
IP9	My illness does not have much effect on my life.					
IP10	My illness strongly affects the way others see me.					
IP11	My illness has serious financial consequences.					
IP12	My illness strongly affects the way I see myself as a person.					
IP13	My illness causes difficulties for those who are close to me.					
IP14	My illness has a negative impact on me.					
IP15	My illness is not a problem for me.					
IP16	My illness doesn't bother me much.					

	Views about your illness	STRONGLY DISAGREE	DISAGREE	NEITHER AGREE NOR DISAGREE	AGREE	STRONGLY AGREE
IP17	There is a lot that I can do to control my symptoms.					
IP18	What I do can determine whether my illness gets better or worse.					
IP19	Recovery from my illness is largely dependant on chance or fate.					
IP20	The course of my illness depends on me.					
IP21	Nothing I do will affect my illness.					
IP22	I have the power to influence my illness.					
IP23	My actions will have no affect on the outcome of my illness.					
IP24	My symptoms are beyond my control.					
IP25	My symptoms will be around whatever I do.					
IP26	My illness will improve in time.					
IP27	There is very little that can be done to improve my illness.					
IP28	My treatment will be effective in curing my illness.					
IP29	The negative effects of my illness can be prevented (avoided) by my treatment.					
IP30	My treatment can control my illness.					
IP31	There is nothing that can help my condition.					
IP32	The symptoms of my condition are puzzling to me.					
IP33	My illness is a mystery to me.					
IP34	I don't understand my illness.					
IP35	My illness doesn't make any sense to me.					

	Views about your illness	STRONGLY DISAGREE	DISAGREE	NEITHER AGREE NOR DISAGREE	AGREE	STRONGLY AGREE
IP36	I have a clear picture or understanding of my condition.					
IP37	The symptoms of my illness change a great deal from day to day.					
IP38	My symptoms come and go in cycles.					
IP39	My illness is very unpredictable.					
IP40	My illness condition is present all the time.					
IP41	I go through cycles in which my illness gets better and worse.					
IP42	I experience my illness symptoms pretty much all the time.					
IP43	The symptoms of my illness are distressing to me.					
IP44	I get depressed when I think about my illness.					
IP45	When I think about my illness I get upset.					
IP46	My illness makes me feel angry.					
IP47	My illness does not worry me.					
IP48	Having this illness makes me feel anxious.					
IP49	I worry a lot about my illness.					
IP50	My illness makes me feel afraid.					

CAUSES OF MY ILLNESS

We are interested in what YOU consider may have been the cause of your illness. As people are very different, there is no correct answer for this question. We are most interested in your own views about the factors that caused your illness rather than what others, including doctors or family, may have suggested to you. Below is a list of possible causes for your illness. Please indicate how much you agree or disagree that they were causes for you by ticking the appropriate box.

	Views about your illness	STRONGLY DISAGREE	DISAGREE	NEITHER AGREE NOR DISAGREE	AGREE	STRONGLY AGREE
C1	Stress or worry.					
C2	Hereditary – it runs in my family.					
C3	A germ or virus.					
C4	Diet or eating habits.					
C5	Chance or bad luck.					
C6	Poor medical care in my past.					
C7	Pollution in the environment.					
C8	My own behaviour.					
C9	My mental attitude, e.g. thinking about life negatively					
C10	Family problems or worries caused my illness.					
C11	Overwork.					
C12	My emotional state, e.g. feeling down, lonely, anxious, empty.					
C13	Ageing.					
C14	Alcohol.					

		STRONGLY DISAGREE	DISAGREE	NEITHER AGREE NOR DISAGREE	AGREE	STRONGLY AGREE
C15	Smoking.					
C16	Accident or injury.					
C17	My personality.					
C18	Altered immunity.					

In the table below, please list in rank-order the three most important factors that you now believe caused your illness. You may use any of the items from the box above, or you may have additional ideas of your own.

The most important causes for me:

_____

_____

_____

6.7. HADS.

HOSPITAL ANXIETY AND DEPRESSION SCALE

Please indicate how you are feeling now, or how you have been feeling in the past day or two, by ticking the column to the right of each of the following statements.

		YES DEFINITELY	YES SOMETIMES	NO, NOT MUCH	NO, NOT AT ALL
1	I wake early and then sleep badly for the rest of the night.				
2	I get very frightened or get panic feelings for apparently no reason at all.				
3	I feel miserable and sad.				
4	I feel anxious when I go out of the house on my own.				
5	I have lost interest in things.				
6	I get palpitations, or a sensation of 'butterflies' in my stomach or chest.				
7	I have a good appetite.				
8	I feel scared or frightened.				
9	I feel life is not worth living.				
10	I still enjoy the things I used to.				
11	I am restless and can't keep still.				
12	I am more irritable than usual.				
13	I feel as if I have slowed down.				
14	Worrying thoughts constantly go through my mind.				

