A MIXED METHODS STUDY INVESTIGATING RE-PRESENTATION, SYMPTOM ATTRIBUTION and PSYCHOLOGICAL HEALTH IN PRIMARY PERCUTANEOUS CORONARY INTERVENTION PATIENTS

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SCHOOL OF NURSING, MIDWIFERY AND SOCIAL WORK
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ABSTRACT

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PhD
A MIXED METHODS STUDY INVESTIGATING RE-PRESENTATION, SYMPTOM ATTRIBUTION and PSYCHOLOGICAL HEALTH IN PRIMARY PERCUTANEOUS CORONARY INTERVENTION PATIENTS
2012

Introduction: Following ST-elevation myocardial infarction (STEMI) and treatment with Primary Percutaneous Coronary Intervention (PPCI), some patients re-present with potential ischaemic heart disease (IHD) symptoms. Symptoms may be related to cardiac ischaemia, reduced psychological health or a comorbid condition, which share similar symptoms and may lead patients to seek help via acute services. The purpose of the study was to investigate the proportion of PPCI patients who re-presented to acute services due to potential IHD symptoms within 6 months of STEMI, and to explore associated factors.

Methods: An explanatory mixed methods study was conducted. Quantitative data were collected at baseline and 6 months from consecutive patients attending two centres in Manchester. Variables were carefully considered based on a conceptual model for re-presentation. These included potential IHD symptom and psychological health assessments using self-report measures: the Seattle Angina Questionnaire (SAQ) and the Hospital and Anxiety and Depression Scale (HADS). Physiological health was measured using the Global Registry of Acute Coronary Events (GRACE) and the Charleson Comorbidity Index (CCI) at baseline. At 6 months re-presentation data were collected using patient records, a telephone interview and a self-report diary card. The experiences of some who re-presented (purposeful sampling) were explored through semi-structured interviews conducted at least 6 months following PPCI. Framework analysis was adopted to analyse data.

Results: 202 PPCI patients returned baseline questionnaires [mean age 59.7 years (SD 13.9), 75.7% male]; 38 (18.8%; 95% CI 14.0% to 24.8%) participants re-presented due to potential IHD symptoms at 6 months; 16 (42.1%) re-presented due to a cardiac event and 22 (57.9%) did not receive a diagnosis. At both baseline and 6 months, mean HADS anxiety scores were higher for the re-presentation group compared to the non-representation group (baseline 9.5 vs 7.1, p=0.006; 6 months 9.4 vs 6.0, p<0.001). Angina symptoms were stable and infrequent at both time points for the groups. Multivariate regression modelling with the inclusion of predictors HADS anxiety, SAQ angina stability, SAQ angina frequency, GRACE and CCI, determined HADS anxiety as a predictor of re-presentation with an adjusted odds ratio of 1.12 (95% CI 1.03 to 1.22, p=0.008). The qualitative interviews with re-presenters included 25 participants (14 men, 27-79 years). Four themes were identified: fear of experiencing a further heart attack, uncertainty and inability to determine cause of symptoms, insufficient opportunity to validate self-construction of illness and difficulty adapting to life after a heart attack.

Conclusion: Elevated levels of anxiety at baseline were predictive of re-presentation with potential IHD symptoms at 6 months. Factors such as shock at experiencing a heart attack, hypervigilance of symptoms and difficulty with symptom attribution appeared to play a role in raised anxiety levels for the re-presentation group. Findings suggested that changes are needed to cardiac rehabilitation and post-STEMI follow-up to address educational needs and psychological issues and changes in STEMI treatment.
DECLARATION

No portion of the work referred to in the 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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## Abbreviations

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<td>A &amp; E</td>
<td>Accident and emergency</td>
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<td>ACS</td>
<td>Acute coronary syndrome</td>
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<td>AMI</td>
<td>Acute myocardial infarction</td>
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<td>CABG</td>
<td>Coronary Artery Bypass Graft</td>
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<td>CCI</td>
<td>Charleson Co-Morbidity Index</td>
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<td>CHD</td>
<td>Coronary Heart Disease</td>
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<td>CI</td>
<td>Confidence Interval</td>
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<td>CR</td>
<td>Cardiac Rehabilitation</td>
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<td>DoH</td>
<td>Department of Health</td>
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<td>ECG</td>
<td>Electrocardiograph</td>
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<td>GP</td>
<td>General practitioner</td>
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<td>GRACE</td>
<td>Global Registry of Acute Coronary Events</td>
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<td>HADS</td>
<td>Hospital Anxiety and Depression Scale</td>
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<td>HR</td>
<td>Hazard Ratio</td>
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<td>HRQoL</td>
<td>Health Related Quality of Life</td>
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<td>ICD</td>
<td>International Classification of Diseases</td>
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<td>Ischemic Heart Disease</td>
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<td>Percutaneous Coronary Intervention</td>
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<td>PPCI</td>
<td>Primary Percutaneous Coronary Intervention</td>
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<td>PTSD</td>
<td>Post Traumatic Stress disorder</td>
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<td>QoL</td>
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<td>STEMI</td>
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<td>WHO</td>
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CHAPTER 1 INTRODUCTION

1.1 Coronary Heart Disease

The most common cause of death in the United Kingdom (UK) is Coronary Heart Disease (CHD) with as many as 88,000 deaths per year (Scarborough et al., 2010). In 2008 more than 12,000 people under the age of 65 died due to CHD (Scarborough et al., 2010). However, in recent years CHD death rates have been falling. It is reported that between 1998 and 2008 the fall in mortality rates for men (55 to 64 years) was 49.0% and for women 55.0%, although the reduction is much smaller for younger individuals (Scarborough et al., 2010). The reduction in mortality rates has been attributed to improved secondary prevention and improved treatments.

Coronary Heart Disease is caused by the formation of atherotamous plaque in the walls of the coronary arteries and these continue to progress over many years (Mehran et al., 2000). They often do not cause a problem until the coronary artery is obstructed by 50.0% or more and this can lead to symptoms such as chest discomfort (also known as angina) or breathlessness with exertion (Mehran et al., 2000). In these instances patients often suffer from stable angina and may receive pharmacological treatment or if symptoms are sufficiently severe coronary revascularisation may be required. Angina is defined as chest discomfort related to narrowing of the coronary artery or arteries due to atherotamous plaque (Mehran et al., 2000).

However, for some patients a sudden catastrophic change in the atherotamous plaque occurs. The plaque becomes unstable and the fibrous cap covering it abruptly erodes or ruptures with subsequent thrombus formation leading to either partial or total occlusion of the coronary artery (Antman et al., 2004). In this instance the initial diagnosis is often acute coronary syndrome (ACS), including Myocardial Infarction (MI) or ‘heart attack’ (Mehran et al., 2000). For some individuals this is the first evidence that they have CHD and it often involves acute presentation to emergency services. Currently 124,000 heart attacks occur each year in the UK (Scarborough et al., 2010).

The term ACS is often used as an umbrella term incorporating a number of diagnoses including unstable angina, Non-ST Elevation Myocardial Infarction (NSTEMI) or ST-Elevation Myocardial Infarction (STEMI) (Lloyd-Jones et al., 2009). In the case of N-STEMI partial occlusion of the coronary artery occurs, which may
alter electrical conductivity in the heart, but does not lead to elevation of the ST segment of the electrocardiograph (ECG) (see section 1.2.1). Conversely total occlusion of the coronary artery does lead to elevation of the ST segment (STEMI) as indicated on the ECG; the changes on the ECG along with clinical signs and symptoms are used when making a diagnosis.

1.2 ST-Elevation Myocardial Infarction

Total occlusion of the coronary artery during ST-Elevation Myocardial Infarction (STEMI) leads to damage or necrosis (death) of the myocardium resulting in an acute life threatening event (DeWood et al., 1980, Thygesen et al., 2007, Alpert et al., 2000). Complete necrosis of the affected area of myocardium occurs within approximately 2 to 4 hours from onset of myocardial ischaemia (Alpert et al., 2000). It is estimated that STEMI affects over 27,000 people in the UK per year (Herrett et al., 2010).

Healing of the myocardium takes up to 6 weeks following STEMI and during this time scar tissue develops in the affected area of the myocardium. Scar tissue does not possess contractile cells and therefore reduced contractility of the affected area of myocardium may lead to varying degrees of reduced left ventricular function, reduced ejection fraction and in some cases heart failure (Sabia et al., 1991).

Early diagnosis and initiation of treatment, involving re-establishing coronary blood flow (known as coronary reperfusion), limits damage to the myocardium and reduces long term morbidity and mortality (Antman et al., 2004, Boersma et al., 1996). The main treatment for STEMI currently is primary percutaneous coronary Intervention (PPCI) (see section 1.4).

1.2.1 Diagnosing STEMI

Diagnosis of STEMI is achieved through multiple means including assessment of clinical presentation, recent clinical history and 12 lead electrocardiography (ECG) changes (Antman et al., 2004).

The 12 lead ECG is a non-invasive test that measures the rhythm and electrical activity in the form of depolarisation (electric discharge) and repolarisation (electric recharging) of the heart (Houghton and Gray, 1997). Ten leads, comprising of four limb leads (one on each limb) and six chest leads, are placed on the body using electrodes. Twelve views of the heart’s electrical activity are derived from the 10
leads and are named; I, II, III, aVR, aVL, aVF for the limb leads, and V₁, V₂, V₃₄, V₅, and V₆ for the chest (Sgarbossa and Wagner, 2007).

When a heart beat occurs, a systematic wave of depolarisation (originating in the sinoatrial node) progresses through the atrium and across the ventricles. This is followed by repolarisation or recharging of the heart, also often referred to as the resting phase (Sgarbossa and Wagner, 2007).

The small changes in voltage (measured in millivolts (mV)) detected across the leads during depolarisation and repolarisation are recorded on the ECG as the P, Q, R, S, T and U waves (Sgarbossa and Wagner, 2007). These are generally displayed as wavy lines on a print out from the ECG machine (Houghton and Gray, 1997).

The definition of STEMI on ECG includes either ST segment elevation in two or more contiguous leads (≥0.2 mV in leads V₁, V₂, V₃₄, or ≥0.1 mV in other leads), or new left bundle branch block (Chou and Knilans, 1996). The location of the infarct (i.e. the area of the heart affected) such as anterior, inferior, posterior and posterior-lateral, is determined by ST-elevation in certain ECG leads. Additionally the degree of myocardial infarction is depicted by the size of the ST-elevation in each lead and the number of leads showing ST-elevation.

Complete necrosis of the myocardium can be seen on ECG as a ‘Q’ wave infarction (Blumgart et al., 1940, Wilson et al., 1933). However, ‘Q’ wave can also indicate non-acute past MI due to previous damage to the myocardium (Thygesen et al., 2007).

Serum biochemical markers are also used following treatment, to indicate the amount of myocardial damage that has occurred. Furthermore, for individuals treated with PPCI, angiography is conducted immediately prior to PPCI to confirm STEMI (Antman et al., 2004).

### 1.2.2 Signs and symptoms of STEMI

A third of patients have abrupt sudden onset of symptoms at the time of STEMI (Schmidt and Borsch, 1990). The symptoms and severity as well as the onset time are all important factors; for an event to be classified as acute generally symptoms are experienced for more than 20 minutes (Bardales et al., 1996). The signs and symptoms of STEMI reported by patients may be ‘typical’ or ‘atypical’ and in some instances STEMI may be ‘silent’, in other words there is an absence or lack of recognition of symptoms (Cohn et al., 2003).
In this study typical symptoms are described in accordance with international guidelines: including The American College of Cardiology (ACC) and American Heart Association (AHA) guidelines for the management of patients with STEMI and those set out by the European Society of Cardiology (ESC)/ACC task force for the redefinition of MI (Antman et al., 2004, Thygesen et al., 2007). However, it is important to acknowledge that currently there is some debate in the literature as to what constitutes typical or atypical STEMI symptoms (Body et al., 2010, DeVon et al., 2011). This is because few studies have been conducted to establish what constitutes typical symptoms and those described by Antman et al (2004) are founded on level B evidence (defined by the authors as limited evidence).

1.2.2.1 Typical symptoms
Antman et al (2004) described STEMI symptoms to include discomfort of the chest in the form of pain, pressure or tightness, with or without radiating to the arm(s). Additional symptoms include jaw, back, neck or epigastria pain, shortness of breath, weakness, diaphoresis, nausea and light headedness. Furthermore, symptoms may occur on exertion or at rest, they may also be accompanied by syncope and tend not to be positional or localised, and are unaffected by movement (Thygesen et al., 2007).

1.2.2.2 Atypical symptoms
Symptoms that are reported to be less typical for STEMI include arm, shoulder, wrist or upper back or upper abdominal pain without the occurrence of chest pain (Alpert et al., 2000, DeVon et al., 2011). Additional atypical symptoms are fatigue, weakness, palpitations, indigestion, stroke like symptoms in the form of numbness or unexplained confusion and a sense of fear (Alpert et al., 2000, DeVon et al., 2011, Canto et al., 2009).

In a review of the literature it has been reported that women are more likely to present with atypical symptoms at the time of an ACS event than men (DeVon et al., 2011). This may be due to the involvement of mainly male cohorts in early CHD studies, leading to representation of typical symptoms that are related to the male STEMI experience (Emslie, 2005). Additionally, women tend to be older than men when they experience an ACS event and the elderly typically report more atypical symptoms (Blomkalns et al., 2005, Thuresson et al., 2005).

DeVon et al (2011) report that women are more likely than men not to experience chest pain during an ACS event. Furthermore, women more frequently report upper
back and upper abdominal pain, palpitations, syncope, weakness, fatigue, indigestion and fear (DeVon and Zerwic, 2003, DeVon et al., 2008, McSweeney et al., 2001).

1.3 Reperfusion treatment

During the past decade two main methods of establishing coronary reperfusion were available. In the early part of the decade the treatment of choice was pharmacological, with the use of powerful thrombolytic drugs ideally delivered within four to 6 hours of the onset of pain.

More recently primary percutaneous coronary intervention (PPCI) has become the preferred treatment due to evidence of its superiority (Keeley et al., 2003). More than 27 randomised trials have indicated the advantages of PPCI compared to thrombolysis (Keeley et al., 2003). Superiority even extends to when PPCI is delayed following transfer of patients from the community setting (District Hospital) to a PCI centre (Dalby et al., 2003). Most importantly there is evidence of a reduction in mortality rates, smaller infarct size and lower rates of recurrent ischaemia (Smith, 2004). A reduction in hospital stay, fewer re-admissions and lower associated costs are also reported (Hartwell et al., 2005).

1.4 Primary Percutaneous Coronary Intervention

It is recommended by the ACC/AHA that a target of 90 minutes from initial medical contact to cardiac reperfusion be achieved (Antman et al., 2004). When PPCI is initiated early, often ST-elevation is completely resolved leading to minimal damage and in many cases full recovery of the myocardium (De Luca et al., 2003, Berger et al., 1999).

PPCI is a non-surgical minimally invasive procedure to open the occluded artery and to allow full reperfusion of the myocardium. The PPCI procedure is done with the use of opaque dye under x-ray conditions and access is gained to the arterial system via either the femoral or radial artery. Once access has been gained and a catheter has been passed into the arterial and then coronary system, angiography is performed to identify the culprit occluded coronary vessel. Following this a guide wire is then passed with the purpose of reaching the narrowed or occluded coronary artery (Kornowski et al., 2000). The narrowed lumen is then widened by forcing the offending atheromatous plaque outwards with the use of a small inflated balloon. In some instances clots of blood may also be mechanically removed. A stent is then
implanted to act as a rigid structure and aid the artery to remain patent (Lansky and Leon, 2000).

1.4.1 Development of a PPCI service

During the latter half of the last decade PPCI was introduced across the UK as the treatment of choice during STEMI. Initially there were logistical challenges to be overcome including changes to the way the ambulance service operated and also the need for a rapid response, 24 hour, seven days per week, on call catheter lab service (Dalby et al., 2003). In 2005 the Department of Health commissioned a PPCI feasibility study ‘The National Infarct Angioplasty Project (NIAP)’ to formally investigate the implementation of PPCI. The study included seven pilot sites, and Manchester was chosen as one of these sites.

Initially the Manchester PPCI service was limited and only included the two PPCI centres, (Central Manchester University Hospitals NHS Trust and University Hospital of South Manchester) and a number of District General Hospitals (DGH) that transferred patients for treatment. Furthermore, the service ran during limited times (8 am to 4pm, five days per week).

The service has evolved over the duration of the past six years and now runs 24 hour, seven days per week across the two PPCI centres and includes the transfer of patients from 11 DGH Trusts. Some of the DGH transfer patients travel more than 15 miles at the time of STEMI to receive treatment.

1.4.2 PPCI patient care

Until quite recently transfer patients were not relocated directly to the PCI centre following the onset of symptoms. Initially they were transported by emergency ambulance to the local accident and emergency (A&E) department where initial assessment and basic treatment in the form of opiates and oxygen were commenced. This normally took no more than 10 minutes before patients were urgently dispatched by emergency ambulance to the PCI centre. It is important to note that transfer to the PCI centre frequently occurred without a family member escorting the patient. In most instances the journey time took approximately 15 to 30 minutes. On arrival at the PCI centre patients were met at the hospital doors by the emergency team and transferred directly to the catheter lab.

Patients who lived within the catchment area of the Manchester PCI centres were transferred by emergency ambulance directly from home to the PCI centre. They were initially seen in the resuscitation area of the A&E department (an area which is
reserved for critically ill patients), where assessment and confirmation of diagnosis took place within 5 to 10 minutes of arrival. Patients were then transferred with two or three members of staff quickly to the catheter lab.

On arrival at the catheter lab the patient would be given a brief description of PPCI treatment, including the risks of the procedure, and written consent would be taken. Again often a family member or friend would not be with the patients at this point. On average the PPCI procedure commences within 10 minutes of patients arriving at the catheter lab.

Following PPCI, patients were generally sent to coronary care, which is set-up to deliver specialist care to acutely ill cardiac patients. Discharge home usually occurred early, generally within three to five days post PPCI. Following discharge, most patients received medical review at six to eight weeks (although this varies dependant on the treating cardiologist) and the opportunity to attend cardiac rehabilitation at eight to 12 weeks post infarction.

### 1.5 Post PPCI re-presentation

Overall PPCI is a highly successful treatment and few patients experience post discharge complications (Aplin et al., 2009). However, some patients do experience further cardiac events or ongoing cardiac ischemic symptoms (Mehran et al., 2009). This may be related to further diseased arteries, as it is usual to only treat the infarct related artery at the time of PPCI (Hannan et al., 2010). Additionally, restenosis (or re-narrowing) of stents used during PCI although rare, may result in additional events or symptoms with the potential of patients re-presenting to acute healthcare services (Popma et al., 2002, Abizaid et al., 2000). Literature pertaining to recurrent cardiac events is discussed in section 2.3.

Furthermore, following STEMI some patients also suffer poor psychological adaptation and in some cases reduced psychological health (Strik et al., 2003, Grace et al., 2004, Frazier et al., 2002). In some instances reduced psychological health manifests itself as physical symptoms that mimic cardiac ischaemia and may lead to the patient re-presenting to acute services (Moser and Dracup, 1996, Parashar et al., 2006, Dammen et al., 2004). The literature related to psychological health following STEMI is presented in section 2.5 and chest pain/potential IHD symptoms is reported in section 2.11.
1.6 The need for a study

In clinical practice during the early development of PPCI the author noticed that there was a propensity for PPCI patients to attend acute services with symptoms similar to those they associated with their STEMI event. These patients also appeared to be extremely distressed when re-presenting with possible ischaemic heart disease (IHD) symptoms. There was a dearth of literature pertaining to patients who had experienced PPCI and re-presented with potential IHD symptoms.

Due to the patients’ previous STEMI they were categorised by healthcare professionals as ‘high risk’ of experiencing a further cardiac event. Admission to hospital and multiple investigations would then ensue. However, despite investigations, often ambiguity remained regarding the cause of the patients’ re-presentation symptoms. The number of PPCI patients re-presenting in this manner was unknown and the underlying reasons related to the re-presentation event were also unclear. The author therefore decided to undertake a study to investigate the number of patients re-presenting to emergency services with possible ischaemic symptoms post PPCI. Furthermore, it was decided to investigate the potential factors associated with re-presentation post PPCI. The author also wished to explore the experiences of those PPCI patients who re-presented to acute services, with a view to improving the care provided.

1.7 Organisation of the thesis

This thesis is divided into ten chapters. The first two chapters (chapters 1 and 2) describe the rationale for the study, the background and review of the relevant literature. Chapter 1 outlines the clinical aspects of ST-elevation myocardial infarction (STEMI) and associated care. The empirical and theoretical literature related to STEMI, psychological health, potential IHD symptoms and re-presentation are described in Chapter 2. Chapter 3 presents the conceptual framework for the study and the variables considered and chosen for inclusion in the logistic regression models.

Aspects of the research methodology are described in chapters 4 to 6. The methodology related to the mixed methods is described in Chapter 4. The quantitative and qualitative methodologies are outlined in more detail in chapters 5 and 6 respectively. Chapters 7 and 8 present the quantitative and qualitative study findings.
The thesis concludes in Chapter 9 with synthesis of the main study findings and discussion in context of the wider literature. Evaluation of the study methods, the strengths and weaknesses of the research and potential future studies are identified along with recommendations for policy and practice.
CHAPTER 2 LITERATURE REVIEW

2.1 Review of the literature

This chapter describes the literature most relevant to the conduct of this study. Review of clinical, psychological and re-presentation literature relating to STEMI and PPCI patients are presented.

The methods used in conducting the literature review and the overall findings of the literature search are presented in Appendix A.

2.2 PPCI the patient journey

Patients have reported feeling anxious and distressed at the time of STEMI and primary percutaneous coronary intervention (PPCI) (Astin et al., 2009). This is not surprising when the severity of STEMI symptoms and the seriousness of the medical condition are taken into consideration. Reports of the patients’ experiences related to PPCI gives healthcare professionals some insight of the patients’ journey and the potential factors associated with the patients’ recovery.

2.2.1 PPCI a curative treatment?

Astin et al (2009) explored the experiences of 29 first time STEMI patients through qualitative interviews three to 12 days post-PPCI. In the qualitative phase of this mixed methods study, PPCI patients reported experiencing feelings of fear when they were initially told that they were experiencing a heart attack (Astin et al., 2009). The authors also evaluated the illness perceptions of those interviewed using the Illness Perception Questionnaire (IPQ-R) (Moss-Morris et al., 2002). Participants were purposively selected to include men (n=16) and women (n=13), as well as younger and older individuals (36 to 78 years). Recruitment took place at one PPCI centre which treated patients from the local catchment area and also those from nearby District General Hospitals (DGH); all patients were admitted directly from home.

This analysis reported that participants were shocked that they had suffered a heart attack and their feelings were exacerbated due to the speed of PPCI treatment. Additionally, participants reported being uncertain as to what had occurred during the acute event and a mismatch between expectations and reality was exhibited. PPCI treatment was seen as curative and participants viewed their condition as ‘acute’ rather than ‘chronic’ (Astin et al., 2009).
In this study the authors noted that it was normal clinical practice to repatriate patients to their local DGH once they were stable. However, the study findings did not indicate whether those interviewed had been repatriated. Repatriation may have influenced the participants’ experiences and it was not possible to contextualise the study findings in relation to this factor. A further limitation of the study was that participant experiences were limited to just one PPCI centre. This does not take into consideration the varied practices across other centres, which are likely to influence the patient experience.

Participants of another qualitative study also reported viewing PPCI treatment as curative (Sampson et al., 2009). In this study 10 PPCI patients and six carers were interviewed in their homes one to four weeks following patient discharge. The participants were purposefully selected across two PPCI centres; one centre accepted only direct admissions and the other treated both direct and transfer admissions. The researchers used maximum diversity sampling to ensure the inclusion of a range of times of admission and admission routes, as well as different ages and both genders (Sampson et al., 2009).

In this study participants reported feeling ‘fixed’ and had difficulty accepting that they had experienced a heart attack (Sampson et al., 2009). The fast speed of change in their physical condition, from extremely ill to feeling ‘normal’ again, was contrary to their expectation of how they would feel following a heart attack (Sampson et al., 2009). Participants found it difficult to comprehend that they had experienced a heart attack.

The participants and their carers were all extremely positive regarding the care that had been delivered and appeared reluctant to offer any reports that may reflect elements of dissatisfaction. Similarly, the participants in Astin et al (2009) study were also grateful and very satisfied with the care that they had received.

A limitation of Sampson et al (2009) study was that the authors did not provide any evidence of data saturation. Additionally, the sample was small with only 16 interviewees (10 patients and six carers), suggesting that maximum diversity of the sample may not have been achieved despite the authors intimating that this had been the case. Furthermore, the length of the interviews appeared relatively short with a mean of 33 minutes (15 to 56 minutes), indicating that the richness and depth of the data may have been lacking.
Previous research conducted by Hirani et al (2006) supports the theory that some CHD patients receiving PCI believe that they have been cured. Hirani et al (2006) study included 214 CHD patients who were treated with conservative therapy (n=70), PCI (n=71) and coronary artery bypass graft (CABG) (n=73). The New Zealand Heart Attack Recovery project version of the Illness Perception Questionnaire (NZ-IPQ) was used to evaluate how illness representations varied within CHD patients receiving the different treatments. The NZ-IPQ contained 16 core items in line with the original IPQ (Weinman et al., 1996). The remainder of items appeared to be similar to those of the IPQ; the reference given for the NZ-IPQ by Hirani et al (2006) in their publication was a personal communication with Petrie (June 14, 1995). The researchers found that patients treated with either CABG or PCI believed that they were ‘cured’ and that their illness had a shorter course compared to individuals treated conservatively with medication (Hirani et al., 2006).

The studies conducted by Astin et al (2009) and Sampson et al (2009) offer some insight of the patients’ viewpoint regarding their experiences of PPCI. The studies suggest that the participants were shocked and found it difficult to understand that they had experienced a heart attack. Furthermore, participants seemed to believe that they were cured following treatment.

2.2.2 Psychological preparation and education of PPCI patients

Education and psychological preparation prior to surgery and invasive procedures have been shown to reduce levels of psychological distress, leading to improved recovery during and after the procedure (Anderson, 1987, Mumford et al., 1982, Curtis., 2000, Sime, 1976, Wallace, 1985). This may also be the case for patients receiving PPCI.

Krohne, et al (1996) not only highlighted the importance of educating patients but went one step further in reporting that patients coped best during a stressful event when the amount and type of information is matched to their coping style. For example a ‘vigilant coper’ prefers high levels of information and an ‘avoidant coper’ requires low levels of information (Krohne et al., 1996). This literature reinforces the early findings of Shaw et al (1986) who report that elective PCI patients are at increased risk of medical complications when a mismatch of coping styles and levels of information received occurs (Shaw et al., 1986).
Prior to PPCI generally patients receive only minimal information. This is because patients are usually feeling very unwell and they have received opiates. Additionally, there is little time available to deliver information as it is important to establish reperfusion of the myocardium quickly (within six hours of onset of pain) to limit damage to the myocardium (De Luca et al., 2003, Berger et al., 1999). Post-event ‘debriefing’ through education and counselling during hospitalisation is also reduced due to the shortened in-patient stay (Koch et al., 1999). Bed rest for PPCI patients is only several hours and hospital stay a matter of days (Koch et al., 1999, Higgins et al., 2000). Clearly the opportunity for clinicians to match the patients’ coping style and the appropriate amounts and types of information is limited throughout the current PPCI patient journey.

In the qualitative aspect of Astin et al. (2009) mixed methods study (previously discussed in section 2.2.1) participants were also asked about their information needs following PPCI (Astin et al., 2008). In this study participants were seen at home by a community cardiac rehabilitation nurse (three to five days post discharge). The participants who did not receive such a visit believed that there had been a gap in care and also reported feelings of heightened uncertainty. The researchers found that the participants preferred to receive face to face information backed up with written information and preferred to be seen at home (Astin et al., 2008). A variety of timing of information delivery was preferred: some individuals required it early following admission whereas others could not absorb information at this point due to the shock of the event. Overall, the participants’ emotional shock at having had a heart attack and the speed of treatment was a barrier to absorbing information. The participants’ main information needs in this study concerned how they might recognise subsequent symptoms relating to a further cardiac event (Astin et al., 2008).

2.2.3 Cardiac rehabilitation following PPCI

Cardiac rehabilitation (CR) is offered to a range of heart patients throughout the UK including PPCI patients. Other coronary heart disease (CHD) groups include individuals with heart failure, non-ST elevation myocardial infarction (N-STEMI) and those treated with coronary artery bypass graft (CABG) surgery and percutaneous coronary intervention (PCI) (British Heart Foundation, 2011).

The CR service has been developed to help patients reduce behavioural related risk factors such as smoking, poor exercise and poor dietary habits (Jamieson et al., 2002). It is also an opportunity to receive psychological support through regular
contact with healthcare professionals. Generally CR is run by specialist CR nurses and may also include a cardiac physiologist or physiotherapist with specialist skills. In some areas of the UK, patients also may receive a home visit by the CR nurse soon after discharge, although this is not routine service across the UK (Astin et al., 2008). On the whole, patients start on a programme six to eight weeks following a cardiac event and the programme may last between 10 and 16 weeks. Patients may be asked to attend either once or twice a week (British Heart Foundation, 2011). In some areas, such as Manchester, patients also receive a phone call between three and five days post-discharge following an acute cardiac event. Programmes vary across the UK, but generally include tailored physical exercises, and education and information regarding medications and aspects of heart disease (British Heart Foundation, 2011).

One area which receives less attention during rehabilitation programmes is the treatment of anxiety and depression (Hughes, 2011). Anxiety and depression are routinely assessed during rehabilitation clinics in the UK, as recommended by The National Service Framework (NSF) for CHD (a guidance document for the care of patients in the UK) (National Service Framework, 2000). Despite this, in practice patients rarely receive referral for psychological conditions (Hughes, 2011). This is regardless of the known deleterious effects raised anxiety and depression levels have in patients following STEMI (see sections 2.6.2 and 2.7.2) (Moser and Dracup, 1996, Frasure-Smith et al., 1993, Grace et al., 2004).

The reported benefits of CR are far reaching and include reduced mortality rates in patients following MI and reduced hospitalisation rates (Oldridge et al., 1988, O'Connor et al., 1989, Black et al., 1998). Additionally, improvement in quality of life (QoL), reduced angina levels and reduced time for patients to return to work have also been reported (Hedback et al., 2001, Petrie et al., 2002, Heller et al., 1993, Black et al., 1998). However, attendance at CR has been reported to vary between 41.0% and 60.0% for MI patients (Petrie et al., 1996, Melville et al., 1999, Lane et al., 2001). A systematic review was conducted by Cooper et al (2002) to investigate the reasons for non-attendance at CR for MI patients and other cardiac groups (e.g. coronary artery bypass surgery, percutaneous coronary intervention and angina). The review included 15 studies and reported that non-attendees were more likely to be older, to have lower income and to deny the severity of their illness. They were also less likely to perceive that their doctor had recommended attendance at CR or believe that they could influence the outcome of their illness (Cooper et al., 2002).
Currently the CR service offered to PPCI patients mirrors the services received by other cardiac groups and it is likely that PPCI patients experience the same benefits as others who attend. However, presently the effectiveness of rehabilitation services has not been evaluated for PPCI patients.

2.2.4 Summary

Following PPCI, patients are reported to express fear and shock at having experienced a heart attack (Astin et al., 2009). These negative emotional constructs appeared to be compounded by the speed of the PPCI treatment (Astin et al., 2009). In two studies the participants reported feeling extremely satisfied with PPCI treatment, but were left with a sense of disbelief at what had occurred (Astin et al., 2009, Sampson et al., 2009). In one study participants could not believe that they have experienced a heart attack and viewed PPCI as ‘curative’ and believed that they were ‘fixed’ (Sampson et al., 2009).

The opportunity to inform and educate PPCI patients about CHD and the treatment that they have received is minimal during admission because of the fast delivery of PPCI and early discharge. Participants’ emotional shock at having had a heart attack and the speed of treatment has been indicated as a barrier to absorbing information (Astin et al., 2008). Additionally, the educational and information needs of patients vary widely, particularly in relation to the timing of delivery (Krohne et al., 1996, Astin et al., 2008).

Cardiac rehabilitation (CR) is offered to PPCI patients on average six to eight weeks following discharge. It is an opportunity to address cardiac risk factors and gain knowledge of CHD and treatments as well as receiving support from specialist trained healthcare professionals (British Heart Foundation, 2011). The monitoring of psychological health is a recommended component of CR (National Service Framework, 2000). However, referral of patients for psychological interventions through CR services is reported to be poor (Hughes, 2011).

The benefits of CR for those who attend include reduced mortality, fewer cardiac symptoms and improved quality of life (Oldridge et al., 1988, O’Connor et al., 1989, Black et al., 1998). However, effectiveness and benefits specifically gained by PPCI patients have not as yet been evaluated. It is possible that they may vary for this group due to the speed of treatment and their beliefs that the treatment is curative.
2.3 Recurrent cardiac events

This part of the review presents the relevant literature related to cardiac events following STEMI. Since the introduction of PPCI, mortality and repeat cardiac event rates have declined (Keeley et al., 2003). However, post-STEMI patients are still at risk of experiencing repeat cardiac events, which may result in them re-presenting to acute healthcare services.

The majority of cardiac event rates for PPCI study cohorts are reported through randomised controlled trials (RCT). Generally, those reported are composite event rates known as multiple adverse cardiac events (MACE), which often combine death, re-infarction, revascularisation and stroke. The literature for this section is summarised in Appendix B, section B.1.

2.3.1 Post STEMI mortality and cardiac events

A clinical trial, the CADILLAC study, investigated the use of antiplatelet therapy and stent usage in 2082 PPCI patients (Stone et al., 2002). The authors reported MACE rates (including death, stroke, re-infarction and revascularisation) of 14.6% at 6 months (Stone et al., 2002). The incidence of re-infarction alone was only 2.1% at 6 months in this study. In a further RCT of 3602 PPCI patients, again receiving a variety of treatments including stents and antiplatelet therapy, the MACE rate (death, stroke, re-infarction and revascularisation) was 11.9% (included 4.8% all cause death) at one year (Mehran et al., 2009). The authors also described a further event rate of 3.8% which included only cardiac death at one year.

It is interesting that the MACE rate in Mehran et al (2009) study is much lower at one year than that of the CADILLAC study at 6 months. This is likely to be because the two studies were undertaken almost a decade apart with Mehran et al (2009) study being conducted more recently. Over recent years advancements in PPCI techniques and improved adjunctive therapies as well as new pharmacological remedies have steadily reduced mortality and cardiac event rates.

Further event rates for PPCI patients have been reported by numerous clinical studies. These involve a variety of treatment options, including direct comparisons of PPCI and different thrombolytic therapies, as well as adjunctive drug therapies, stenting and transfer of patients for PPCI.

In the ADMIRAL study a re-infarction rate of 2.0% at 6 months, and MACE rates of 10.3% at 30 days and 11.7% at 6 months were reported (Montalescot et al.,
2001). Slightly lower rates for MACE of 4.8%, 5.8% and 8.0% are described at 30 days for the studies GUSTO IIb, PAMI and PRAGUE respectively; these rates only include PPCI patients (Stone et al., 1998, Grines et al., 1999, GUSTO IIb angioplasty substudy investigators, 1997, Widimský et al., 2003). The 30 day follow-up data taken from these studies are of limited value as they only allow a partial view of the patients’ recovery. All of these studies were again conducted more than a decade ago, with the possibility of an overestimation in MACE rates when extrapolated to current PPCI cohorts. Moreover, the event rates reported by Mehran et al (2009), Stone et al (2002) and the other investigators are unlikely to reflect those found in a general PPCI population. This is because the study cohorts were highly selected due to the clinical study designs. Cohorts that are unselected and representative of the general PPCI population will be referred to in this section of the thesis as ‘representative’.

It was demonstrated in a recent study comparing the characteristics of a representative PPCI population with those of an RCT population (taken from the DANAMI-2 trial) that outcome was worse for the representative group (Jakobsen et al., 2010, Andersen et al., 2003). The authors were interested to compare the outcomes of patients who did not meet the DANAMI-2 trial entry criteria with those that did (Andersen et al., 2003). From a review of medical case notes they found that patients from the representative population had a more adverse risk profile at baseline and poorer outcomes than the trial participants. After one and two years the cumulative incidence of MACE (all-cause mortality, MI, and stroke) was higher in the representative population (17.8% and 22.0%) compared to the DANAMI-2 population (13.6% and 17.3%) (Jakobsen et al., 2010). When the representative group included both those who did and did not meet the DANAMI-2 entry criteria, rates of re-infarction at one and two years were similar between the groups (representative, 7.3% and 8.9% and DANAMI-2, 6.0% and 8.5%).

Observational studies reporting cardiac event rates and including heterogeneous representative PPCI populations are relatively rare. However, in one such observational study of 1832 STEMI patients treated with either thrombolysis (lysis) (n=392) or PPCI (n=1440), the combined death and cardiac event (heart failure or MI) rate at one year was 13.5% for thrombolysis and 13.6% for PPCI patients (Lambert et al., 2010). In this Canadian study conducted across 80 acute care hospitals, the researchers set out to investigate the association of reperfusion treatment delivery timelines with clinical outcome. Study data were captured retrospectively directly from medical records by a certified medical record librarian.
and the STEMI diagnosis was confirmed by a cardiologist who reviewed the first admission ECG for each patient included in the study. Although the combined event rate in this study reflect those likely to be apparent in a clinical cohort, there may have been some inaccuracies in the reported event rates due to the retrospective data collection used in the study.

A further representative PPCI cohort was investigated in a study recently published as an abstract and presented at the British Cardiac Society conference (Austin et al., 2011). The authors investigated whether the route of admission (i.e. direct admission or inter-hospital transfer) for PPCI treatment influenced outcome (Austin et al., 2011). This was a retrospective study based in the north of England with the inclusion of 2268 STEMI patients: 510 (22.5%) transfer patients from a non-PPCI centre and 1758 (77.5%) direct admissions.

The authors reported baseline (in-hospital) MACE (death, re-infarction or stroke) of 4.8% for the total cohort (6.7% for transfer patients and 4.3% for direct admissions). At one year mortality rates were 9.7% for transfer patients and 7.0% for direct admissions (Austin et al., 2011). One year MACE rates are not stated and additionally rates of re-infarction are not reported at either baseline or one year.

### 2.3.2 Summary

Following STEMI, patients are at risk of mortality and recurrent cardiac events that may lead them to attend acute healthcare services with cardiac ischaemic symptoms. Mortality and cardiac event rates following PPCI are often identified through RCT cohorts and are frequently presented as composite events rates, making interpretation and relevance to representative PPCI cohorts difficult. However, such results do offer some indication of the event rates for PPCI cohorts.

The incidence of patients experiencing composite events (death, re-infarction, revascularisation and stroke) reported through clinical trials is between 4.8% and 10.3% at 30 days, 11.7% to 14.6% at 6 months and 11.9% at 12 months (GUSTO IIb angioplasty substudy investigators, 1997, Montalescot et al., 2001, Mehran et al., 2009). Re-infarction is low for PPCI patients with a rate of only 2.0% at 6 months being reported in RCTs (Stone et al., 2002, Montalescot et al., 2001).

Higher composite event rates (all-cause mortality, MI, and stroke) are reported for cohorts that are representative of general PPCI populations: 4.8% in-hospital, 17.8% at 12 months and 22.0% at two years (Austin et al., 2011, Jakobsen et al., 2010). The incidence of re-infarction is higher than those presented through RCTs,
but is still relatively low (7.3%) at 12 months for representative PPCI cohorts (Jakobsen et al., 2010).

2.4 Biological factors

During an acute coronary syndrome (ACS) event such as STEMI, biological changes occur in the body. These include an intense inflammatory response, increased platelet activation and changes to hormones often related to emotional responses (Koukkunen et al., 2001, Suleiman et al., 2006). Biological markers (bio-markers) are used to assess the level of change in the body and these include inflammatory bio-markers (C-reactive protein, interleukin (IL) 6 and tumour necrosis factor (TNF)α), as well as hormones related to emotional response (serotonin, norepinephine and cortisol) (Morrow et al., 1998, Koukkunen et al., 2001, Suleiman et al., 2006).

The role of inflammatory response in ACS has attracted increasing attention from researchers. An association has been determined between the magnitude of increase in inflammatory bio-markers and increased mortality and recurrent cardiac ischaemia over median 17 to 23 months post MI (Koukkunen et al., 2001, Suleiman et al., 2006). This may be related to a relationship between heightened inflammatory responses with increased heart rate, and decreased heart rate variability that have previously been reported (Kleiger et al., 1987, Kaplan et al., 1987). Increased heart rate has been linked to the progression of CHD and reduction in heart rate variability has been associated with increased cardiac mortality and morbidity following MI (Kleiger et al., 1987, Bigger et al., 1992, Kaplan et al., 1987, Hamaad et al., 2005).

Acute coronary syndrome and acute psychological distress have been shown to share similar biological changes. Increases in inflammatory bio-markers, similar to those seen during ACS, occur within one to two hours of acute psychological distress (Koukkunen et al., 2001, Steptoe et al., 2007). Additionally, cortisol is influenced by psychological distress and also plays a part in limiting inflammatory response (Nijm and Jonasson, 2009, Cannon, 1929).

Further pathophysiological processes are jointly associated with the development and progression of CHD and psychological distress (anxiety and depression) (Smith et al., 2007, Watkins et al., 2002, Musselman et al., 1996). This includes processes that influence cardiac arrhythmic mechanisms such as diminished baroflex cardiac control (Smith et al., 2007, Watkins et al., 2002). Cardiac arrhythmias have been
reported as one of the main causes of early death following STEMI (Frasure-Smith et al., 1993, Frasure-Smith et al., 1995a). Therefore, pathophysiological factors influencing arrhythmic control may go some way to explaining the increased mortality rates in those suffering psychological distress following STEMI (see section 2.5) (Carney et al., 2005, Musselman et al., 1998). Additionally, it has been reported that increases in platelet activation occur during episodes of depression, which may also play a part in mortality following STEMI (Musselman et al., 1996).

The potential relationship between inflammation, emotional hormones, behavioural factors, heart rate variability, psychological distress and CHD outcome have been investigated in 'the Heart and Soul study’ (Martens et al., 2010). More than a thousand stable CHD outpatients were followed up over five years and outcome was assessed in terms of developing an ACS event (Martens et al., 2010, Robins et al., 1981). At baseline general anxiety disorder was assessed using the computerised Diagnostic Interview Schedule for DSM-IV (Robins et al., 1981). Emotional hormones including serotonin, norepinephrine and cortisol were measured and additionally high-sensitivity C-reactive protein (CRP) was used as a measure of inflammation. Heart rate variability was also measured at baseline. Behavioural factors such as physical activity levels, adherence to medication, alcohol intake and smoking were also assessed through self-report questionnaires. Participants were then evaluated yearly for five years, with regards to experiencing a new cardiac event.