6.8. TCQ.

THOUGHT CONTROL QUESTIONNAIRE

Most people experience unpleasant and/or unwanted thoughts (in verbal or picture form) which can be difficult to control. We are interested in the techniques that you generally use to control such thoughts.

Below are a number of things that people do to control these thoughts. Please read each statement carefully, and indicate how often you use each technique by circling the appropriate number. There are no right or wrong answers. Do not spend too much time thinking about each one.

When I experience an unpleasant/unwanted thought:

		NEVER	SOMETIMES	OFTEN	ALMOST ALWAYS
1	I call to mind positive images instead.	1	2	3	4
2	I tell myself not to be so stupid.	1	2	3	4
3	I focus on the thought.	1	2	3	4
4	I replace the thought with a more trivial bad thought.	1	2	3	4
5	I don't talk about the thought to anyone.	1	2	3	4
6	I punish myself for thinking the thought.	1	2	3	4
7	I dwell on other worries.	1	2	3	4
8	I keep the thought to myself.	1	2	3	4
9	I occupy myself with work instead.	1	2	3	4
10	I challenge the thoughts validity.	1	2	3	4
11	I get angry at myself for having the thought.	1	2	3	4
12	I avoid discussing the thought.	1	2	3	4
13	I shout at myself for having the thought.	1	2	3	4
14	I analyse the thought rationally.	1	2	3	4
15	I slap or pinch myself for having the thought.	1	2	3	4
16	I think pleasant thoughts instead.	1	2	3	4

		NEVER	SOMETIMES	OFTEN	ALMOST ALWAYS
17	I find out how my friends deal with these thoughts.	1	2	3	4
18	I worry about more minor things instead	1	2	3	4
19	I do something that I enjoy.	1	2	3	4
20	I try to reinterpret the thought.	1	2	3	4
21	I think about something else.	1	2	3	4
22	I think more about the more minor problems that I have.	1	2	3	4
23	I try a different way of thinking about it.	1	2	3	4
24	I think about past worries instead.	1	2	3	4
25	I ask my friends if they have similar thoughts.	1	2	3	4
26	I focus on different negative thoughts.	1	2	3	4
27	I question the reasons for having the thought.	1	2	3	4
28	I tell myself that something bad will happen if I think the thought.	1	2	3	4
29	I talk to a friend about the thought.	1	2	3	4
30	I keep myself busy.	1	2	3	4

6.9. SSI.

SOCIAL SUPPORT INVENTORY

We are interested in the amount of support you have received in the past from those close to you and the amount you expect to receive following your trauma.

Please read each question carefully. You will notice that below the question is a line with two extreme answers, one at each end. Please place a mark on the line that represents how you feel about the question.

HOW CRITICAL OF YOU IS THE PERSON CLOSEST TO YOU?

VERY CRITICAL----------NOT AT ALL

HOW CRITICAL ARE YOU OF THE PERSON CLOSEST TO YOU?

VERY CRITICAL----------NOT AT ALL

HOW SATISFACTORY IS THE EMOTIONAL SUPPORT YOU GET FROM THOSE CLOSE TO YOU?

VERY GOOD----------VERY POOR

HOW SATISFACTORY IS THE PRACTICAL SUPPORT YOU GET FROM THOSE CLOSE TO YOU?

VERY GOOD----------VERY POOR

WHAT TYPE OF SUPPORT DO YOU EXPECT TO GET FOLLOWING YOUR TRAUMA?

VERY GOOD----------VERY POOR

6.10. Patient Information sheet.

INFORMATION SHEET **12/7/01**

Emotional Distress after a Myocardial Infarction (Heart Attack).

Introduction

Having a heart attack is likely to be a very distressing experience. Part of recovering from such an experience may involve emotionally coming to terms with what has happened.

We would like to invite you to help us study peoples' emotional experience after a heart attack. We wish to look at whether certain factors in a person's life help them to cope with their experience emotionally.

What will I have to do if I take part?

The study will involve about 80 patients.
If you agree to take part you will be asked some questions by the researcher at three different times. To begin with you will be asked some questions by the researcher whilst you are still on the ward. Then, about six weeks after your admission to hospital, you will be sent five questionnaires together with a stamped addressed envelope to return the completed forms to the researcher. The questionnaires should take about half an hour to fill in. Finally, about 3 months after your admission you will be sent six questionnaires together with a stamped addressed envelope to return the completed forms to the researcher. These questionnaires should take about three quarters of an hour to fill in.

What are the possible risks of taking part?

It is possible that the questionnaires may cause you to think about what has happened to you and you may become upset. If this happens and you feel that it is difficult to carry on, then you are free to withdraw from the study at any point. You may wish to contact your Consultant or GP to discuss your concerns. If your doctor is concerned at any time he would take you out of the study

Are there any possible benefits?