The authors found that general anxiety disorder (GAD) was associated with increased risk of cardiovascular events with a hazard ration of 1.62 (95% CI 1.11-2.37; p<0.01) this was following adjustment for demographics, comorbidity (including depression), severity of cardiac disease and adherence to medication. However, no association was found between raised anxiety and emotional hormones, the inflammatory bio-marker and behavioural factors (Martens et al., 2010). The negative findings related to anxiety and inflammation in this study may have been related to the use of only one inflammatory bio-marker (CRP), other markers may have elicited alternative findings. Additionally, the study cohort was drawn from a stable CHD outpatient population rather than those who had recently experienced an ACS event. Levels of inflammation and emotional hormones are unlikely to have been equivalently raised to those during an ACS event.

In a more recent study Steptoe et al (2011) investigated whether intense acute psychological distress and elevated inflammation were an integrated biobehavioural reaction during an ACS event. The researchers established the level of acute
distress and fear of dying in 208 ACS patients through interviews conducted within 2.56 ± 1.5 days of admission (Whitehead et al., 2005). Samples of blood were taken on admission to measure inflammatory markers and samples of saliva were collected over the course of a day to assess the level of cortisone. The researcher found that those who reported intense distress and fear of dying also experienced higher levels of inflammatory biological response and reduced cortisol production (Steptoe et al., 2011).

2.4.1 Summary

Inflammatory biological changes measured through bio-markers, occur during an ACS event. The magnitude of rise in these biomarkers has been found to indicate deleterious outcome during follow-up (Koukkunen et al., 2001, Suleiman et al., 2006). Similarly, inflammatory biomarkers are also released shortly after intense psychological stress, as are hormones related to emotional stress such as cortisol (Koukkunen et al., 2001, Steptoe et al., 2007, Nijm and Jonasson, 2009). Additionally, pathophysiological processes related to cardiac arrhythmic mechanisms and platelet aggregation have also been reportedly associated with ACS and psychological distress (Carney et al., 2005, Musselman et al., 1998).

The Heart and Soul study did not find any relationship between inflammatory biomarkers, ‘emotional’ hormones and general anxiety disorder in the occurrence of cardiac events in a stable group of CHD patients (Martens et al., 2010). However, in a more acute group, ACS patients with intense distress had increased inflammatory biomarkers and reduced cortisol levels, suggesting a link between intense distress and biological changes (Steptoe et al., 2011).

2.5 Psychological health

Psychological health is known to be negatively affected for up to 50.0% of patients following STEMI (Frazier et al., 2002, Mayou et al., 2000, Frasure-Smith, 1991). In particular anxiety and depression reportedly lead to deleterious effects in STEMI patients (Frasure-Smith et al., 1993, Moser and Dracup, 1996). Additionally, anxiety and depression have been shown to coexist or to act as a comorbidity for each other (Bech, 2006, Greist and Jefferson, 1995, Zimmerman et al., 2000).

This section of the review describes the classification system for psychological/psychiatric conditions, as well as the symptoms associated with anxiety and depression. Additionally literature pertaining to the relationship between anxiety and depression are described. Appendix B, section B.2 presents a
summary of the most pertinent literature relating to psychological health in patients with CHD or chest pain. Additionally Appendix C presents a summary of common self-report instruments used to assess psychological health.

2.5.1 Classification of psychological and psychiatric disorders

In Europe classification of mental and behavioural disorders are made using the ‘International Classification of Diseases’ (ICD), constructed by the World Health Organisation (WHO). ICD-10 sub-section F32 identifies depressive episode, F33 recurrent depressive disorder, F41 anxiety disorders and F41.2 identifies mixed anxiety and depressive disorder (World health Organisation, 2006).

In the United States diagnosis is usually made using the ‘Diagnostic and Statistical Manual of Mental Disorders- Fourth Edition, Text Revision (DSM-IV-TR)’; it is often referred to as the DSM and it is published by the American Psychiatric Association (American Psychiatric Association, 2000). The DSM categorises psychiatric diagnoses into five dimensions (Axis I to Axis V), Axis I refers to clinical disorders such as depression and anxiety disorders (American Psychiatric Association, 2000).

2.5.2 Signs and symptoms of anxiety and depression

2.5.2.1 Anxiety disorders

One of the major symptoms of anxiety is a sense of foreboding with expectation that something unpleasant is occurring or about to happen (American Psychiatric Association, 2000). Further symptoms include feeling on edge, restlessness, difficulty in concentrating and relaxing.

Somatic symptoms include palpitations, chest pain, trembling, dizziness, abdominal discomfort and nausea (American Psychiatric Association, 2000). Physiological changes are attached to these sensations with increased levels of norepinephrine leading to tachycardia, raised blood pressure and respiration rate (American Psychiatric Association, 2000). Generally diagnosis is made when symptoms have been present daily for a number of consecutive weeks, over several months.

In some individuals anxiety becomes chronic and has serious consequences relating to their activities of daily living, this is classified as anxiety disorder. Anxiety disorder is an ‘umbrella’ term which includes such conditions as general anxiety disorder (GAD), obsessive compulsive disorder (OCD), post traumatic stress disorder (PTSD), phobias and panic disorder (PD) (American Psychiatric Association, 2000).
2.5.2.2 Depression

Depression involves a lowering of mood and reduced activity which interferes with an individual’s daily activities (American Psychiatric Association, 2000). An episode of depression is described as mild, moderate or severe and a two week duration of symptoms is usually required for diagnosis (American Psychiatric Association, 2000).

As well as a lowering of mood and reduced physical activity symptoms may include insomnia, anorexia, guilt, sadness, irritability, fatigue, difficulty concentrating, lethargy, loneliness, tearfulness, poor sex drive, inability to enjoy things, being short tempered, weight loss, worthlessness and a reduction in self-esteem and self-confidence (American Psychiatric Association, 2000). When severe depression occurs individuals may also experience suicidal thoughts (WHO, 2006).

2.5.3 The relationship between anxiety and depression

Almost half of the general population suffer an episode of psychiatric disorder involving either anxiety or mood at some point in their life (Kessler et al., 2005).

Anxiety and depression are considered to be strongly associated and this is partly due to the overlap of symptoms between the two disorders (Bech, 2006). Either one of the disorders may be classed as the primary disorder (i.e. primary anxiety or primary depression) and for some individuals both disorders occur concurrently (Bech, 2006).

2.5.3.1 Primary anxiety

General anxiety disorder (GAD) has been reported to affect 4.1% of the United States population as a lifetime disorder for ages (Grant et al., 2005). It has also been found that up to 23.0% of those with GAD suffer panic disorder (PD) (Zimmerman et al., 2000). Additionally, primary anxiety may lead to depression, particularly in those with functional limitations due to their anxiety (Zimmerman et al., 2000). Seminal works have previously reported that 8.0% to 39.0% of those with GAD experience concurrent depression (Wittchen et al., 1994, Kessler et al., 1994).

2.5.3.2 Primary depression

In the general population depression is reported to affect 12.0% of men and 25.0% of women at some point in their life (Tighe, 2006). Moreover, the prevalence of current general anxiety disorder in those suffering primary depression is reportedly
15.0% to 21.0% (Rush et al., 2005, Zimmerman et al., 2000). Additionally, Zimmerman et al. (2000) showed that almost half of depressed patients attending a psychiatric outpatient facility suffered some kind of anxiety disorder. More than a decade earlier Hamilton (1989) reported that almost all (97.5%) of 499 depressed subjects were found to suffer anxiety and 87.4% experienced somatic anxiety.

The concurrent occurrence of anxiety during depression can in some individuals obscure the depression altogether and in others have serious consequences (Rush et al., 2005, Sims, 2006). Schaffer et al. (2000) report that depressed patients with an anxiety disorder are more susceptible to suicidal ideation than those who do not have anxiety.

2.5.4 Anxiety and depression: one entity or separate?

The similarity between symptoms of anxiety and depression such as fatigue, insomnia or sleep disturbance, poor concentration and restlessness, often leads to difficulty in separating the two disorders (Bech, 2006). This has led to debate in the literature regarding whether anxiety and depression should be treated separately or as a combined disorder (Gotlib, 1984, Dobson, 1985, Feldman, 1993, Endler et al., 1998, McWilliams et al., 2001, Endler et al., 2003). The traditional view is that the two are distinct constructs with overlapping features (Endler et al., 2003, McWilliams et al., 2001, Rush et al., 2005). However, other researchers have previously suggested that a model combining the symptoms of anxiety and depression and known as 'general distress' disorder is more appropriate (Clark and Watson, 1991, Watson et al., 1995b, Watson et al., 1995a). Currently evidence in the literature appears to indicate that anxiety and depression occur concurrently and one acts as a comorbidity for the other (Rush et al., 2005).

The psychological health section of the literature review is summarised in combination with the other psychological health sections in section 2.9.

2.6 Anxiety

Approximately half of patients following STEMI experience raised anxiety, which is associated with further cardiac ischaemia, re-infarction and cardiac mortality (Frazier et al., 2002, Grace et al., 2004, Frasure-Smith, 1991, Moser and Dracup, 1996).

Some of the symptoms of anxiety mimic those of cardiac ischaemia and it can be difficult for patients to differentiate between symptoms (Dammen et al., 2004, Ros
et al., 1997). This may result in patients seeking help through acute healthcare services when symptoms occur that the patient believes to be cardiac related (Moser et al., 2006).

### 2.6.1 Prevalence of anxiety in STEMI patients

At the time of an ACS event 37.0% of patients reportedly suffer elevated or threshold anxiety scores, and following STEMI between 45.0% and 59.0% suffer extreme to moderate anxiety levels (Strik et al., 2003, Grace et al., 2004, Frazier et al., 2002). At present there are no published reports relating to the prevalence of anxiety in PPCI patients, although studies have investigated anxiety in STEMI and ACS patients.

In a Dutch study conducted by Strik et al (2003) rates of anxiety as high as 59.5% were reported for men at one month (baseline) following their first STEMI and rates of depression as 47.1% (Strik et al., 2003). Anxiety and depression symptoms were assessed using the 90-item Symptom Check List (SCL-90) (Derogatis et al., 1973): for description of psychological assessment instruments. In this study 137 (43.1%) patients received invasive treatment (thrombolysis, PCI or CABG) for STEMI during admission and of those 59.9% suffered heightened symptoms of anxiety at baseline; similar levels (59.2%) were reported for those not receiving an intervention (Strik et al., 2003). This study is also discussed in sections 2.6.2.2 and 2.7.2.3.

The proportion of patients receiving PCI (or PPCI) during admission was not described and accordingly levels of anxiety and depression related to type of intervention were not presented. A limitation of the study was that only men who had experienced their first STEMI were included. The highly selective study cohort limits the ability to generalise the study findings to the wider STEMI population.

Frazier et al (2002) investigated the association between STEMI and anxiety using the Spielberger State Anxiety Inventory (SAI) (Spielberger, 1993) in 101 patients. The participants completed the SAI within 48 hours of their admission for STEMI and 21.8% reported extreme, 24.8% moderate and 22.8% mild levels of anxiety. The treatment received for STEMI in this study was not described. However, due to the timing of the study it is most likely that patients received thrombolysis.

In a further study including a heterogeneous cohort of 906 ACS patients (424 unstable angina and 482 MI patients) 36.6% of participants reported elevated or threshold anxiety scores (Grace et al., 2004) (also discussed in section 2.7.1 and
2.12.3.2). The purpose of this study was to examine anxiety symptomatology at the time of ACS and over the following 12 months. The researchers also investigated the type of IHD symptoms that patients experienced during their ACS event. Anxiety was assessed (between two and five days following admission for the ACS event) using two self-report measures, the Middlesex Hospital Questionnaire (MHQ)-Phobic Anxiety Subscale and the Anxiety Subscale of the PRIME-MD (Spitzer et al., 1994, Crown and Crisp, 1996).

Many of the symptoms reported in Grace et al (2004) study were consistent with an episode of severe anxiety in the form of panic disorder (PD). This was reported in 11.0% of patients at baseline, 10.0% at 6 months and 7.0% at 12 months post event. In 80.4% of those with PD, recurrent cardiac events were also recorded during the one year follow-up. The authors also reported that psychological sequelae following STEMI declined over time. Grace et al (2004) found that in 50.0% of patients with raised anxiety levels at two to five days post STEMI, levels completely resolve by 12 months.

2.6.1.1 Prevalence of anxiety according to gender
The prevalence of anxiety has been shown to be higher for women (9.7%) in the general population than for men (4.7%) (Regier et al., 1990). Likewise, anxiety has also shown to be more prevalent in women than men following MI (Lane et al., 2002, An et al., 2004, Frasure-Smith et al., 1999, Moser et al., 2003).

Frasure-Smith et al (1999) found in their study of gender, depression and one year MI prognosis, that women had significantly higher anxiety scores than men during index hospital admission. Additionally, in two further studies investigating anxiety in MI patients, women were again significantly more likely than men to register high state anxiety scores at 72 hours (Moser et al., 2003) and six days (mean) (Lane et al., 2002) following MI.

2.6.2 The consequences of raised anxiety in CHD patients
Researchers report close links between raised anxiety levels and increased death rates due to CHD, both in the general population and following STEMI (Kawachi et al., 1994, Frasure-Smith, 1991, Roest et al., 2010). Furthermore, a relationship between anxiety and recurrent cardiac events has also been reported following STEMI (Moser and Dracup, 1996).
2.6.2.1 Anxiety and mortality post MI

In a recent meta-analysis including 12 papers, Roest et al (2010) investigated the relationship between anxiety and outcome in MI patients. The authors identified an association between anxiety and poor prognosis following MI with a 36.0% increased risk of cardiac and all cause mortality and cardiac events combined (the main combined end point). No heterogeneity was seen for the 12 studies reporting the combined end point and the pooled odds ratio (OR) 1.36 (95% confidence interval, 1.18 to 1.56; p<0.001) across the studies for 1649 anxious compared to 4101 non-anxious MI patients. In the case of STEMI, mortality was associated with raised anxiety within three months of the acute event.

The meta-analytic review demonstrated that there was some disparity in the findings related to anxiety and mortality and cardiac events; some studies showed an association between anxiety and poor prognosis and some did not (Roest et al., 2010). There was no real pattern related to the papers that did and did not find a statistical significant association between anxiety and outcome. The studies reporting an association participant numbers ranged from 87 to 896, anxiety was measured at a variety of time points (during admission to two months post discharge) and age ranged from 55 to 60 years. Similarly in the studies reporting no association between anxiety and poor outcome, participant numbers ranged from 65 to 2449 and anxiety was measured ≤3 days and 6 to 60 days post MI, and age ranged between 54 to 63 years.

There appeared to be a greater association between anxiety and cardiac events than with mortality. Only one paper reported a significant association between anxiety and cardiac mortality whereas two reported an association with mortality or recurrent cardiac events in multivariate analysis (Roest et al., 2010). This may have been attributed to treatments improving over time.

A more recent study, the Heart and Soul Study, was published after the Roest et al (2010) meta-analysis (Martens et al., 2010). Martens et al (2010) found that general anxiety disorder (GAD) was associated with increased risk of cardiovascular events (including stroke, heart failure, MI, peripheral ischaemia and death) with an adjusted relative risk of 1.62 (95% CI 1.11-2.37; p<0.01) (Martens et al., 2010). This study has also been discussed previously in section 2.4.
2.6.2.2 Anxiety and cardiac complications post MI

Several studies have found a relationship between anxiety and recurrent cardiac ischaemia and cardiac complications (Moser and Dracup, 1996, Strik et al., 2003, Moser et al., 2007, Cherrington et al., 2004).

In a study involving 318 men who had suffered their first MI, multivariate Cox regression analysis identified anxiety as an independent predictor of cardiac events (hazard ratio 2.79, 95% CI 1.11 to 7.03; p=0.029). Almost 8.0% (25) of participants either suffered cardiac death or experienced recurrent STEMI (Strik et al., 2003); depression was not associated with increased mortality or recurrent cardiac events.

This study also reported that for those who were anxious healthcare consumption for cardiac events was increased during the mean 3.4 year follow-up. Stepwise logistic regression modelling showed anxiety to be an independent predictor of increased healthcare use (OR 2.00, 95% CI 1.24 to 3.22; p=0.005). In contrast depression did not predict increased health care consumption (p=0.65) (Strik et al., 2003). This study has previously been discussed in sections 2.6.1 and 2.7.2.3.

Moser and Dracup (1996) investigated raised anxiety levels following STEMI in 86 patients and found that those with raised anxiety levels were more prone to in-hospital cardiac complications. The Brief Symptom Inventory (Derogatis and Melisaratos, 1983) was used to measure anxiety levels and showed that 70.0% of patients had abnormal anxiety scores, 26.0% of whom had scores in the psychiatric range. Those with high rather than low state anxiety (30.0%) experienced a 4.9 times higher frequency of complications including ventricular fibrillation, ischaemia and re-infarction. Patients with higher anxiety also experienced increased levels of chest pain (on a 0 to 10 scale) during STEMI compared to those with low anxiety [7.9 (SD 2.4) vs 6.2 (SD 2.6)] (Moser and Dracup, 1996). The cohort in this study was relatively small and therefore the findings should be viewed with caution.

In a further small study involving 49 MI patients, Cherrington et al (2004) demonstrated that almost half (44.9%) of the cohort experienced at least one in-hospital cardiac complication. Complications included cardiac arrhythmias, heart failure, ischaemia, re-infarction, cardiac arrest and cardiac death. Interestingly logistic regression analysis determined that patients with more negative illness representation, as measured by the illness perception questionnaire (IPQ) (Weinman et al., 1996), were more likely to experience complications. The study
did not demonstrate anxiety to be predictive of complications, although this may have been due to the small size of the cohort.

In contrast Moser et al (Moser et al., 2007) found, in a much larger study (536), that patients with higher levels of anxiety experienced significantly more in-hospital complications. Additionally the authors reported that a combination of raised anxiety and reduced perceived control were predictive of an increased risk of complications. The Control Attitudes Scale (CAS) (Moser and Dracup, 2004, Moser and Dracup, 1995) was used to measure both perceived control and lack of control related to cardiac disease.

The reviewed studies appear to demonstrate that there is an association between anxiety and in-hospital complications, although there is also a suggestion that other factors such as perceived control and illness representations also play a part. The reported association between raised anxiety levels and recurrent cardiac complications may also go some way to explain the increased resource utilisation for STEMI patients reported by Strik et al (2003). However, it is also possible that raised anxiety may also lead to hypervigilance and healthcare seeking without cardiac complications.

2.6.2.3 Anxiety a protective effect in CHD

Patients have been shown to experience shock and anxiety at having suffered a heart attack. Wiles (1998) reported such a finding in a qualitative study involving 25 first time MI patients treated with thrombolysis (lysis). In contrast with the majority of literature that reports the deleterious effects of raised anxiety following MI, it has been suggested by several researchers that a strong emotional reaction (such as anxiety) offers a protective affect to CHD patients (Carinci et al., 1997, Herrmann et al., 2000).

In the Herrmann et al (2000) study, the cohort (5017) was heterogeneous including hospitalised patients and outpatients, and those with confirmed MI, cardiac ischaemia, cardiac arrhythmias or hypertension; all had been referred for an exercise ECG. Anxiety and depression scores, assessed through the Hospital Anxiety and Depression Scale (HADS) (Zigmond and Snaith, 1983), were used to predict mortality. Raised HADS anxiety scores predicted improved survival with lower mortality. Conversely, depression was shown to predict increased mortality. The mixed study cohort including unwell and stable patients as well as those with cardiac ischaemia and arrhythmias limits the generalisability of findings to the more specific STEMI population.
However, Carinci et al (1997) investigated 2449 STEMI patients as part of a sub-study of the RCT GISSI-2 trial. The GISSI-2 trial investigated a variety of thrombolytic therapies for the treatment of STEMI (Zuanetti et al., 1996, Maggioni et al., 1993). The self-report measure the Cognitive Behaviour Assessment Hospital Form (Bettinardi and Zotti, 1995), containing 16 psychological dimensions, was used to assess psychological health. The researchers found that individuals reporting increased anxiety and neuroticism (a personality trait) had a reduced mortality rate at 6 months post-STEMI.

The authors suggested that anxiety may have influenced patients to accept social support and adhere to behavioural risk factor modifications, although evidence through the study was not provided to support this theory (Carinci et al., 1997). The infrequently used self-report measure (in CHD cohorts) and the highly selective RCT study cohort used in this study may have influenced the study findings. Other researchers in more recent studies have not reflected Carinci et al (1997) study findings.

2.6.2.4 Determining anxiety in STEMI patients

The assessment and management of anxiety by healthcare professionals during an acute IHD event have been shown to be inconsistent and generally lacking (Frazier et al., 2002). Grace et al (2004) reported that over half (62.0%) of ACS patients are not screened for anxiety related symptoms during their hospital admission.

Failure to screen patients in this situation prevents the opportunity to initiate relevant treatments. Patients who remain untreated or experience persistent raised anxiety levels may experience a negative response to pain (Asmundson, 1999). Additionally they may suffer greater intensity of cardiac symptoms (Aitkens et al., 2001) and heightened heart beat awareness (Eifert et al., 2000); this is particularly prevalent in women (Keogh et al., 2004). When extreme acute anxiety remains untreated, individuals are at risk of suicidal behaviour (Reich et al., 1993, Brown and Barlow, 1992) or are susceptible to develop major depression (Reich et al., 1993). Both these complications are reported to increase mortality in CHD patients (Reich et al., 1993, Brown and Barlow, 1992).

Treatment of anxiety is discussed in section 2.8. Additionally, the anxiety section of the literature review is summarised in combination with the other psychological health sections in section 2.9.
2.7 Depression

Depression is associated with the development and progression of CHD in the general population, as well as increased mortality rates due to cardiac causes (Rabins et al., 1985, Wulsin and Singal, 2003). Additionally, the presence of depression after STEMI is associated with significant deterioration in quality of life, increased cardiac symptoms, vital exhaustion as well as higher mortality rates (Dickens et al., 2006, Barth et al., 2004, van Melle et al., 2004, Parashar et al., 2006, McGowan et al., 2004).

2.7.1 Prevalence of depression in CHD patients

Researchers report that between 31.0% and 35.0% of STEMI patients experience depression within the first month of their acute event (Creed, 1999, Grace et al., 2004, Ladwig et al., 1994). Currently there are no published reports regarding the prevalence of depression in the PPCI population.

Grace et al (2005) studied depression in 906 patients at the time of an ACS event (also described in section 2.6.1, 2.11.5 and 2.12.3.2). Depression was measured 2 to 5 days post ACS event (unstable angina or MI), using the Beck Depressive Inventory (BDI) (Beck, 1978). Elevated depressive symptomology (BDI scores ≥10) was seen in 31.3% of participants at baseline and scores were shown to decline over time: BDI scores ≥10 at 6 months in 25.2% and at 12 month 21.7% of participants (Grace et al., 2005a).

In one of the most quoted studies to investigate the relationship between depression and STEMI (n=222), 15.8% of patients were found to have major in-hospital depression (Frasure-Smith et al., 1993). Major depression was assessed using a modified version of the National Institute of Mental Health Diagnostic Interview Schedule (DIS) (Robins et al., 1981). Additionally, participants completed the self-report BDI and 30.6% of patients reported mild to moderate (BDI scores ≥10) symptoms of depression at baseline (Frasure-Smith et al., 1993). Interviews and the BDI were completed within one week (range 5 to 15 days) of MI. At the time of infarction 50.0% (n=108) of patients received thrombolysis and 30.0% (n=76) received either PCI or CABG during their admission; psychological status was not categorized according to treatment regimes. This study is also discussed further in section 2.7.2.1.

In a further study, which only included men (552), 37% experienced severe to moderate levels of depression shortly following STEMI (Ladwig et al., 1994). Ladwig
et al (1994) used the self-report measure Montgomery and Asberg Depression Rating Scale (MADRAS) (Montgomery and Asberg, 1979) within 17 to 21 days of STEMI to assess the participants levels of depression. This study was conducted prior to the introduction of revascularisation therapies (such as lysis and PPCI). It is therefore likely that patients would have endured more cardiac morbidities, symptoms and physical limitations following their index MI due to the lack of revascularisation. The physical deleterious effects of the MI may have influenced the participants’ psychological health, leading to increased levels of depression in this study. Furthermore, the study did not include women limiting the ability to generalise the results to STEMI populations as a whole.

One week prior to STEMI a slightly higher prevalence of depression was reported (23.8%); this was much higher in women under 60 years of age living in an inner city area (46.0%) (Dickens et al., 2004). Dickens et al (2004) reviewed 587 first STEMI patients using HADS (mean 3.6, SD 2.1 days post STEMI) and asked patients to report their mood the week prior to their infarction. Timing of depression is discussed further in section 2.7.2.2.

### 2.7.1.1 Prevalence of depression according to gender

Depression in the general population is reportedly more prevalent in women than men, occurring between half and three time more than in men (Kessler, 2003).

There are mixed reports regarding whether different rates of depression exists between the genders in CHD patients. Welin et al (2000) report that significantly more women compared to men experience depression (55.6% and 34.4%, p=0.013) following STEMI. Todaro et al (2005) also found higher levels of past and present depression in women compared to men in a study of CHD patients who attended cardiac rehabilitation. The data collection method incorporated semi-structured psychiatric interviews to explore depression levels (Todaro et al., 2005).

In elective PCI patients 6 months after receiving stent implantation women were not found to experience higher levels of depression than men. Ladwig et al (2000) used the HADS to measure anxiety and depression and showed that there was no difference in depression between the genders. However, women did have a significant increase in sleep disorders and a non-significant increase in anxiety. There was also a trend for women to have severe negative health perception which was associated with high angina levels, both before and after coronary stent implantation (Ladwig et al., 2000).
2.7.2 The consequences of depression in CHD patients

Depression has been associated with increased mortality rates for CHD groups and in particular the available data suggest that mortality and depression are closely associated following STEMI (Frasure-Smith et al., 1993, van Melle et al., 2004).

2.7.2.1 Depression, CHD and mortality

Depression is an independent risk factor for mortality when other known factors including age, gender, severity of STEMI and smoking have all been controlled for (Lespérance et al., 2002, Frasure-Smith et al., 1993, Dickens et al., 2008). Individuals with CHD or following an MI, are twice as likely to die if they experience depressive symptomology than if they do not (Barth et al., 2004, van Melle et al., 2004).

Two meta-analyses reported the deleterious effects of depression on outcome in CHD and MI patients respectively (Barth et al., 2004, van Melle et al., 2004). In the van Melle et al (2004) study the authors investigated the association between MI, depression and prognosis. Twenty-two publications were included, combining 16 study cohorts with a total of 6367 MI patients; average study follow-up was 13.7 months. The authors addressed publication bias, which is a known limitation of meta-analysis, by including non-English and unpublished studies in their literature search. Patients with depression were more likely to die (all cause mortality) following MI than those without depression (odds ratio [OR], fixed 2.38; 95% confidence interval [CI], 1.76–3.22). Furthermore, depressed patients were also more likely to suffer cardiac mortality (OR fixed, 2.59; 95% CI, 1.77–3.77) and were at increased risk of new cardiac events (OR random, 1.95; 95% CI, 1.33–2.85) (van Melle et al., 2004).

Similar findings were seen in Barth et al’s (2004) meta-analysis of 20 studies investigating the impact of depressive symptoms or depressive disorders on mortality (cardiac or all cause) in CHD patients. The studies included in the analysis were conducted between 1988 and 2003 and involved MI, PCI, CABG and ACS patient cohorts. The researchers found that depressive symptoms and clinical depression had an adverse outcome in terms of mortality in CHD patients. Individuals with CHD and depressive symptoms or clinical depression were more likely to die within 2 years of assessment, than those without either depressive symptoms or clinical depression (depressive symptoms OR 2.24; CI 1.37–3.60 and clinical depression OR, 2.61; 1.53–4.47) (Barth et al., 2004).
A limitation of both van Melle et al (2004) and Barth et al (2004) studies was that the publications included by the authors did not indicate whether the participants were already suffering from depression at the time of their MI. Lifetime diagnosis of depression has been reportedly found in up to 25.0% of those with CHD suffering depression (Lespérance et al., 1996). It is possible that CHD patients who experience depression throughout their lives are at increased risk of mortality and this may be due to the biological changes associated with psychological distress (see section 2.4), although this has not directly been contributed to depression.

Frasure-Smith et al (1993) study (previously discussed in section 2.7.1) was included in the meta-analyses of both van Melle et al. (2004) and Barth et al (2004). The authors reported major depressive disorder (MDD) to be an independent risk factor for increased mortality up to 6 months post STEMI. At 6 months following infarction, deaths had occurred in 17.0% of depressed and only 3.0% of non-depressed patients (Frasure-Smith et al., 1993). Following correction for other cardiovascular risk factors, the relative risk was 3.5 times greater for the depressed than non-depressed. The impact was at least equivalent to Left Ventricular (LV) dysfunction and previous STEMI (Frasure-Smith et al., 1993). Mortality rates associated with depression did not significantly change over time; similar mortality rates existed for those who were depressed at 12 and 18 months as those identified with symptoms of depression immediately after STEMI (Frasure-Smith et al., 1995a, Frasure-Smith et al., 1993).

The Frasure-Smith et al (1993) study investigated the mortality rates of STEMI patients based on depression scores five to 15 days following MI. It is possible that these individuals were already depressed at the time of their STEMI or may have had a long history of depression and this may have influenced the study findings. However, assessing depression prior to a new ACS event is fraught with difficulties. The unpredictability of the ACS event leads to challenges in obtaining pre-MI depression information, often the only sources of information being recall on behalf of the patient or through primary care records.

An association between increased mortality rates and depression following STEMI has also been reported by Lespérance et al (2002). The authors measured depression using the BDI in 896 STEMI patients during index hospital admission. Patients who were either moderately (BDI scores 10-18) or severely (BDI scores ≥19) depressed during admission were more likely to experience cardiac death or non-fatal STEMI during 12 month follow-up (26.1% vs 34.2% respectively). At five year follow-up moderate and severe depression were also associated with combined
cardiac mortality and re-infarction. In this study a small proportion (13.2%) of patients received PCI following discharge; no relationship was seen between level of depression, cardiac events and being treated with PCI (Lespérance et al., 2002).

2.7.2.2 The timing of depression

The timing of depression post Acute Coronary Syndrome (ACS) event and MI has been considered by researchers. Grace et al (2004 and 2005) found that both depression and anxiety declined from baseline to six and 12 months following an ACS event. This study has previously been discussed in section 2.6.1 and 2.7.1. It is unclear whether progression or resolution of depression influences outcome during recovery, as mixed study findings have been reported (Grace et al., 2005c, de Jonge et al., 2006, Dickens et al., 2007, Dickens et al., 2008, Dickens et al., 2005, Lane et al., 2002).

In the Grace et al (2005c) study involving 750 unstable angina and MI patients, almost a quarter (23.2%) of participants self-reported through interview, having experienced depressed mood for ≥2 weeks prior to MI (categorised as ‘depressive history’). A further 31.3% self-reported symptoms of depression occurring during admission (two to five days post MI) on the BDI (score ≥10 = mild to severe symptoms); 14.0% reported experiencing symptoms of depression at both time points (≥2 weeks prior to MI and during admission). At five years 15.3% deaths were recorded and symptoms of depression (BDI score ≥ 10) during admission were predictive of mortality after controlling for other factors (including sociodemographic factors, MI diagnosis, smoking, Killip class and medical history). The Hazard ratios for BDI scores (<10 compared to >10) for the new onset depressive symptom group (during admission) ranged from 1.90 at two years and to 1.53 at five year follow-up (Grace et al., 2005c).

Similar findings were reported by a Dutch study involving 468 post MI patients (de Jonge et al., 2006). The researchers assessed depression at baseline (three months) and 12 months using the Composite International Diagnostic Interview (CIDI) (Smeets and Dingemans, 1993) schedule. Incident depression (new onset since MI) was predictive of cardiac events (hazard ratio [HR] 1.76; 95% confidence interval [CI] 1.06 to 2.93) at mean 2.5 years post MI when controlling for age, gender, education level, LVEF <40% and revascularization. When controlling for the same factors non-incident depression (pre-MI) was not shown to be predictive of cardiac events.
However, Lane et al (2002) found over multiple time points (baseline, four and 12 months and three years) in a cohort of 288 MI patients that depression measured using the BDI was not associated with cardiac mortality. In a further study Dickens et al (2005) found similar results. The researchers compared the mortality rates of 588 post MI patients, who self-reported depressive symptoms one week prior to MI (96) with those who developed ‘new onset’ depression 12 months following MI (71) and with those who did not develop depression at any point (273) (Dickens et al., 2008).

The Hospital Anxiety and Depression Scale (HADS) was completed three to five days following MI and patients were asked to reflect back to their emotional state the week before their MI. The authors validated this unconventional use of HADS by interviewing 313 patients using the Schedule for Clinical Assessment in Neuropsychiatry (SCAN) (World Health Organisation Division of Mental Health, 1996) to assess whether patients met ICD-10 classification for depression. They determined that total HADS ≥17 cut-off provided the best sensitivity (87.7%) and specificity (84.7%) to diagnose depressive disorder (Dickens et al., 2004).

Dickens et al (2007) used the National Tracing Service to check all the participants’ mortality status at mean 6.7 years post MI and death certificates were used to confirm the cause of death for the 7.3% of participants who died due to cardiac causes and 8.9% who died from non-cardiac causes during follow-up. The authors found no difference in survival at 6.7 years follow-up between new onset depression (at 12 months) and those who were not depressed. Additionally, in this publication the authors report that those depressed prior to MI and remaining so at 12 months had significantly better survival than those who were not depressed.

However, a later publication of results from the same study (by the same authors) determined with the use of Kaplan-Meier survival curves, that survival was significantly worse for those with ‘new onset’ depression (at 12 months) than for those who developed depression prior to MI. Cox regression identified that cardiac death was predicted by greater age, pre MI angina, higher MI Killip Class, receipt of beta blockers at discharge and new onset of depression (HR 2.33, p=0.04) (Dickens et al., 2008).

The authors of this study did not measure depression shortly after MI unlike in other studies. It is possible that the increased mortality rates reported by others were due to early post MI onset depression, which was not reported by Dickens et al (2007 and 2008). A comparison of HADS scores shortly after MI (to compare depressed individuals with those who were not depressed) would have been a
useful comparison in this study. Clearly the mixed results of the presented studies in this section demonstrate the complexity of the timing of depression and their influence on outcome.

2.7.2.3 Absence of relationship between MI, depression and mortality

However, not all studies report an association between depression and increased mortality rates following MI, although these are in the minority (Strik et al., 2003, Mayou et al., 2000).

In the study by Strik et al (2003) (previously discussed in section 2.6.1 and 2.6.2.2) no relationship was seen between cardiac deaths or cardiac events and depressive symptomology at mean 3.4 years following STEMI; 30.1% of individuals required hospitalisation for cardiac events. The researchers assessed depression and anxiety using the SCL-90 at one month following STEMI in 318 patients. In this study 43.1% of patients received invasive treatment (thrombolysis, PCI or CABG) during their admission compared to 55.0% who did not (1.9% unknown); there was no difference in depression rates between these two groups (41.6% vs 51.4%, p=0.085) (Strik et al., 2003). Additionally, a further study of 344 post MI patients by Mayou et al (2000) (also see section 2.12.3.2) also did not find that psychological distress was related to increased mortality rates.

2.7.2.4 Depression, MI and social support

Evidence in the literature suggests that depression following an MI is associated with reduced social support (Lett et al., 2007). In turn, low social support and depression combined (Pfiffner and Hoffmann, 2004, Lett et al., 2007, Berkman et al., 1992), and also lack of a confidant in isolation (Dickens et al., 2004, Kawachi et al., 1996, Pfiffner and Hoffmann, 2004) have been attributed to increased mortality. Additionally, reduced social support leads to diminished health related quality of life (HRQoL), poorer adherence to medication and reduced attendance at cardiac rehabilitation following MI (Molloy et al., 2008). Furthermore, Frasure-Smith et al (2000) reported that high levels of social support at the time of STEMI reduces mortality and improves depression during the first year of recovery.

The participants included in the Lett et al (2007) study were a sub-set of patients participating in the ‘Enhancing Recovery in Coronary Heart Disease’ (ENRICHD) trial (Berkman et al., 2003). The ENRICHD study is discussed in more detail in section 2.8.1. In brief, 2481 post MI patients were recruited to the ENRICHD study with either baseline low perceived social support (LPSS) (60.0%), or with depression
(66.0%), or with both LPSS and depression (35.0%). Participants were randomised to receive either usual post MI care or Cognitive Behavioural Therapy (CBT) (Berkman et al., 2003). In the Lett et al (2007) sub-study the researchers set out to investigate what types of social support were of greatest importance in reducing mortality rates and re-infarction.

The authors found that there was no direct relationship between reduced social support and outcome (mortality and re-infarction rates) at median 2.1 year follow-up. However, they did find that there was a relationship between low social support, depression and poor outcome. When depression was not present, improved outcome was indicated by high levels of perceived functional social support measured using the ENRICHD Social Support Instrument (ESSI) (ENRICHD Investigators, 2000) (p=0.036) and the Perceived Social Support Scale (PSSS) (Blumenthal et al., 1987), (p=0.004).

In this study a control group (i.e. patients without depression and/or low social support) was not included. This limited the degree to which the prognostic significance of social support could be determined and the extent that the study findings could be generalised to further MI cohorts. Additionally, only 28.4% of the original ENRICHD study cohort completed all the study questionnaires. Those who completed all the measures were a more affected group again limiting the generalisation of results; they were more likely to be depressed, had lower ESSI scores, had lower income and were more likely to have experienced previous cardiovascular events (Lett et al., 2007). The ENRICHD study is discussed in more detail in section 2.8.1.

Pfiffner and Hoffmann (2004) similarly found that men attending cardiac rehabilitation following their first MI were more likely to die when they reported reduced social support in the form of lack of a partner, coupled with depression and anxiety. This was a study conducted in Germany and involved 222 men for seven years post STEMI. In contrast to Lett et al (2007) study the participants in this study had good well-being with only 10.0% reporting poor well-being, raised anxiety and/or depression, or poor self-value.

The limitations of this study included the study sample, which only included men who had experienced their first MI. Moreover it is possible that the study sample was selected from a cohort with relatively good social support, as the sample was selected purely from those who attended cardiac rehabilitation. In a study conducted by Molloy et al (2008) ACS patients with more social support (in the
form of a large social network) were three and a half times more likely to attend cardiac rehabilitation than those with reduced support.

Not all investigators have found a relationship between social support, depression and increased mortality. Dickens et al (2004) reported higher mortality rates for STEMI patients with lack of a close confidant when compared to those who did have a confidant (8.2% vs 4.5%, p = 0.17). In contrast to the findings of other studies (Lett et al., 2007, Pfiffner and Hoffmann, 2004) the authors determined that cardiac death was not associated with depression.

Clearly social support plays an important part in the recovery of patients following an MI. Social support was demonstrated as beneficial in the Montreal Heart Attack Readjustment Trial (M-HART); when MI patients reported close friendships a greater improvement was seen in psychological distress compared to those without close friends (Cossette et al., 2001).

Overall, in the presented studies, lack of social support leads to increased mortality and poor outcome particularly when coupled with depression (Lett et al., 2007, Pfiffner and Hoffmann, 2004). When patients do have good support networks or a close confidant they appear to adopt healthy behaviours and experience improvements in depressive symptoms and improved HRQoL (Molloy et al., 2008, Frasure-Smith et al., 2000, Cossette et al., 2001).

2.7.2.5 Depression, MI, angina and vital exhaustion

Further consequences related to depression following MI have also been reported. In particular depression has been associated with angina symptoms, vital exhaustion and reduced HRQoL (Parashar et al., 2006, Dickens et al., 2006, McGowan et al., 2004). Psychological health and HRQoL are discussed in section 2.10.1.

Numerous studies have reported an association between cardiac ischaemic symptoms and depression in post MI and CHD patients (Frasure-Smith et al., 1995a, Ladwig et al., 1994, Mayou et al., 2000, Lane et al., 2000a).

In the study conducted by Ladwig et al (1994) 377 post-STEMI patients with moderate or severe depression were two and three times (respectively) more likely to experience angina during 6 month follow-up. Mayou et al (2000b) also reported that patients who were psychologically distressed soon after STEMI were more
likely to experience chest pain. Most chest pain reported was nonspecific in accordance with the Rose criteria (Rose et al., 1977).

Additionally, in a study of 2096 post MI patients depression was associated with angina symptoms (Parashar et al., 2006). This study was part of an observational study the `Prospective Registry Evaluating outcomes after Myocardial Infarction: Events and Recovery’ (PREMIER) study which investigated the processes of care and outcome of a cohort of MI patients (Spertus et al., 2006).

Health status was measured at baseline (during admission), one and six months post MI using the Seattle Angina Questionnaire (SAQ) (Spertus et al., 1995) and symptoms of depression using the Primary Care Evaluation of Mental Disorders Brief Patient Health Questionnaire (PHQ) (Spitzer et al., 1994).

Participants with transient (at baseline), new (at one month), and persistent (both baseline and one month) symptoms of depression experienced significantly more angina at six months than those who were not depressed (see section 2.10.1). In multivariate analysis at six months odds ratios (ORs) for angina in those with transient, new and persistent depression were 1.62, 2.64 and 2.73 respectively (all p<0.01) (Parashar et al., 2006).

An association has also been reported between depression and vital exhaustion for 305 first MI patients (McGowan et al., 2004). McGowan et al (2004) measured depression using the Hospital Anxiety and Depression Scale (HADS) (Zigmond and Snaith, 1983) and Vital exhaustion using the The Maastricht Questionnaire (MQ; vital exhaustion) (Appels et al., 1987) at mean 3.6 days following MI. The MQ (vital exhaustion) is a 21 item self-report measure assessing pre-MI symptoms including fatigue, depression, poor concentration and insomnia. Vital exhaustion and depression were highly correlated even when controlling for age, gender and comorbidity (r=0.59, p<0.01). Likewise the authors found a high level of association between the MQ fatigue dimension and HADS depression (r= 0.50, p<0.01) also when controlling for the same factors.

The depression section is summarised in section 2.9 along with psychological health and anxiety sections.