The research does not offer any therapy for any emotional distress. However, if you feel that you require some help with the emotional consequences of your illness, the researcher will be able to inform the ward staff or your GP about this on your behalf.

Do I have to take part?

No, taking part is voluntary. If you would prefer not to take part you do not have to give a reason. Your doctor would not be upset and your treatment would not be affected. If you take part but later change your mind you can withdraw at any time.

Your medical records may be inspected by the researcher.
We would want to inform your GP that you are taking part, with your permission.

What do I do now?

The researcher will be available to discuss any concerns you have regarding the study and you can let him know if you are interested in taking part. You will then be asked to sign a consent form.

Thank you very much for considering taking part in our research. Please discuss this information with your family, friends, Consultant or GP if you wish.

Russell Sheldrick.
Trainee Clinical Psychologist
Department of Clinical Psychology
University of Manchester

Version 2

6.11. Consent Form.

CONSENT FORM

Title of Project: Emotional Distress following Myocardial Infarction and Subarachnoid Haemorrhage

Name of Researcher: Russell Sheldrick.

Please initial boxes

1. I confirm that I have read and understand the information sheet dated 12/7/01 (version 2) for the above study and have had the opportunity to ask questions. ☐

2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. ☐

3. I understand that sections of any of my medical notes may be looked at by responsible individuals from [company name] or from regulatory authorities where it is relevant to my taking part in research. I give permission for these individuals to have access to my records. ☐

4. I agree to take part in the above study. ☐

Name of Patient	Date	Signature
Name of Person taking consent (if different from researcher)	Date	Signature
Researcher	Date	Signature

1 for patient; 1 for researcher; 1 to be kept with hospital notes

6.12. Covering letter for T2 questionnaires.

Participant Name
Participant Address

DATE

Dear Participant,

Re: Research into Emotional Distress after a Subarachnoid Haemorrhage (Brain Haemorrhage).

Thank you for agreeing to take part in the above research project whilst you were in hospital recently and for completing the first set of questionnaires. I hope that you are still able to take part in the study.

I enclose the second set of questionnaires for you to complete and return in the stamped addressed envelope as soon as possible. There are five questionnaires altogether. If you have any difficulties completing the questionnaires please contact me at the above address.

I also enclose copies of the Information sheet for the study and the consent form that you signed when I met you.

I look forward to hearing from you in due course.

Yours sincerely,

Russell Sheldrick.
Trainee Clinical Psychologist

6.13. Reminder letter for T2 questionnaires.

Participant Name
Participant Address

DATE

Dear Participant,

Re: Research into Emotional Distress after a Subarachnoid Haemorrhage (Brain Haemorrhage).

I recently sent you a letter and some questionnaires to complete as part of the above study. Unfortunately, I have not yet received your reply. This may be because you no longer wish to be involved in the study in which case I apologise for troubling you with a further letter.

However, in case the letter has gone astray in the post I am including the questionnaires once again together with a stamped addressed envelope. There are five questionnaires altogether. If you have any difficulties completing the questionnaires, please contact me at the above address.

If I have not received a reply within the next two weeks, I shall assume that you wish to withdraw from the study.

Thank you for your help.

Yours sincerely,

Russell Sheldrick.
Trainee Clinical Psychologist

6.14. Covering letter for T3 questionnaires.

Participant Name
Participant Address

DATE

Dear Participant,

Re: Research into Emotional Distress after a Subarachnoid Haemorrhage (Brain Haemorrhage).

Thank you for agreeing to take part in the above research project whilst you were in hospital recently, and for completing both sets of questionnaires so far. I hope that you are still able to take part in the study.

I enclose the third and final set of questionnaires for you to complete and return in the stamped addressed envelope as soon as possible. There are five questionnaires altogether. If you have any difficulties completing the questionnaires, please contact me at the above address.

I look forward to hearing from you in due course.

Yours sincerely,

Russell Sheldrick.
Trainee Clinical Psychologist

6.15. Reminder letter for T3 questionnaires.

Participant Name
Participant Address

DATE

Dear Participant,

Re: Research into Emotional Distress after a Subarachnoid Haemorrhage (Brain Haemorrhage).

I recently sent you a letter and some questionnaires to complete as part of the above study. Unfortunately, I have not yet received your reply. This may be because you no longer wish to be involved in the study in which case I apologise for troubling you with a further letter.

However, in case the letter has gone astray in the post I am including the questionnaires once again together with a stamped addressed envelope. There are five questionnaires altogether. If you have any difficulties completing the questionnaires, please contact me at the above address.

If I have not received a reply within the next two weeks, I shall assume that you wish to withdraw from the study.

Thank you for your help.

Yours sincerely,

Russell Sheldrick.
Trainee Clinical Psychologist