2.8 Treatment of anxiety and depression

The majority of treatments for anxiety and depressive disorders are similar due to the overlap of symptoms and common co-occurrence of the two disorders. The use
of pharmacology such as selective serotonin reuptake inhibitors (SSRIs) and non-
pharmacology treatments such as Cognitive Behavioural Therapy (CBT) are
common in patients with anxiety and depression (Taylor et al., 2005, Berkman et
al., 2003). However, STEMI patients are reported to be under treated with only
5.0% receiving anti-depressant medication, and 3.0% receiving individualised
psychological help or counselling (Grace et al., 2005a, Hughes, 2011).

2.8.1 Non-pharmacology treatments

A number of non-pharmacological methods for the treatment of anxiety and
depression in CHD patients have been studied with mixed results. Treatments such
as group counselling, Cognitive Behavioural Therapy (CBT) and a home
psychological nursing intervention have all been evaluated (ENRICHD Investigators,
2001a, Frasure-Smith et al., 1997, Jones and West, 1996). Generally CBT is a
weekly or bi-monthly programme involving either individual or group therapy. It is
a practical programme which encourages individuals to alter unhelpful, self-critical
thought processes and adopt positive patterns of behaviour.

In a Cochrane systematic review no positive effects were found for non-
pharmacological psychological interventions on mortality rates for STEMI and CHD
cohorts (Rees et al., 2004). Small significant reductions for anxiety and depression
were reported and insufficient data was available to combine results related to
quality of life. The review combined the results of 36 RCTs, which used a wide
variety of psychological treatments some of which involved stress management
programmes (required to include CBT for inclusion in the review). Additionally,
interventions often included relaxation techniques and cognitive techniques such as
self-instruction training (Meichenbaum, 1985), cognitive challenge or coping
strategies to be used at times of stress. Sub-analysis for studies using similar
psychological interventions and study methodologies were conducted. Ten studies
(3425 patients) incorporated a stress management programme and compared this
to standard care. Stress management programmes did not show any benefit in
relation to mortality (OR 0.88, 95% CI 0.67 to 1.15) (Rees et al., 2004).

A further study investigating a home based nursing education and psychological
support programme did not show any positive benefits in terms of cardiac related
mortality for distressed STEMI patients (Frasure-Smith et al., 1997). This RCT
involved 1376 STEMI patients receiving either an intervention programme (n=692)
or usual care (n=684). The interventional group received monthly psychological
health assessment (by telephone) and for those identified with psychological
distress, a nursing intervention generally including emotional support, education, practical advice, and referral to other healthcare services was implemented. Distress, depression, anxiety and perceived social support were all assessed using multiple self-report measures. The interventional programme did not reduce cardiac mortality and additionally higher levels of mortality (cardiac and all cause) were seen in women receiving the intervention.

One of the largest randomised studies (n=2481) to evaluate psychological intervention in STEMI patients was the ENRICHD study (ENRICHD Investigators, 2000). The ENRICHD trial compared the use of CBT (n=1238) and normal care (n=1243) in STEMI patients with either depression (39.0%) or low perceived social support (LPSS) (26.0%), or both (34.0%). Current depressive episodes were diagnosed using the Depression Interview and Structured Hamilton (DISH) (Freelander et al., 2002); a semi-structured interview developed specifically for ENRICHD. The DISH used the Hamilton Rating Scale for Depression (HRSD) (Williams, 1988) a 17-item depression rating scale. Patients were also asked to complete the BDI to self-report the severity of depressive symptoms. The ENRICHD Social Support Instrument (ESSI) (ENRICHD Investigators, 2000) and the Perceived Social Support Scale (PSSS) were also used to assess LPSS.

At 29 months follow-up survival curves showed no difference in recurrent STEMI or death between the two groups (log-rank p=0.94). However, after 6 months, CBT treatment was found of benefit for psychosocial outcome in both the depressed and the LPSS groups compared to usual care. The mean (SD) change in HRSD scores in the depressed group receiving CBT was -10.1 (7.8) compared to -8.4 (7.7) receiving usual care (p<0.001). The mean (SD) change in ESSI score for the LPSS group receiving CBT was 5.1 (5.9) compared to 3.4 (6.0) receiving usual care (p<0.001). CBT also had a positive effect at 6 months on mean BDI scores of 9.1 compared to 12.2 in the usual care group (p<0.001; the effect was not maintained at 30 months (p=0.61) (ENRICHD Investigators, 2003).

The use of antidepressants increased accumulatively over time in both groups and by the end of the study, SSRIs were received by 14.6% of the usual care and 21.0% of the intervention group. The relatively high usage of SSRIs in the two groups was acknowledged as influencing the methodological design of the study and masking any clinical benefits of CBT in the intervention group (ENRICHD Investigators, 2003, Joynt and O’Connor, 2005).
2.8.2 Pharmacology treatments

The general use of antidepressants in CHD patients is controversial. Mixed results from studies relate to the type of pharmacological treatment involved. Study results have shown that the use of tricyclic antidepressants increase the occurrence of cardiac events (Cohen et al., 2000, Glassman et al., 1993). In the case of selective serotonin reuptake inhibitors (SSRIs) the risk of death is reduced in CHD patients (Cohen et al., 2000, Taylor et al., 2005, Glassman et al., 2002).

Tricyclic antidepressants have been shown to increase the risk of MI in otherwise healthy individuals (Cohen et al., 2000). The authors compared depressed workers treated with either tricyclics or SSRI medication with workers who were not treated with antidepressants. After three years they found that those treated with tricyclic agents had relative risk (RR) of STEMI in the region of 2.2 (95% CI 1.2 to 3.8) and for those prescribed SSRIs 0.8 (95% CI 0.2 to 3.5), compared to individuals not taking antidepressant medication. This study highlighted that depressed individuals taking SSRIs did not appear to be at increased risk of STEMI.

In an additional study the Sertraline Anti Depressant Heart Attack Randomized Trial (SADHART), although not statistically significant, showed a potential benefit of receiving SSRIs for depressed ACS patients (Shapiro et al., 1999). A trend toward reduction in morbidity and mortality among SSRI treated patients was seen. This study investigated the use of sertraline (an SSRI medication) for the treatment of STEMI patients. The ENRICHD study offered further reassurance for the safety and efficacy of SSRIs in the treatment of depression for STEMI patients. In the ENRICHD study the risk of death and nonfatal recurrent STEMI was lower for patients taking SSRIs with adjusted hazard ratio of 0.57 (95% CI 0.38-0.85) than those who were not taking the medication (ENRICHD Investigators, 2001b).

The prognostic benefit of SSRIs may be due to an antiplatelet effect that the drug appears to offer (Bruce and Musselman, 2005). In a review of 15 recent studies investigating the effect of SSRIs on platelet activity, the majority of studies showed SSRIs to have a protective role in ischaemic heart disease (IHD). This is particularly relevant for patients who have increased platelet activation such as those who smoke.

The treatment of anxiety and depression section is summarised in section 2.9 along with the other psychological health sections.
2.9 Summary of psychological health, anxiety, depression and treatment sections

The presented studies go some way in identifying how common anxiety and depression are in the STEMI population. Anxiety rates as high as 45.0% in a mixed cohort of patients within 48 hours of STEMI (Frazier et al., 2002) and 60.0% for men at one month following their first MI are reported (Strik et al., 2003). Major depression has been found in 16.0% of a mixed cohort of STEMI patients during initial hospitalisation (Frasure-Smith et al., 1993). Additionally, moderate to severe depression has been identified in 37.0% of men within 17 to 21 days of STEMI (Ladwig et al., 1994). Anxiety and depression have also been reported to occur more frequently in women than men both in the general population and in post MI patients (Frasure-Smith et al., 1999, Lane et al., 2002, Kessler, 2003, Regier et al., 1990, Todaro et al., 2005).

Serious consequences, including increased mortality and cardiac events are found in those with anxiety and depression following STEMI (van Melle et al., 2004, Roest et al., 2010, Moser and Dracup, 1996). Depression is most strongly associated with mortality (van Melle et al., 2004), whereas anxiety shares a stronger relationship with cardiac ischaemic events (Moser and Dracup, 1996). Increased mortality rates of 17.0% at 6 months and 26.0% to 34.2% at 12 months are reported in depressed individuals following STEMI (Frasure-Smith et al., 1993, Lespérance et al., 2002).

It has been suggested by researchers that new onset depression influences mortality and/or cardiac events more than pre-STEMI depression (Dickens et al., 2008, de Jonge et al., 2006, Grace et al., 2005c). However, other authors present conflicting evidence (Lane et al., 2002). Currently the literature is unclear and further investigation of the potential association between timing of depression and outcome is required.

Low social support also appears to negatively influence outcome of MI patients particularly when coupled with depression (Pfiffner and Hoffmann, 2004, Lett et al., 2007, Berkman et al., 1992). Additionally good social support networks, or a close confidant and/or close friendships appear to aid adherence to medication, improves attendance at cardiac rehabilitation and improves symptoms of depression during recovery (Cossette et al., 2001, Molloy et al., 2008).

Despite the known prevalence and serious consequences of anxiety and depression for ACS patients, 62.0% did not receive psychological assessment during acute
admission for chest pain in one study (Grace et al., 2004). Additionally, under-treatment of STEMI patients is also reported with only 5.0% receiving antidepressants and 3.0% receiving non-pharmacological treatments (Grace et al., 2005a, Hughes, 2011). For those who do receive treatment the reported benefits appear to be mixed (Taylor et al., 2005, Berkman et al., 2003). The use of pharmacology with SSRI medication has been shown to reduce mortality and re-infarction rates (Taylor et al., 2005). However, non-pharmacology intervention with the use of CBT does not appear to improve mortality rates for STEMI and CHD cohorts (Rees et al., 2004, ENRICHD Investigators, 2001a).

Some of the symptoms of anxiety and depression are similar to those associated with IHD and can make establishing a diagnosis challenging for healthcare professionals (Dammen et al., 2004, American Psychiatric Association, 2000). This is particularly pertinent when cardiac events and mortality are known to be increased in STEMI patients who experience psychological sequelae. The occurrence of symptoms related to either cardiac ischaemia or psychological sequelae may in some circumstances lead to re-presentation to acute healthcare services, although the degree to which this occurs is currently unknown.

### 2.10 Quality of life

Previous sections of this literature review describe the serious clinical outcomes (mortality and cardiac events) that are associated with MI and reduced psychological health following STEMI. Additionally, psychological distress, angina and reduced social support have all been implicated in decreased health related quality of life (HRQoL) following MI (McGowan et al., 2011, Dickens et al., 2006, Hirani et al., 2006, Zhang et al., 2004).

The definition of quality of life (QoL) varies from author to author, but generally refers to how well individuals are satisfied or dissatisfied with certain domains of their life including social, economic, pleasure, happiness and fulfilment (Costanza et al., 2007, Schuessler and Fisher, 1985). The term QoL is also used in health care and is often interchangeable with the term health related quality of life (HRQoL), which has a more focused meaning in healthcare terms. Instruments that measure HRQoL include both generic measures such as the Short Form 36 (SF 36) (Ware, 1993) and also disease specific measures including the Seattle Angina Questionnaire (SAQ) (Spertus et al., 1995).
2.10.1 Psychological health and reduced quality of life

The majority of studies demonstrating an association between reduced HRQoL and diminished psychological health in post-MI patients relates to increased symptoms of depression.

Dickens et al (2006) found that symptoms of depression predicted reduced HRQoL following STEMI in a prospective cohort of 260 STEMI patients. Thrombolysis was received by 73.2%, ‘surgical intervention’ by 8.6% and for the remaining 18.2% of patients details of treatment were not provided. Self-report measures HADS (Zigmond and Snaith, 1983) and SF-36 (Ware, 1993) were used to measure psychological health and HRQoL respectively, one week prior to STEMI (classed as baseline), and at 6 and 12 months (see section 2.7.2.2 for further details of timings).

The authors reported that only depression at 6 months rather than baseline contributed to reduced HRQoL at 12 months (Dickens et al., 2006). They also acknowledged in another publication (Dickens et al., 2004) that symptoms of depression were higher immediately post-infarction than one week prior to STEMI and this may explain the lack of significance related to baseline depression predicting reduced HRQoL at 12 months in this study. Nevertheless, in the 2006 study it is clear that depression did contribute to reduced HRQoL following STEMI.

Further evidence of an association between depression and reduced HRQoL following MI was reported in an observational study involving 1873 MI patients (Parashar et al., 2006). This study was part of the ‘Prospective Registry Evaluating outcomes after Myocardial Infarction: Events and Recovery’ (PREMIER) study (Spertus et al., 2006), which investigated the processes of care and outcome of post MI patients.

Symptoms of depression were assessed during admission (baseline) and at one month following MI using the Primary Care Evaluation of Mental Disorders Brief Patient Health Questionnaire (PHQ) (Spitzer et al., 1994). At 6 months HRQoL was measured using the Seattle Angina Questionnaire (Spertus et al., 1995), additionally mortality and hospitalisation were also recorded. The study findings at 6 months showed that patients demonstrating persistent depression (at baseline and one month) reported worse HRQoL (mean SAQ score 15.8 points lower) than participants without depression at any time point. The authors reported that patients with depression (at baseline and one month) also experienced more
symptoms of angina and increased physical limitation. See section 2.7.2.5 for more details of this study.

Symptoms of depression were only assessed during admission and at one month, it is possible that additional measurement at a later time point may have explained further the reduced HRQoL at 6 months. The type of revascularisation treatment (if any) received by participants was not described, although due to the timing of recruitment (2003 to 2004) it is likely that the majority received lysis.

One study that specifically investigated HRQoL and psychological health in PPCI patients was the main study related to the current study (McGowan et al., 2011). The purpose of the main study was to compare the HRQoL of STEMI patients receiving thrombolysis (lysis) (183) or PPCI (202) (McGowan et al., 2011). At baseline and 6 months HRQoL was measured using the self-report measure Medical Outcomes Study Short Form 36 (SF-36) (Ware, 1993) and additionally psychological health was assessed using the HADS (Zigmond and Snaith, 1983).

The initial analysis showed that at baseline significantly higher mean levels of psychological distress (total HADS) were seen in the PPCI group (mean 13.2, SD 7.9) compared to the lysis group (11.4, SD 8.9, p=0.035). Hierarchical multiple regression analysis with four blocks of independent variables (demographic, comorbidity, clinical and psychological factors) was conducted to identify variables at baseline that may have influenced SF-36 Physical Component Score at 6 months. Cholesterol levels (p=0.031) and depression (p<0.001) contributed to the final model. Symptoms of anxiety and depression at baseline contributed to decreased physical HRQoL at 6 months for the total study cohort. Treatment type in this study did not predict reduced HRQoL at 6 months (p=0.199). This study is presented in abstract form and further detailed analysis is presented in the final study report (McGowan et al., 2012) (see section 9.6.1.2).

The presented studies highlight that reduced psychological health is related to lower HRQoL following STEMI and that there is a likely relationship between depression and increased angina symptoms. Furthermore, the early results from the main study (related to the current study) suggest that the type of revascularisation treatment (lysis or PPCI) does not influence HRQoL at 6 months following STEMI.

2.10.2 Cardiac ischaemic symptoms and quality of life

One of the reasons attributed to reduced HRQoL following MI is related to angina and cardiac ischaemic symptoms (Hirani et al., 2006, Zhang et al., 2003).
Revascularisation treatment in the form of PPCI has been associated with improved outcome in terms of mortality and morbidity. Additionally, in the DANAMI-2 sub-study HRQoL was higher in the PPCI group compared to the thrombolysis group at one month (Mortensen et al., 2005). The DANAMI-2 sub-study investigated HRQoL in STEMI patients (1351) who were either randomised to receive PPCI (684) or thrombolysis (lysis) (667). The self-report measures the SF-36 (Ware, 1993), the Rose angina questionnaire (Rose et al., 1977) and HADS were used respectively to measure HRQoL, angina symptoms and psychological health. Psychological health in this study was equivalent at one month for the PPCI and lysis groups. The researchers attributed the improved HRQoL at one month to fewer angina symptoms in the PPCI group. However, the authors only present descriptive analysis and multivariate analysis does not appear to have been undertaken. It is therefore not possible to establish whether there was an association between HRQoL, angina and psychological health when other factors were controlled for (Mortensen et al., 2005).

A further disadvantage of this study was that the investigators only undertook assessment of HRQoL and angina symptoms at one month post-STEMI. It is therefore not possible to ascertain whether the higher levels of HRQoL in the PPCI compared to the lysis group were sustained over a longer period of time. The DANAMI-2 sub study findings are contrary to those of our main study (McGowan et al., 2011) (see section 2.10.1), which indicated that at 6 months there was no difference in HRQoL between PPCI and lysis cohorts. The difference in the results for these two studies is likely to be related to the different timing of follow-up (6 month for our study and one month for DANAMI-2). It is possible that the early benefits of PPCI in terms of HRQoL are ameliorated over the course of the subsequent 6 months and with other factors (such as depression and cardiac ischaemic symptoms) playing a more central role in recovery (McGowan et al., 2011, Mortensen et al., 2005).

When cardiac ischaemic symptoms (such as angina) occur in non-STEMI patients a reduction in HRQoL has been reported. In the Stent or Surgery (SoS) trial patients were randomised to receive either PCI (488) or CABG (500) for index ACS event (Zhang et al., 2003). Those treated with PCI who experienced ongoing angina (post-PCI) had reduced HRQoL at 6 and 12 month follow-up (Zhang et al., 2003). In this study, patients’ HRQoL status was evaluated using the Seattle Angina Questionnaire (SAQ) (Spertus et al., 1995).
It is not clear what proportion of the study cohort comprised of elective compared to acute PCI patients and therefore it is difficult to interpret these findings in the context of other acute PCI cohorts such as PPCI patients. Additionally, the Zhang et al (2003) study was conducted almost a decade ago, and during this time there have been substantial advancements in PCI techniques, cardiac stents and pharmacology. This is likely to have reduced the occurrence of post-PCI angina and may have reduced the transferability of the study findings to current patient cohorts.

However, it remains possible that angina plays a role in influencing HRQoL following PCI, although psychological health is also likely to be implicated in this complex relationship particularly following MI. The presence of reduced psychological health (particularly anxiety) following STEMI has been associated with increased cardiac events (Roest et al., 2010); this is likely to indicate the occurrence of additional IHD symptoms such as angina, which in turn may influence HRQoL.

2.10.3 Summary

Reduced health related quality of life (HRQoL) has been attributed to increased symptoms of depression up to 6 months for MI and PPCI patients (McGowan et al., 2011, Parashar et al., 2006). Additionally, new onset depression has also been reported to negatively affect HRQoL at 12 months post MI (Dickens et al., 2006). An association between depression increased IHD symptoms and reduced HRQoL has also been reported up to 12 months following an MI (Parashar et al., 2006).

Additionally treatment with PPCI has been reported to improve HRQoL within one month of treatment in comparison with thrombolysis (lysis) (Mortensen et al., 2005). Mortensen et al (2005) believed that the HRQoL benefits of PPCI over lysis related to the reduction in cardiac symptoms for PPCI patients. However, in a more recent study of lysis and PPCI patients HRQoL at 6 months did not differ based on treatment (i.e. PPCI versus lysis) (McGowan et al., 2011).

2.11 Cardiac ischaemic symptoms

This section of the review describes the most pertinent literature relating to ischaemic heart disease (IHD) symptoms. Typical and atypical IHD symptoms are discussed as are the experiences of patients regarding symptom attribution. The literature relating to the potential causes of non-cardiac chest pain are described, and in particular the relationship between psychological health and IHD symptoms is described.
2.11.1 Typical cardiac ischaemic symptoms

It has been reported that 83.0% of patients experience chest pain at the time of STEMI (Ryan et al., 2007). This is supported by International guidelines that purport chest pain/discomfort as one of the most common (or typical) symptoms associated with STEMI or ACS event (Thygesen et al., 2007, Antman et al., 2004). Other typical symptoms listed in the guidelines include jaw, back, neck or epigastria pain, shortness of breath, weakness, diaphoresis, nausea and light headedness (Antman et al., 2004, Thygesen et al., 2007). See section 1.2.2 for further details of typical and atypical STEMI symptoms.

Ryan et al (2007) determined that STEMI symptoms tend to occur in five main clusters, three of which included chest pain (Table 2-1). This was established through review of the literature. Data from nine studies pertaining to STEMI symptoms, were included in secondary latent class cluster analysis (McCutcheon, 1987) for categorical and continuous variables.

Table 2-1 clusters of symptoms related to STEMI identified by Ryan et al (2007)

<table>
<thead>
<tr>
<th>Cluster number</th>
<th>Symptoms at high probability of occurring</th>
</tr>
</thead>
<tbody>
<tr>
<td>One</td>
<td>a) Chest discomfort, b) shoulder, arm or hand discomfort, c) weakness.</td>
</tr>
<tr>
<td>Two</td>
<td>a) Chest discomfort, b) shoulder, arm or hand discomfort.</td>
</tr>
<tr>
<td>Three</td>
<td>a) Chest discomfort, b) shoulder, arm or hand discomfort, c) nausea or vomiting, d) shortness of breath, e) sweating, f) dizziness or light-headedness, g) weakness, h) fatigue.</td>
</tr>
<tr>
<td>Four</td>
<td>a) Shoulder, arm or hand discomfort, b) abdominal pain, c) indigestion.</td>
</tr>
<tr>
<td>Five</td>
<td>High probability of symptoms: None. Medium probability of symptoms: a) chest discomfort, b) shortness of breath.</td>
</tr>
</tbody>
</table>

Ryan et al (2007)

In this study women (39.0%) were less likely to suffer chest pain than men (61.0%), although women experienced more intense discomfort and pain than men. The Ryan et al (2007) study reported that all of the five clusters of symptoms in their analysis included some symptoms considered to be atypical.

In a further study conducted across 11 hospitals in Sweden and including 1939 ACS patients, Thuresson et al (2005) set-out to establish the most common symptoms (additional to chest pain) and the intensity of pain to affect STEMI (42.0%) and Non-STEMI (58.0%) patients. All those included in the study had experienced either chest pain or discomfort. The authors devised a questionnaire, previously tested in a pilot study (Hartford et al., 1993), to establish the symptoms and intensity of pain experienced by patients. The questionnaire was administered and completed by patients within the first few days of their admission. The most common
additional symptoms reported for STEMI patients were nausea or cold sweat (66.0%). The four most frequently cited symptoms additional to chest pain or discomfort for the cohort, are described in Table 2-2.

Table 2-2 The four most common symptoms for STEMI and Non-STEMI patients

<table>
<thead>
<tr>
<th>Symptom</th>
<th>STEMI (%)</th>
<th>Non-STEMI (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nausea or cold sweat</td>
<td>66.0</td>
<td>42.0</td>
</tr>
<tr>
<td>Tiredness or weakness</td>
<td>54.0</td>
<td>53.0</td>
</tr>
<tr>
<td>Pain in left arm</td>
<td>53.0</td>
<td>51.0</td>
</tr>
<tr>
<td>Feeling of sickness</td>
<td>41.0</td>
<td>38.0</td>
</tr>
</tbody>
</table>

Thuresson et al (2005)

In 50.0% of STEMI patients symptoms started suddenly and reached maximum intensity within only a few minutes of onset; intensity expressed as >5 on a 10-point scale. For the elderly (both ACS and STEMI) pain was transient over a period of an hour and subsequently pain gradually intensified over a number of hours to reach maximum intensity. However, similar to the findings of Ryan et al (2007) women experienced more intense pain than men (Thuresson et al., 2005).

In this study the cohort only included patients who reported experiencing chest pain at the time of their ACS event, leading to the exclusion of those who may have presented with other more diverse or atypical symptoms. Additionally the questionnaire used was not a validated instrument and the authors did not present evidence of reliability for use in the study cohort.

2.11.2 Atypical cardiac ischaemic symptoms

Clinicians and researchers report that atypical symptoms are less likely to be due to cardiac ischaemia than typical symptoms (Dammen et al., 2004, Antman et al., 2004). Atypical cardiac ischemic symptoms have been professed to include arm, shoulder, wrist, upper back or upper abdominal pain without the occurrence of chest pain, fatigue, weakness, palpitations, indigestion, stroke like symptoms, unexplained confusion and a sense of fear (Alpert et al., 2000, DeVon et al., 2011, Canto et al., 2009).

In a clinic based study involving 199 patients who attended for chest pain assessment 83.9% were found not to have CHD (Dammen et al., 2004). Significantly more participants with non-CHD causes of pain experienced atypical symptoms compared to those diagnosed with CHD (80.0% vs 29.0%, p<0.001).
The main purpose of the study was to identify and compare psychiatric morbidities, pain characteristics and attributions between individuals with and without CHD. Cardiology assessment was conducted by a cardiologist including physical examination, clinical history, risk factor assessment and a standard bicycle ergometer test (exercise test) (Nordenfeldt et al., 1985). Patients underwent psychiatric assessment and diagnosis, which was conducted by a psychiatrist according to the structured clinical interview DSM-IV (World Health Organisation Division of Mental Health, 1996).

The authors found that both CHD and non-CHD groups had similar rates of psychiatric disorders (68.8% vs 73.1%, p= 0.618). Somatic symptoms such as musculoskeletal disease (33.0%) and dyspepsia (23.0%) were also reported for the total cohort (Dammen et al., 2004). This appears to suggest that pain may be related to both cardiac and non-cardiac causes concurrently. Table 2-3 details the psychiatric disorders reported for the CHD and non-CHD groups.

<table>
<thead>
<tr>
<th>Chest pain diagnosis</th>
<th>non-CHD n=167 (83.9%)</th>
<th>CHD n=32 (16.1%)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychiatric disorders (%)</td>
<td>122 (73.1)</td>
<td>22 (68.8)</td>
<td>0.681</td>
</tr>
<tr>
<td>Panic Disorder (%)</td>
<td>69 (41.3)</td>
<td>7 (21.9)</td>
<td>0.038*</td>
</tr>
<tr>
<td>Post Traumatic Stress Disorder (%)</td>
<td>0</td>
<td>1 (3.1)</td>
<td>0.161</td>
</tr>
<tr>
<td>Current major depression (%)</td>
<td>12 (7.2)</td>
<td>0</td>
<td>0.221</td>
</tr>
</tbody>
</table>

* Significant, p<0.05

The findings of the Dammen et al (2004) study indicates that atypical IHD symptoms are more common in non-cardiac patients. However, one research group challenges the concept of what constitutes atypical symptoms and the cause of the symptoms (Body et al., 2010). In the Body et al (2010) study the symptoms of 796 patients attending A&E with suspected ACS were evaluated. The authors identified that atypical symptoms such as pain radiating down the arm or shoulder were likely to indicate Myocardial Infarction (MI). Conversely, they found that symptoms considered to be typical such as pain in the left anterior chest or jaw, neck, throat and left arm or left shoulder were of no diagnostic or prognostic value. Additionally, pain similar to previous myocardial ischaemia indicated a lower probability of STEMI.

The potential concurrent cardiac and non-cardiac symptoms and the conflicting evidence relating to what constitutes cardiac ischaemic pain indicates the complexity surrounding confirming a diagnosis when symptoms occur, particularly for patients with a history of MI. When typical or atypical chest pain and ischaemic
symptoms occur in known CHD patients, it is generally assumed that symptoms are due to cardiac ischaemia until proven otherwise. To prevent mis-diagnosis expensive, potentially anxiety provoking and often unnecessary investigations are often undertaken to reach a diagnosis. Early diagnosis and initiation of treatment during recurrent ACS events following STEMI has been shown to save lives (Keeley et al., 2003). Generally the importance of this action is stressed to patients following their initial STEMI should they experience a repeat episode (Boersma et al., 1996). However, symptom attribution is often challenging for patients and may lead to delays in seeking help (Zerwic et al., 2003).

2.11.3 Cardiac ischaemic symptom attribution

Recognition of symptoms can be difficult for both first time MI patients and for those who have previously experienced an infarct (Ruston et al., 1998). Chest pain is perceived by the public to be the most likely symptom related to heart attack (Goff et al., 1998).

The public’s view of what symptoms constitute a heart attack were investigated in the React (Rapid Early Action for Coronary Treatment) study (Goff et al., 1998). In this population based survey of 1294 respondents, 89.7% reported that chest pain was a symptom of heart attack and 56.6% believed it to be the most important symptom (Goff et al., 1998). This was confirmed in a further smaller questionnaire based study involving 414 members of the general public; chest pain again was identified as the most expected symptom of a heart attack (Zerwic, 1998).

Clearly patients’ expectations of what symptoms constitute a heart attack play an important part in their assessment of whether or not to seek help. Additionally patients have been reported to use stereotypical symptoms (such as chest pain) to interpret their symptoms during myocardial infarction (MI) (Ruston et al., 1998, Moser et al., 2006, Zerwic et al., 2003).

Ruston et al (1998) found in a qualitative study of delayed MI presentation that patients identified with the dramatic symptoms portrayed in films. The term ‘the Hollywood’ heart attack has often been used by authors to describe such stereotypical symptoms (Ornato and Hand, 2001, Moser et al., 2006, Lefler, 2004). When STEMI symptoms are not stereotypical, confusion and delay in seeking help through acute services can occur (Ruston et al., 1998, Moser et al., 2006). This was particularly demonstrated in a study conducted by Zerwic et al (2003) in a group of 212 STEMI patients. A structured interview guide the Myocardial Infarction Symptoms Profile (MISP) (Johnson and King, 1995) was used to collect information
related to the patients’ experiences of symptom before and during STEMI. The authors found that 52.4% of patients experienced symptoms that were not at all similar to their expectation of those associated with a heart attack. Delay in seeking help was associated with lack of similarity of symptoms experienced with those expected (Zerwic et al., 2003).

However, the presence or absence of chest pain does not always mean that patients do not recognise that something is seriously wrong even if they do not identify that they are having a heart attack (Schmidt and Borsch, 1990). In these instances some individuals seek help via their general practitioner (GP) for affirmation of symptoms (Alonzo, 1986, Gray et al., 1993, Ruston et al., 1998).

No studies have directly reported the experiences of patients who have suffered post-PPCI IHD symptoms. Such a study may aid practitioners in developing and delivering appropriate symptom attribution information and education programmes for PPCI patients.

2.11.4 Non-cardiac causes of cardiac ischaemic symptoms

Patients with CHD may present to healthcare services with both cardiac ischaemia and non-cardiac chest pain (Dammen et al., 2004, Ros et al., 1997, Mayou et al., 2000, Jain et al., 1997). Clinicians report that non-cardiac causes of IHD symptoms and chest pain include musculoskeletal, gastric and pulmonary diseases and psychological distress (personal communication with Dr Fath-Ordoubadi, Consultant Cardiologist, on 3rd September 2006). However, few studies involving CHD patients have been undertaken to provide detailed reports of the non-cardiac physiological causes of pain; generally these are combined and categorised as ‘non cardiac’. The relationship between psychological health and IHD symptoms has received the most interest by researchers.

Nevertheless, one early study involving a small sample of 45 CHD patients did set out to investigate the prevalence of gastric and psychological causes of IHD like pain (Ros et al., 1997). Two groups of patients were selected: 18 known CHD patients with repeated admissions due to chest pain that was unresponsive to treatment (unresponsive chest pain group) and 27 patients with confirmed angiographic narrowing of their coronary arteries (IHD group). The number of recurrent admission for the unresponsive chest pain group was not stated. Physical examination was performed by a gastroenterologist and psychological health was assessed through psychiatric interview (with the specific aim of identifying panic disorder). The background of the interviewer was not stated by the authors.
The cause of chest pain during 49 month (mean) follow-up for the unresponsive chest pain group was found to be unrelated to IHD symptoms and was equally divided between psychological and gastric problems (Table 2-4). In the IHD group concurrent IHD and non-cardiac symptoms were reported in 33.3% of patients.

Table 2-4 Causes of chest pain during 49 month follow-up for the unresponsive chest pain group and IHD group.

<table>
<thead>
<tr>
<th>Cause of pain</th>
<th>Unresponsive chest pain group (%)</th>
<th>IHD group (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>IHD pain</td>
<td>Nil</td>
<td>18 (66.7)</td>
</tr>
<tr>
<td>Panic disorder</td>
<td>9 (50.0)</td>
<td>5 (18.5)</td>
</tr>
<tr>
<td>Gastroesophageal dysfunction</td>
<td>9 (50.0)</td>
<td>4 (14.8)</td>
</tr>
</tbody>
</table>

Ros et al (1997)

This study is limited due to the small size of the study cohort, the purely descriptive nature and highly selective cohort. Additionally the authors did not consider other non-cardiac causes of pain such as musculoskeletal and pulmonary diseases. However, the study suggests that both psychological and gastric problems may lead to recurrent pain for CHD patients. This supports the findings of the Dammen et al (2004) study (see section 2.11.2) that also confirmed the presence of psychiatric conditions in CHD patients who attended cardiology clinics for assessment of chest pain. Additionally the two studies imply that symptoms related to CHD and psychiatric disorders occur concurrently.

2.11.5 Psychological sequelae and cardiac ischaemic symptoms

The findings of the study by Ros et al (1997) and Dammen et al (2004) indicate that psychological health is an important consideration when assessing CHD patients who present with chest pain or potential ischaemic symptoms.

Concurrent CHD and psychiatric distress have been investigated in a study conducted in an acute setting in India (Srinivasan and Joseph, 2004). This study included 337 patients who attended an accident and emergency (A&E) department with chest pain. The HADS was used to determine levels of anxiety and depression prior to conducting psychiatric interviews for individuals scoring ≥8 HADS (Zigmond and Snaith, 1983).

In this study 9.0% of patients experienced both CHD and psychiatric disease. Participants’ diagnoses were categorised with 53.0% suffering CHD, 8.0% psychiatric diagnosis, 9.0% both CHD and psychiatric diagnosis and 24.0% had neither CHD nor psychiatric diagnosis. It is possible that some depressed and anxious patients were missed in the Srinivasan and Joseph (2004) study due to the
use of HADS. This tool does not record somatic symptoms and previous studies conducted in India report that patients who present with anxiety and depression do so with many somatic symptoms (Neerakal and Srinivasan, 2003, Chaturvedi et al., 1987). The cultural differences in the way that patients present with somatic symptoms also make the results of the study difficult to apply to Western populations.

However, several similar studies have been conducted in Western populations. Grace et al (2004) undertook a study to investigate the occurrence of IHD symptoms and levels of anxiety and depression in 906 Canadian patients at the time of an ACS event. The authors also investigated health utilisation and progression of psychological health for 12 months following admission. The study cohort comprised of 53.2% MI and 46.8% unstable angina patients who were admitted to one of twelve coronary care units. This study has previously been discussed in section 2.6.1, 2.7.1 and is discussed further in section 2.12.3.2.

At baseline 36.6% suffered elevated or threshold anxiety scores as measured by the self-report questionnaire Primary Care Evaluation of Mental Disorders (PRIME-MD) (Spitzer et al., 1994). For more than half of these patients raised anxiety persisted for six and twelve months. Anxious patients reported experiencing more atypical symptoms at the time of admission than those who were not anxious. However, during 12 month follow-up although there was a trend for anxious patients to visit their GP and A&E on more occasions than non-anxious individuals, this was not significant (Grace et al., 2004).

In a further study also conducted in Canada involving 441 patients attending A&E with chest pain, 24.5% (81) were reported to be suffering from severe anxiety in the form of panic disorder (PD) (Fleet et al., 1996). The purpose of this study was to report the prevalence of PD and psychiatric co-morbidity in patients who attend A&E with chest pain; psychiatric diagnosis was confirmed through interview by a trained psychologist using the Anxiety Disorders Interview Schedule- Revised (ADIS-R) (DiNardo and Barlow, 1988). A range of other self-report measures were also used including BDI (Beck, 1967) and STAI (Spielberger, 1970).

Reports of atypical or non-ischaemic pain were high (80.0%) in the PD group (Fleet et al., 1996). Additionally significantly more of the PD group (75.0%) received a non-cardiac diagnosis compared to the non-PD group (52.3%, p=0.0002). Comorbid psychiatric conditions including anxiety and depression were significantly
higher in the PD than the non-PD group. Anxiety and depression were not specifically stated for CHD compared to non-CHD patients in this study.

The limitations of this study include the restricted investigation of non-cardiac causes of chest pain. The authors specifically investigated panic disorder and did not explore the possibility of musculoskeletal, gastric or pulmonary problems. Furthermore in this study patients who arrived by ambulance were not recruited; excluding such patients limits the transferability of the results to acutely ill patient groups (e.g. STEMI and PPCI) and those displaying dramatic expression of somatic symptoms of psychiatric distress.

The studies conducted by Srinivasan and Joseph (2004) and Fleet et al (1996) failed to identify the severity of CHD that patients suffered. The severity of a cardiac event in the form of STEMI, N-STEMI, stable or unstable angina may have had great bearing on the results of these studies. Following an acute ischaemic event anxious and depressed patients are more likely to experience an increased number of physical symptoms which are more likely to be atypical for cardiac ischaemia (Mayou et al., 2000, Grace et al., 2004).

2.11.6 Summary

Cardiac and non-cardiac chest pain or symptoms similar to those normally associated with ischaemia have been reported in CHD patients who attend either A&E or cardiology clinics for assessment (Dammen et al., 2004, Ros et al., 1997). Similar findings may be reflected in patients who have experienced STEMI, although as yet this has not been reported in the literature.

The occurrence of non-cardiac chest pain and/or cardiac ischaemic symptoms can lead to difficulty determining an accurate diagnosis for CHD patients. In 68.8% of patients, who present to cardiology clinics for assessment of chest pain, concurrent ischaemic symptoms and non-CHD chest pain were present (Dammen et al., 2004). This increases the complexity of confirming an accurate diagnosis for the cause of pain. In the case of STEMI and PPCI patients who, as previously mentioned, may go on to suffer a further ischaemic event, chest pain and ischaemic symptoms are treated with prudence. There is a tendency for clinicians to err on the side of caution and instigate costly investigations to ensure an ischaemic event is not missed.

psychological sequela and non-cardiac ischaemic symptoms and/or chest pain in CHD patients. However these studies investigated only limited causes of symptoms and presentation. They did not cover all physiological possibilities such as musculoskeletal, gastric abnormalities and lung disease. It is possible that a proportion of patients presented with these non-cardiac causes of pain and/or symptoms.

These studies also failed to report whether symptoms were severe enough to lead to hospitalisation. Furthermore, the studies were not specific to STEMI patients and none included PPCI patients. Both STEMI and PPCI patients are known to suffer recurrent ischaemic pain in the recovery period and are classed as ‘high risk’ for repeat ischaemic events compared to the general population (Keeley et al., 2003). For PPCI patients this includes the additional problem of stent thrombosis and restenosis (Clark et al., 2004).

The design of the studies conducted by Srinivasan and Joseph (2004), Dammen et al (2004) and Fleet et al (1996) are all cross-sectional studies and although they are helpful in identifying the prevalence of disease in certain samples, they do not allow greater depth of understanding where the course of a disease is concerned. A more in-depth investigation of all possible causes of presentation and symptoms, incorporating cardiac ischaemia, physiological causes and a wider range of psychological sequela including general anxiety disorders and depression, would be beneficial in advising clinicians in the assessment of patients.

2.12 Re-presentation

Currently little data are available relating to re-presentation rates due to both cardiac and non-cardiac chest pain or potential ischaemic heart disease (IHD) symptoms for PPCI patients. This section of the review reports the findings from clinical and observational studies for a variety of STEMI and ACS groups.

2.12.1 PPCI re-presentation rates

The majority of literature relating to re-presentation rates for PPCI patients is currently in the form of hospitalisation or major adverse cardiac event (MACE) data from either observational studies or randomised clinical trials (RCTs). However, due to the highly selective patient cohorts included in RCTs the event rates presented often do not reflect those found in more heterogeneous cohorts ‘representative’ of general STEMI patient groups (previously discussed in section 2.3) (Jakobsen et al., 2010). Observational studies can be more helpful in indicating potential re-
presentation rates as in contrast to RCTs, they tend to reflect more representative non-selected populations (Jakobsen et al., 2010). Cohorts that are representative of the general PPCI population will be referred to in this section of the thesis as ‘representative’.

2.12.1.1 Observational studies

Several observational studies have been reported in PPCI cohorts (Lambert et al., 2010, Ortolani et al., 2009). However, in the two observational studies presented in this section re-presentation rates only include selective combined rates, rather than both cardiac and non-cardiac re-presentation rates (Lambert et al., 2010, Ortolani et al., 2009).

In Lambert et al (2010) study (previously discussed in section 2.3.1) the combined death and readmission rate at one year for 1440 PPCI patients was 13.6%. Hospitalisations due to heart failure or MI were classified as readmissions in this study (Lambert et al., 2010).

In a further Italian study, 1124 PPCI patients from one PCI centre were included in the analysis if they had received a glycoprotein IIb/IIIa agent to reduce thrombus during or after PCI (Ortolani et al., 2009). This accounted for 83.0% of the PPCI population treated at the PCI centre during the recruitment period 2003 to 2006. Outcome data were obtained retrospectively from codified hospital discharge records (obligatorily archives for all Italian hospitals) and Municipal Civil Registries.

In this study all cause mortality and re-infarction was quoted as 21.0% for the total study cohort at 2 year follow-up (Ortolani et al., 2009). Additionally, MACE (i.e. death, re-infarction, target vessel revascularisation) was found to be 27.6% for the study group. In this study it was demonstrated that patients had improved outcome when they received a IIb/IIIa agent early (in the emergency department or during ambulance transfer) compared to late (in the catheter lab), MACE (23.0 vs. 30.0%, p=0.01) (Ortolani et al., 2009).

The study cohort in Ortolani et al (2009) study was closer to a representative patient cohort than those usually included in an RCT. However, the study sample still remained more selective than a general STEMI patient population as patients were only included if they had received treatment with a IIb/IIIa agent (83.0% of patients). Treatment with IIb/IIIa agents is often reserved for high risk patients, although practices do vary from centre to centre (Antman et al., 2004).
2.12.1.2 Randomised controlled trials

A range of MACE rates from RCTs conducted in PPCI cohorts are presented in section 2.3.1 and these are helpful to a degree in projecting potential re-presentation rates for PPCI patients. Generally MACE rates comprise of composite events such as death (due to CHD), recurrent MI, revascularisation and stroke; the variables included often differ between studies.

Reported MACE rates for PPCI patients are between 4.8% and 10.3% at 30 days, 11.7% to 14.6% at 6 months and 11.9% at 12 months (GUSTO IIb angioplasty substudy investigators, 1997, Montalescot et al., 2001, Mehran et al., 2009). However, the usefulness of combined MACE and clinical event rates is limited when considering re-presentation of PPCI patients. This is partly due to the inclusion of only a few selected clinical events. Furthermore, the inclusion of death may distort the event rate, as some of those who die will not have reached hospital.

In addition, most of the evidence presented in both the observational studies and RCTs only reflect readmissions due to cardiac causes, and re-presentations due to other physical origins and psychological issues are not included.

2.12.2 Rescue PCI Patients

Rescue PCI patients are a similar group to PPCI patients as they have suffered STEMI and received PCI under urgent /emergency conditions following failure of initial thrombolytic therapy to open the infarct related artery (Mortensen et al., 2000). Re-presentation and rehospitalisation information for this group may be helpful in extrapolating rates for PPCI patients.

Two randomised controlled trials, REACT and MERLIN (Gershlick et al., 2005, Sutton et al., 2005) have evaluated the effectiveness of rescue PCI and although they do not report readmission rates, they do present MACE rates. In the MERLIN study 307 failed thrombolysis patients were randomised to receive either rescue PCI (153) or conservative therapy (154). The MACE rate (stroke, re-infarction and heart failure) for the rescue PCI group was 54.3% at one year (Sutton et al., 2005). In the REACT study the MACE rate (death, stroke, re-infarction and severe heart failure) for rescue PCI patients at 6 months was 15.3% (Gershlick et al., 2005); this rate was far lower than that of the MERLIN study even when taking into consideration the difference in follow-up. In this study 427 patients were randomised to receive rescue PCI (144), conservative therapy (141) or repeat thrombolysis (142).
The disparity of event rates for these two studies is likely to have been influenced by multiple factors, including differences in the patient’s medical condition at the time of presentation, infarct size, time to receiving rescue PCI, type of initial thrombolytic drug therapy and drug therapy during and after rescue PCI. The usefulness of the clinical event rates from these two studies is limited when considering the PPCI population, by the large differences in the values.

2.12.3 Causes of re-presentation

Most of the re-presentation literature relating to MI cohorts reviewed in this section did not take into account the type of index MI treatment (PPCI or lysis). Additionally, the studies tended to only include patients who were hospitalised and excluded those who attended the accident and emergency (A&E) department for assessment. The majority also did not define reasons for readmission other than cardiac related causes; generally there was an implicit assumption that patients only return with cardiac causes. Readmissions for other causes such as physical and psychological comorbidities were not included, despite the evidence that anxiety and depression are associated with CHD and share similar symptoms with IHD (Frazier et al., 2002, Dammen et al., 2004). Patients with psychological sequelae often present to emergency services with similar symptoms to those assigned to cardiac causes (Fleet et al., 1996).

2.12.3.1 Re-presentation to accident and emergency department

Hubbard et al (2007) went some way towards investigating the re-presentation to A&E due to potential cardiac ischaemia for a mixed PCI cohort within 30 days of PCI treatment (n=2,731). The final diagnosis related to the re-presentation event was also reported for some of the study cohort. Patients were defined as re-presenting with a potential cardiac event if they had blood taken and tested for a serum cardiac biomarker during their attendance at the A&E department. Data were collected retrospectively through the hospital billing systems and through the patients’ hospital records.

A total of 328 (12.0%) patients re-presented to the A&E department within 30 days following PCI treatment and 73 (2.7% of the entire cohort) were found to have a non-cardiac complaint and were not tested for cardiac ischaemia through collection of serum biomarkers. The remaining 255 (9.3%) were investigated for a potential cardiac ischaemic event.
A diagnosis was available for 70.0% of the 255 patients who re-presented and were investigated for a potential cardiac event. The diagnoses included 7.0% (15/255) MI, 20.0% coronary atherosclerosis, 6.0% heart failure and 5.0% cardiac dysrhythmia; the largest proportion 33.0% (84/255) had non-specific chest pain.

Although the study involved a mixed PCI cohort, 80.0% of patients were treated at the time of index PCI as an emergency or under urgent conditions and of those almost half (46.0%) received emergency PCI for MI. The odds of re-presenting to A&E for patients who had received either emergency or urgent index PCI were twice as high as for those who had received elective PCI (OR 1.98, 95% CI 1.3, 3.0). Furthermore, the odds of women re-presenting was twice as high compared to men (OR 1.9, 95% CI 1.5, 2.5) (Hubbard et al., 2007).

There were a number of limitations associated with this study including 30.0% of the re-presentation group who did not have a diagnosis recorded. This may have been due to the retrospective data collection methods used, which can make accurate data collection difficult (see section 4.8.5.1). The large proportion of patients without a diagnosis may have led to inaccurate categorisation of the patients’ diagnosis (i.e. cardiac, non specific chest pain etc). Furthermore, the authors only categorised cardiac related diagnoses and the non-cardiac diagnoses were not described; anxiety and depression were also not measured. It is possible that the 33.0% of patients with a diagnosis of non-specific chest pain were suffering from psychological problems. Potentially, the larger number of emergency PCI patients who re-presented compared to the elective PCI group may also have been related to psychological distress. The higher number of women compared to men re-presenting also adds weight to this possibility as both anxiety and depression are more prevalent in women than men in the general population.

2.12.3.2 Emergency re-presentation due to psychological sequelae

Heightened anxiety at the time of an ACS event was not found to increase the number of attendances to A&E over 12 month follow-up in a study of 906 ACS patients (Grace et al., 2004). The main aim of the study was to evaluate anxiety symptomology, cardiac symptoms (at the time of index ACS event), health utilisation and recurrent cardiac events over one year following an ACS event. The cohort comprised of 424 unstable angina and 482 MI patients. This study was previously discussed in sections 2.6.1, 2.7.1 and 2.11.5.

The authors reported that there was no difference in A&E attendances at 12 months between those with anxiety (mean attendances =1.11) and those without anxiety.
(mean=0.83, p=0.17). Anxiety levels were measured using the Middlesex Hospital Questionnaire (MHQ), phobic anxiety scale (Crown and Crisp, 1996) and the PRIME-MD, anxiety subscale (Spitzer et al., 1994). At 12 months recurrent cardiac events (ACS, arrhythmia, heart failure, stroke, atherosclerosis requiring treatment and mortality) were predicted by older age, depression, cardiovascular disease family history and increased anxiety.

The main focus of the Grace et al study (2004) was not A&E attendances and the authors used patient reports at 6 and 12 months to record A&E visits. It is therefore possible that there was under reporting of A&E attendances in this study as often patients may have difficulty recalling such events over a long period of time. Furthermore, the authors did not report details related to A&E visits such as diagnosis. The number of cardiac events was recorded for those with and without anxiety, but non-cardiac events were not reported.

Additionally, Mayou et al (2000) also did not find a statistically significant difference at 12 months post MI, between patients with distress (HADS psychological distress ≥ 19) and those without distress, in terms of A&E attendance. At one year following MI 15.0% of those with psychological distress compared to 2.5% without distress returned to A&E on more than 4 occasions. Yet the authors do report that increased anxiety and depression symptoms at baseline predicted the overall number of primary care visits (p<0.05) and general practitioner (GP) surgery (p<0.05) visits at 3 and 12 months. Additionally there was a trend for distressed patients to have more emergency outpatient appointments and A&E visits than non-distressed patients (Mayou et al., 2000).

Like Grace et al (2004), at one year, Mayou et al (2000) relied on patient reports regarding whether they had attended A&E more than four times; this may have led to unreliable findings due to poor recall. Furthermore, although one of the study aims was to examine the association between baseline anxiety and depression and service utilisation, the authors do not state how many A&E attendances or hospital admissions patients experienced during follow-up. The lack of detail makes it difficult to reliably apply the study findings to other MI study cohorts such as PPCI patients.

2.12.3.3 Anecdotal reports

In accordance with the published literature anecdotal reports from clinicians describe the return of post-PPCI patients to emergency services with symptoms indicating potential myocardial ischaemia. Due to the patients’ cardiac history they
are regarded as high risk and are usually rehospitalised and undergo investigations for ischaemia including angiography. It is unclear what proportion of PPCI patients in these circumstances receives a diagnosis of acute ischaemia or how many present with non-cardiac symptoms. According to Hubbard et al (2007) 9.3% of PCI patients returning to A&E with cardiac ischaemic symptoms were diagnosed with a further cardiac event and 2.7% had a non-cardiac complaint (see section 2.12.3.1). However the study cohort was mixed and included 20.0% of stable patients, it is therefore difficult to extrapolate these findings to PPCI patients. Furthermore, it is not known what proportion of PPCI patients who re-present go on to receive a diagnosis of negative cardiac ischaemic aetiology.

2.12.4 Summary

Most of the literature that focuses on re-presentation rates for PPCI patients reports either hospitalisation or composite cardiac event rates. The data were obtained from both observational studies and randomised controlled trials.

In observational studies death and readmissions were reported in 13.6% of patients at one year, and death and re-infarction in 21.0% at two years following PPCI (Ortolani et al., 2009, Lambert et al., 2010). A variety of composite event rates for PPCI cohorts have been reported through RCTs and are between 4.8% and 10.3% at 30 days, 11.7% to 14.6% at 6 months and 11.9% at 12 months (GUSTO IIb angioplasty substudy investigators, 1997, Montalescot et al., 2001, Mehran et al., 2009). Event rates including stroke, MI and heart failure for rescue PCI patients are reportedly 9.0% at 6 months (Gershlick et al., 2005) and 54.3% at one year (Sutton et al., 2005, Gershlick et al., 2005).

The re-presentation rates to acute services (such as A&E) due to potential IHD symptoms for PPCI patients have not previously been reported. However, for a mixed cohort of PCI patients they were reportedly 12.0% at 30 days following index PCI (Hubbard et al., 2007). Almost half of the PCI cohort comprised of emergency PCI patients. Re-presentations were categorised as 2.7% non-cardiac, 37.0% cardiac related, 33.0% non-specific chest pain, whilst 30.0% did not have a diagnosis available. Patients treated during their index admission with emergency PCI were twice as likely to re-present as those in receipt of elective PCI. The authors did not describe other non-cardiac causes of re-presentation nor did they measure the psychological health of the study cohort (Hubbard et al., 2007).

Symptoms of anxiety and depression during admission for MI have been found to play a part in additional emergency GP surgery visits and A&E attendances (Mayou...
et al., 2000). However, raised anxiety has not been linked to increased A&E attendances at 12 months following an MI or ACS events (Mayou et al., 2000, Grace et al., 2004). Although it is possible that the study methods used by the authors of the two studies sited may have led to underestimation of attendances to A&E by the participants.

Few studies have investigated re-presentation rates due to cardiac and non-cardiac factors for STEMI cohorts and no current studies are available reporting such event rates for PPCI patients. There is clearly a gap in the current literature base regarding this group and the factors that lead to re-presentation.

2.13 Summary of literature review

The literature presented in this review demonstrates that following STEMI, patients are prone to experiencing recurrent cardiac events and/or symptoms of ischaemic heart disease (IHD) (Austin et al., 2011, Jakobsen et al., 2010, Mehran et al., 2009). Additionally, patients have a propensity towards increased psychological distress, which has been associated with increased IHD symptoms such as angina (Lespérance et al., 2002, Parashar et al., 2006, Martens et al., 2010, Roest et al., 2010). However, the psychological health of STEMI patients treated with PPCI is currently under represented in the literature. The literature search and review presented in this chapter support the conceptual framework presented in Chapter 3, which includes aspects of symptoms, psychological and physiological health.

Following PPCI it has been reported through qualitative studies that patients experience distress and shock due to having experienced a heart attack and this is compounded by the speed of treatment (Sampson et al., 2009, Astin et al., 2009). Timely PPCI treatment benefits patients in terms of clinical improvement and patients view PPCI positively whilst also regarding it as a curative treatment (Sampson et al., 2009, Keeley et al., 2003, Astin et al., 2009). However, the hasty speed of PPCI treatment has been reported by patients to act as a barrier to information absorption (Astin et al., 2008). Additionally, one of the main information needs of PPCI patients in the Astin et al (2008) study related to future symptom attribution.

A number of non-cardiac physiological conditions, as well as anxiety and depression, can share similar symptoms to those associated with IHD (Dammen et al., 2004, Mayou et al., 2000, Ros et al., 1997). Cardiac and non-cardiac symptoms have also been shown to occur concurrently in CHD patients (Dammen et al.,
This may lead to difficulty with symptom attribution for STEMI patients who experience potential IHD symptoms and may result in them re-presenting to healthcare services. This is particularly relevant when patients believe that their symptoms are related to their heart.

Currently clinical re-presentation rates for PPCI patients at one year are reported as 13.6% (death and readmission) and at two years 21.0% (death and MI) in observational studies (Ortolani et al., 2009, Lambert et al., 2010). However, the studies reviewed only include selective clinical events and do not include PPCI patients who attend acute healthcare services with potential IHD symptoms due to cardiac and non-cardiac causes. Additionally, the psychological health of PPCI patients attending for review of acute IHD symptoms has not previously been presented.

2.14 Justification for a study

There is currently a paucity of evidence relating to the re-presentation of PPCI patients to acute services due to potential IHD symptoms. Clearly there is evidence that repeat cardiac events and diminished psychological health are problems faced by patients following STEMI (Strik et al., 2003, Mehran et al., 2009). Additionally, repeat cardiac symptoms are associated with reduced psychological health (Dickens et al., 2006, Parashar et al., 2006).

The similarity of IHD symptoms and those related to psychological health complicates ascertaining the cause of symptoms for patients (Ruston et al., 1998). Clinicians report that there is a tendency for PPCI patients to return to acute healthcare services when such symptoms occur. The proportion of patients representing in this manner within the first 6 months of PPCI treatment is currently unknown. Additionally the factors associated with re-presentation due to potential IHD symptoms are unclear.

There is clearly a gap in the current literature base relating to PPCI patients and the factors that lead to re-presentation to acute healthcare services. This leads to the need for a study to determine re-presentation rates for PPCI patients returning due to potential IHD symptoms. Additionally the associated factors related to re-presentation require investigation. Patients who re-present with potential IHD symptoms are a distinct group. Undertaking a study to better understand this group will build evidence to aid development and adaptation of aftercare services. This is particularly relevant as PPCI is a comparatively new service in the UK and
psychological health, patient recovery and after care has to date received little attention or evaluation not only for those who re-present, but also the cohort as a whole.
CHAPTER 3 CONCEPTUAL FRAMEWORK

3.1 Introduction

This chapter specifies the conceptual model and conceptual framework that underpins this study. The evidence to support the concepts and inter-linkages between concepts are discussed.

The conceptual model is further expanded and a more detailed description of the concepts investigated through this study are also presented in this chapter. Furthermore, the relationships between concepts in this study that were investigated through the logistic regression model are defined and discussed.

3.2 Definition of a conceptual framework and conception model

A conceptual framework is a means of describing the important theories, relevant evidence and knowledge, as well as the linkages between concepts that form the basis of a research study (Fawcett, 2000, Parahoo, 1997). The important concepts that underpin the area being investigated may be drawn from a combination of theories, the literature and clinical practice (Polit and Beck, 2004). Mock (2007) suggests that the use of a framework in conducting research demonstrates the overall conceptual design of the study.

The terminology used when referring to ‘conceptual framework’ is often interchangeable with ‘theoretical framework’; likewise, the term ‘model’ is used in place of ‘framework’ by some authors and nurse researchers (Fawcett and Gigliotti, 2001, Mock et al., 2007). For the purposes of this study the term ‘conceptual framework’ will be used to define the factors and relationships between the concepts that underpin re-presentation (due to potential IHD symptoms) within 6 months of STEMI and PPCI treatment.

Earp and Ennett (1991) refer to a conceptual model as a visual representation of the conceptual framework and include only the most important concepts involved within an area of interest. The purpose of a model is to aid the understanding and simplification (in a diagrammatic form) of the underlying concepts involved in a conceptual framework (Earp and Ennett, 1991). The illustrative nature of a model enables linkages between concepts to be easily visualised.
3.2.1 Development of the study conceptual framework and model

Factors and ideas that arose from an in-depth review of the literature and appraisal of clinical practice (e.g. psychological health, physiological health, sociodemographic factors and symptoms) were considered for inclusion in the conceptual model and conceptual framework for this study.

The definitions throughout this chapter include:

- **Re-presentation**, refers to individuals who re-presented to acute healthcare services with potential Ischaemic Heart Disease (IHD) symptoms.
- **Potential IHD symptoms**, refers to the occurrence of symptoms that participants believed to be related to their heart (potential IHD symptoms are also referred to as symptoms in this section of the thesis).

3.3 The conceptual model

Figure 3-1 demonstrates the factors at baseline deemed by the author in consultation with the supervisory team, to be most importantly connected with re-presentation; those considered to be directly related included potential IHD symptoms, psychological health and physiological health. Symptoms and physiological health were also regarded to be inter-related with psychological health in this study. Additionally, sociodemographic aspects were considered to play an indirect part in re-presentation.

The linkages between factors considered to be directly associated with re-presentation are demonstrated in Figure 3-1 with block arrows and the double head arrow indicate the inter-related concepts. The factors and linkages between the aspects of the model are discussed in the conceptual framework section 3.4.

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1 Initially during the developmental stages of this study the researcher planned to use chest pain to determine the re-presentation group. However, not all patients, and in particular women, experience chest pain. It was therefore decided to include a range of symptoms that are typically regarded as related to IHD symptoms (see section 1.2.2 and 2.11).
3.4 Conceptual Framework

Re-presentation (due to potential IHD symptoms) following STEMI is initiated by the occurrence of symptoms often perceived by patients to be angina and interpreted as an indication of serious health issues.

The cause of potential IHD symptoms at the time of a re-presentation event may be related to an escalation of cardiac symptoms (that have been occurring since STEMI) or a psychological or an alternative physiological cause (Dammen et al., 2004, Mayou et al., 2000, Lambert et al., 2010, Ros et al., 1997). This is due to the similarity of symptoms between cardiac problems (e.g. Acute Coronary Syndrome (ACS), heart failure, an arrhythmia or angina), anxiety and/or depression and physiological problems (such as musculoskeletal, pulmonary, or a gastrointestinal event) (see section 2.5.2 and 2.11). In some instances patients may experience symptoms that are a combination of psychological and physiological health factors and differentiating between the causes of symptoms can be challenging for not only patients but also healthcare professionals (Dammen et al., 2004, Grace et al., 2004, Mayou et al., 2000).

Sociodemographic attributes (e.g. age, gender, education and employment status), may influence symptoms, psychological and physiological factors involved with re-presentation. However, they are unlikely to be directly linked to re-presentation. Age, for example, may be implicated with physiological and psychological health (see section 3.5.4.3). In the case of physiological health, older individuals are more
likely to experience more comorbidities than younger individuals, and in turn the
comorbidities may lead to increased symptom occurrence (Alexander et al., 2007,
Weir et al., 2006). Conversely, anxiety and depression may influence factors such
as delayed return to work or changing social and family roles for younger
individuals following STEMI (Charmaz, 1994, Fukuoka et al., 2009).
Sociodemographic factors were therefore considered to be indirectly associated with
re-presentation in this study.

3.5 Expansion of the conceptual framework and
consideration of variables for the regression
model

The development of the conceptual framework and model discussed in section 3.4
included a detailed investigation of the possible factors (listed in Table 3-1)
involved with re-presentation. Additionally the potential links between the factors
and with re-presentation are demonstrated in Figure 3-2. The development of the
regression model and the decisions taken by the author to include or exclude
attributes from the regression model are discussed in this section.

Table 3-1 Expansion of the conceptual model and the main factors considered for inclusion in
the regression model

<table>
<thead>
<tr>
<th>Conceptual model</th>
<th>Attributes investigated for the regression model</th>
</tr>
</thead>
</table>
| Symptoms               | Angina frequency at baseline  
|                        | Angina stability at baseline  
|                        | Physical limitation (due to angina symptoms) at baseline  
| Psychological          | Anxiety at baseline (new onset)  
| health factors         | Depression at baseline (new onset)  
|                        | Pre-STEMI anxiety and depression  
| Physiological health   | Severity of STEMI  
| factors                | Comorbiditity  
|                        | History of previous Ischaemic Heart Disease (IHD) event  
|                        | Further cardiac revascularisation (at 6 months)  
| Sociodemographic       | Education  
| factors                | Social support  
|                        | Marital status  
|                        | Age  
|                        | Gender  

Figure 3-2 Expansion of the conceptual model and factors investigated for inclusion in the regression model

- Education
- Marital Status
- Social Support
- Gender
- Age
- Comorbidity
- Angina Symptoms
- Anxiety
- Depression
- Pre-STEMI anxiety & depression
- Further revascularisation at 6 months
- Severity of STEMI / CHD
- History of previous IHD events

Representation with potential IHD symptoms
3.5.1 Potential IHD symptoms

The symptoms experienced by participants at baseline may have been due to either psychological or physiological factors or a combination of both; this is likely to be attributed to the reported similarity between symptoms. Reduced psychological health and ACS events share similar symptoms, such as chest pain, breathlessness, nausea, sweating and an impending sense of doom (Yingling et al., 1993, American Psychiatric Association, 2000, Dammen et al., 2004). Furthermore, other physiological problems such as gastric and pulmonary conditions may also mimic certain aspects of cardiac symptoms including chest pain and breathlessness (Dammen et al., 2004, Ros et al., 1997). See section 2.11 of the literature review relating to cardiac ischaemic symptoms.

The similarity between symptoms can lead to difficulty with symptoms attribution for the affected patient (Dracup et al., 1997, Ruston et al., 1998). It is therefore possible that when patients believe that they are experiencing IHD symptoms (such as angina) following STEMI, they may seek help via acute services.

In the current study symptoms were assessed at baseline and 6 months using a self-report instrument, the Seattle Angina Questionnaire (SAQ); the SAQ was specifically developed to measure ‘angina’ symptoms (Spertus et al., 1995) (see section 5.10.2). Although the SAQ was intended by the authors to measure angina, the similarity between cardiac and other disease symptoms is likely to lead to the capture of all symptoms that participants believe to be angina. The frequency and stability of angina symptoms (for those who experienced symptoms) were recorded (using SAQ) to gain a sense of the severity of angina. Furthermore, whether angina symptoms led to physical restrictions were also recorded to determine the impact that angina had on the participant’s lifestyle. The variables angina frequency, angina stability and physical limitation (due to angina) were included in the regression model to determine whether the presence of IHD symptoms at baseline predicted re-presentation at 6 months.

3.5.2 Psychological health factors

Anxiety and depression following STEMI have been reported to negatively influence physical health factors (Carney et al., 2002, Dickens et al., 2007, Grace et al., 2004, Roest et al., 2010). Anxiety has also been reported as an important factor during A&E attendances for chest pain and ACS events (Grace et al., 2004). Furthermore, reduced psychological health is known to influence the Health Related Quality of Life of patients who suffer IHD symptoms (Parashar et al., 2006). The
literature relating to anxiety and depression are described in section 2.5. The potential relationships between symptoms, psychological and physiological health are discussed in section 3.5.1.

3.5.2.1 New onset anxiety and depression

Raised anxiety and depression levels are not uncommon during and following STEMI (Strik et al., 2003, Frazier et al., 2002). Anxiety and depression are reportedly interlinked and often occur concurrently (Bech, 2006). Additionally the associated symptoms may mimic those related to physiological problems such as cardiac ischaemia (Dammen et al., 2004). Due to the similarity between symptoms it is possible that raised levels of anxiety and/or depression at baseline may be implicated with re-presentation.

In particular individuals suffering from anxiety may experience a sense of foreboding and believe that something terrible is going to happen (American Psychiatric Association, 2000). Likewise, at the time of an ACS event patients report feeling anxious and a foreboding fear of dying (Whitehead et al., 2005, Grace et al., 2004). Whitehead et al (2005) determined that almost three quarters of patients experienced a fear of death during an ACS event. Grace et al (2004) found that as many as 30.0% of ACS patients experienced raised anxiety levels at the time of IHD event. Furthermore, it is reported that individuals with elevated anxiety levels at the time or shortly after an ACS event are at greater risk of further ischaemia, re-infarction and cardiac mortality (Frasure-Smith, 1991, Grace et al., 2004, Moser and Dracup, 1996). It has also been reported that individuals who have heightened anxiety during an ACS event experience more symptoms (Grace et al., 2004).

Depression has also been strongly associated with mortality and morbidity following STEMI (Frasure-Smith et al., 1993, Frasure-Smith et al., 1995a). Likewise, the symptoms of depression have been linked with physical limitation and symptom burden in IHD patients (Parashar et al., 2006). Furthermore, Grace et al (2004) reported that heightened depressive symptoms at the time of an ACS event were predictive of recurrent cardiac events. Dickens et al (2008) also found that new onset of depression was predictive of increased cardiac mortality up to 7 years following MI. Although, neither Grace et al (2004) nor Dickens et al (2008) state explicitly whether the recurrent cardiac events or cardiac mortalities led to an acute re-presentation, it seems reasonable to assume that in some cases this may have occurred.
The suggestion in the literature that a relationship may exist between ‘new onset’ anxiety and depression with re-presentation has not previously been investigated. In the current study it was decided to include measures of anxiety and depression at baseline within the study regression model, to investigate the potential association between reduced psychological health and re-presentation. In the current study anxiety was considered to be of the greatest theoretical importance due to the evidence suggesting the relationship between anxiety and increased symptoms at the time of an ACS event and during A&E attendance due to chest pain (Fleet et al., 1996, Grace et al., 2004).

3.5.2.2 Pre-STEMI anxiety and depression

The literature suggests that depression prior to MI leads to poor outcome following STEMI. Dickens et al (2005) found that in first MI patients symptoms of depression (measured one week prior to MI) were associated with worse cardiac failure. Currently there is no direct evidence suggesting that anxiety prior to MI leads to poor outcome. However it has been reported that anxiety in the general population leads to increase incidence of death due to CHD and sudden cardiac death (Kawachi et al., 1994); additionally symptoms of anxiety and depression tend to coexist (Bech, 2006).

The occurrence of cardiac failure and increased mortality associated with reduced psychological health following an ACS event (such as STEMI) may in turn lead to cardiac symptoms and potentially an acute re-presentation. The author considered including the factor ‘pre-STEMI anxiety and depression’ in the regression model. However, it was decided not to include this factor because determining levels of anxiety and depression accurately pre-STEMI was particularly problematic, due to retrospective data collection (see section 4.8.5). Retrospective data collection is known to be inherently problematic in terms of data accuracy and validity (Schulz and Grimes, 2002).

Furthermore, the variable ‘pre-STEMI anxiety and depression’ was a dichotomous variable (yes or no) and wherever possible continuous variables were included in the regression model. This was due to the likely small number of participants to represent with IHD symptoms, and Peduzzi et al (1996) stated that for a reliable estimation, the ratio of individuals to predictor variables should be at least 10:1 for each of the outcome categories. The discussion regarding the number of variables to be included in the regression model is presented in section 5.23.1.
3.5.3 Physiological health factors

A number of factors may influence or relate to the physiological health of individuals following STEMI and result in patients re-presenting to acute services. Factors include the severity of STEMI, comorbid illnesses, a past history of IHD events and further cardiac revascularization post STEMI (Sidney et al., 2005, Montalescot et al., 2001, Stone et al., 2002). The potential role that each of these factors may play in re-presentation is discussed in this section of the thesis. The choice of factors for inclusion in the regression model and the justification for their inclusion is also addressed.

3.5.3.1 Severity of STEMI

Myocardial infarction involves death of the myocardial cells (Thygesen et al., 2007). Damage to the myocardium is limited or completely reversible when PPCI occurs early (within 90 minutes) of onset of symptoms. However, when treatment is delayed or extensive areas of the heart are affected by infarction, long term damage may ensue (Berger et al., 1999, De Luca et al., 2003). The full description of the clinical background surrounding STEMI can be found in section 1.2.

Establishing how much damage has occurred to the myocardium in the form of size and area of infarcted myocardium can have important prognostic and physiological implications for patients (Antman et al., 2008). Individuals with more cardiac damage due to larger infarcts are found to suffer increased mortality and cardiac morbidity than individuals with less myocardial injury due to smaller infarcts (De Luca et al., 2003, Fabiani et al., 1993, Sabia et al., 1991). Cardiac morbidity in the form of further ACS events and heart failure (HF) post STEMI are reported to lead to additional symptoms and hospitalisation (Morrow et al., 2000, Hubbard et al., 2007). It would therefore seem reasonable to include a measure of ‘severity of STEMI’ as a variable in the regression model, as one can extrapolate that if cardiac mortality and morbidity are lessened then re-presentation rates may also be reduced.

Currently there is not one measure that can describe the severity or size of infarct. In clinical practice clinicians use a combination of factors to assess the size and the clinical impact of the infarct. The joint European Society of Cardiology and American Heart Association (AHA) taskforce for the redefinition of myocardial infarction (2007) recommended that the size of infarct is identified by: interpretation of the electrocardiogram (ECG), assessment of Killip class during the acute phase, the use of biomarkers (generally troponin ‘T’ or ‘I’) at 9 to 12 hours post STEMI (Killip and
Kimball, 1967, Mills et al., 2011) and measurement of left ventricular (LV) function (Antman et al., 2004, Sabia et al., 1991) (see section 1.2). However, the use of multiple clinical measures for the assessment of the severity of an infarct can be challenging. The difficulties involved with defining severity of STEMI are acknowledged by National Institute of Clinical Excellence (NICE, 2010a) and the American College of Cardiology (ACC) and the AHA taskforce for practice guidelines for the management of patients with STEMI (Antman et al., 2004, Antman et al., 2008). Some authors recommend the use of an aggregated risk assessment measure to predict outcome as a means of defining the severity of STEMI (Granger et al., 2003, Fox et al., 2006) and such scores are now recommended by NICE (2010b). It was therefore decided to incorporate an aggregated measure to record predicted outcome following STEMI as a measure of severity of STEMI in this study.

Although there are a number of aggregated mortality risk assessment tools available, such as the ‘Thrombolysis in Myocardial Infarction’ (TIMI) risk score for STEMI (Morrow et al., 2000) and the multivariable model developed in the ‘Global Utilization of Streptokinase and t-PA for Occluded Arteries’ (GUSTO)-I trial (Lee et al., 1995), the the ‘Global Registry of Acute Coronary Events’ (GRACE) score (Granger et al., 2003, Fox et al., 2006) is currently advocated by NICE (2010b) as the most appropriate composite risk prediction measure.

Due to the potential relationship that ACS symptoms may have with re-presentation, it was decided to include the GRACE score in the regression model, as it is a continuous variable and includes parameters adopted in clinical practice at admission to assess the severity of STEMI. The GRACE score is discussed in detail in section 5.10.4.2.

3.5.3.2 Comorbidity

Participants with comorbid conditions may be more likely to seek help from acute services than individuals who do not have comorbid conditions (Sidney et al., 2005). This may be due to the degree of illness symptoms that each individual experiences relating to other disease states (Taneva et al., 2004, Sidney et al., 2005).

Symptoms associated with a comorbidity may be similar to those related to IHD (Dammen et al., 2004, Ros et al., 1997). This may lead to participants finding it difficult to distinguish between symptoms related to IHD, anxiety and depression and comorbidities, which may bring them back to acute services. It was therefore
decided to determine whether there was an association between comorbidity and re-presentation, by including comorbidity in the regression model.

A number of specific comorbidities were considered for inclusion in the regression model as individual variables; they included pulmonary diseases, gastric abnormalities and heart failure (Dammen et al., 2004, Ros et al., 1997). Their inclusion was considered due to some of the similarity in symptoms that are shared with IHD. For example individuals suffering pulmonary disease and heart failure may experience breathlessness, a symptom that can be present during ACS events (McMurray, 2010). Likewise, gastric abnormalities can lead to chest pain (Ros et al., 1997), which may be mistaken by patients as a cardiac event. Diabetes was also considered for inclusion in the model, as diabetes is a known risk factor in the development and progression of IHD (Goldberg, 2000, Franklin et al., 2004). It is reported that individuals with IHD and diabetes experience more atypical symptoms and delay in seeking help at the time of STEMI (Franklin et al., 2004).

The recording of the individual comorbidities required the use of either dichotomous (i.e. present or not present) or categorical variables. The use of the individual comorbid conditions was discarded due to the limited number of variables that could be included in the regression model (see section 5.23.1). It was decided to use the Charlson Co-morbidity Index (CCI) at baseline to record the frequency and severity of comorbid conditions, because it is a composite, continuous measure (Charlson et al., 1987). The CCI is discussed in detail in section 5.10.5.

3.5.3.3 History of previous IHD events

Participants who have previously experienced an ACS event or stable angina prior to STEMI, are at greater risk of further cardiac ischaemia due to ongoing disease (GUSTO IIb angioplasty substudy investigators, 1997, Stone et al., 2002, Montalescot et al., 2001). It is therefore likely that those who have a history of a past IHD event will be susceptible to IHD symptoms and may seek help via acute services. It was deemed possible that a history of past IHD event may be associated with re-presentation and was therefore an important variable requiring inclusion in the regression model.

3.5.3.4 Further cardiac revascularisation

Primary Percutaneous Coronary Intervention (PPCI) has been shown to be highly effective at re-establishing coronary flow in the infarct related artery. However, it is not uncommon for STEMI patients to require additional revascularisation of further
diseased vessels following PPCI (Stone et al., 2002). This may be due to the identification of more diseased vessels at the time of STEMI requiring a staged Percutaneous Coronary Intervention (PCI) procedure, usually 6 weeks following the PPCI. The additional diseased vessels may lead to participants experiencing angina symptoms during the time that they are awaiting PCI. Furthermore, individuals may develop new disease, with the formation of further atherosclerotic plaque, and this may lead to either an ACS event or ongoing angina symptoms (Hubbard et al., 2007). In both instances symptoms may lead to re-presentation. It is therefore justifiable to include further revascularisation in the regression model, as it indicates additional diseased coronary arteries.

3.5.4 Sociodemographic factors

There is evidence to suggest that sociodemographic factors may be interlinked with symptoms, psychological and physiological health following an ACS event (Alexander et al., 2007, Mortensen et al., 2007). Factors such as age, gender, education and social support have all previously been investigated in relation to STEMI (Moss et al., 1969, Shaw et al., 2006, Rothwell et al., 2005, Berkman and Glass, 2000). These sociodemographic factors are discussed in the following sections.

3.5.4.1 Education

The participants’ level of education was considered for inclusion in the regression model, as it was considered that level of education may influence patients’ interpretation of symptoms. The majority of studies investigating education in MI patients have considered whether education level influences the time taken for patients to seek help. Several studies determined that level of education does not positively or negatively influence time delay in seeking help during MI (Dracup et al., 2008, Ell et al., 1994, Wielgosz et al., 1988). This evidence may indicate that the level of education does not influence the interpretation of symptoms that then leads to help seeking. It was therefore decided to not include level of education in the model.

3.5.4.2 Social support

A large body of evidence suggests that supportive social relationships have a benefit on health (Berkman and Glass, 2000). In the cardiac population, particularly post MI patients, a growing literature has shown that low social support is associated with increased mortality, cardiac events and poor quality of life (Berkman et al., 1992, Burg et al., 2005, Molloy et al., 2008, Ruberman, 1992).
Dickens et al (2004) reported that individuals without the social support of a close confidant were more likely to suffer further cardiac events following MI than individuals with a confidant. Additionally, reduced social support has been reported as a precursor to depression and poor recovery post MI (Lett et al., 2007, Dickens et al., 2004).

It seems reasonable to consider that higher mortality and increased prevalence of depression due to reduced social support may increase the prospect of individuals experiencing symptoms and the possibility of re-presenting to acute services. Furthermore, the inability to check out symptoms with a loved one or confidant may influence the time that it takes an individual to call for help during an ACS event (Horne et al., 2000). It was therefore decided to include social support in the regression model to determine whether social support was associated with re-presentation in this study.

Marital status was considered as a measure of social support in the current study, but this was discounted because marital status does not necessarily indicate whether an individual receives emotional or social support. A more reliable method of ascertaining the level of emotional and social support was with the use of a specific measure. The Enhancing Recovery in Coronary Heart Disease (ENRICHD) social support instrument (ESSI) was specifically designed and validated for use in cardiac patients (Vaglio et al., 2004, ENRICHD Investigators, 2000). For the purposes of this study the ESSI was chosen to collect data related to the quality and availability of social support to participants (Mitchell et al., 2003). The instrument is described further in section 5.10.3.

3.5.4.3 Age

At the time of STEMI elderly individuals are more likely to present with atypical symptoms and delay seeking help (Antman et al., 2004, Rothwell et al., 2005, Dracup et al., 1995). Elderly individuals are also more likely to have more comorbid conditions at the time of STEMI (Alexander et al., 2007).

Issues for younger individuals may relate to socioeconomic matters such as employment, and the role within the family or wider society (Charmaz, 1994, Charmaz, 1997). For those who are in employment, returning to work may be delayed due to the occurrence of depression (Fukuoka et al., 2009).

Age was considered for inclusion in the regression model. However, age is one of the attributes included in the instruments chosen to measure comorbidity (the CCI).
and severity of STEMI (the GRACE score). It was therefore decided to exclude age as an individual variable to avoid repeating information and any possible instability of the model due to collinearity (see 5.23.1).

3.5.4.4 Gender
Underlying gender differences exist in the STEMI population: men are twice as likely to suffer a STEMI than women, yet women are more likely to die from the infarct (Shaw et al., 2006, American Heart Association, 2007, Rothwell et al., 2005). Women also tend to be older, have more comorbid conditions and present later than men when they experience a STEMI (Rothwell et al., 2005, Thompson et al., 2006). It has also been reported that women suffer decreased psychological health and poorer physical outcome than men at one and 12 months following PPCI (Mortensen et al., 2007). This may be due to a higher prevalence of depression in the general female population and additionally both anxiety and depression occur more frequently in women following MI (Yuval et al., 2007, Welin et al., 2000, Tighe, 2006).

Furthermore, women present with more ‘atypical’ IHD symptoms and are less likely to suffer chest pain during an ACS event than men (DeVon et al., 2011, Canto et al., 2009). Public health and patient information regarding ‘typical’ IHD symptoms are generally based on the reference standard which, has been developed through mainly male cohorts during early CHD studies (Emslie, 2005, DeVon et al., 2011). It is therefore likely that women interpret their symptoms in accordance with the reference standard for ‘typical’ symptoms and this may lead to inappropriate re-presentation for women (Moser et al., 2006).

Due to the inherent differences in the STEMI population related to sex, the author chose to include gender in the regression model. This was to ascertain whether an association was present between gender and re-presentation.

3.6 Variables included in the regression model
The choice of factors for inclusion in the regression model can be seen in Figure 3-3. The justification for including these attributes in the regression model can be found in section 3.5. Section 5.23.1, describes the final logistic regression model that was devised for pragmatic reasons.
3.7 Summary

The conceptual model that underpinned this study is described in this chapter of the thesis. The factors that the author (in discussion with the supervisory team) believed were most important and directly connected with re-presentation included symptoms, psychological health and physiological health. Sociodemographic factors were also considered to be indirectly related to re-presentation.

The conceptual model was expanded to consider 15 factors that made up the four broad categories (symptoms, psychological health, physiological health and sociodemographic factors) to form the model. The inter-relationships between the factors and the relationship they each held with re-presentation were explored. Furthermore, the decisions made by the author to include certain factors in the regression model are discussed. The final factors chosen for inclusion in the regression model were symptoms (angina frequency, angina stability and physical limitation), anxiety and depression, severity of STEMI, comorbidity, previous IHD event, further re-vascularisation, gender and social support (Figure 3-3).
Figure 3-3 variables included in the regression model

Potential IHD Symptoms
- Angina Frequency at baseline
- Angina Stability at baseline
- Physical limitation (due to angina symptoms) at baseline

Psychological health factors
- Anxiety
- Depression

Physiological health factors
- Co-Morbidity
- History of previous IHD events
- Severity of STEMI
- Further revascularisation at 6 months

Sociodemographic factors
- Social Support
- Gender

Re-presentation with potential IHD symptoms
CHAPTER 4 MIXED METHODS

4.1 Introduction

The following chapter presents the mixed methods section of this study. The rationale for using a mixed methods methodology will be presented; in addition other study designs that were considered will be discussed.

This study was part of a larger study that set out to compare the treatment received by STEMI patients, i.e. thrombolytic therapy and PPCI. The PPCI group was the focus for the current study and the re-presentation and angina symptoms data were all unique data-sets for the study presented in this thesis.

4.2 Study purpose

The purpose of this study was to investigate acute re-presentation due to potential IHD symptoms of patients within 6 months of STEMI and PPCI treatment. The factors (e.g. symptoms, psychological and physiological) associated with re-presentation were explored from the patients’ perspective. See section 3.2.1 for the definition of re-presentation related to this study.

4.3 Study question

What proportion of patients re-presented and what were the factors (e.g. symptoms, psychological and physiological) associated with re-presentation within 6 months of STEMI and PPCI treatment?

4.4 Study design

This study was an explanatory mixed methods study, including quantitative and qualitative methods designed to address an issue that became apparent through clinical practice. The issue under investigation related to the unknown proportion of patients who re-presented to acute healthcare services following STEMI (and PPCI) due to potential IHD symptoms; see section 2.14 relating to the justification for a study.

The quantitative methods included an exploratory prospective cohort study. The quantitative element of the study led to the purposeful selection of participants for the qualitative study, which involved adapted grounded theory.
A number of possible quantitative and mixed methods study designs were considered to answer the study question. A purely qualitative design was not considered as it was not possible to ascertain the proportion of individuals representing with potential IHD symptoms through a qualitative study.

4.5 Rationale for using mixed methods

The method chosen to conduct this study was mixed methods, combining both quantitative and qualitative data collection.

Quantitative research is based on positivism (the dominant paradigm of enquiry), whereas qualitative research is based on a naturalistic approach (Charmaz, 2006). A quantitative study sets out to understand concepts by using quantifiable and statistical techniques that allow generalisation across a wider population. However, the quantitative aspects of a study do not take into account the social aspects of a situation or phenomena that may be of equal importance to the quantitative results (Cronbach 1975). A qualitative element to a study is useful in exploring new concepts such as re-presentation (due to potential IHD symptoms) post STEMI and PPCI treatment. The method enables in-depth exploration of the factors associated with a phenomena from the patients’ own unique perspective (Corbin and Strauss, 2008).

The justification for combining quantitative and qualitative methods in this study was to gain a better understanding of the phenomena under investigation (re-presentation). The use of both methods enabled a more robust analysis as neither method individually was adequate in answering the study question (Tashakkori and Teddlie, 1998, Miles and Huberman, 1994). Mixed methods research is defined by Greene et al (1989) as including at least one quantitative method designed to collect numerical data and one qualitative method designed to collect narrative (or words) (Greene et al., 1989). Polit and Hungler (1999) suggest that mixed methods research can aid the understanding of relationships and causal processes. Rossman and Wilson (1985) further indicate three distinct advantages of conducting mixed methods studies:

- Corroboration of study findings through convergence.
- Elaboration of findings by offering additional richness and depth to conclusions.
- Initiation, leading to new explanations and additional areas for future research. (Rossman and Wilson, 1985, pages 633-637).
4.6 Mixed methods designs

Numerous mixed methods research design classifications have been proposed and used by past researchers (Greene et al., 1989). Many of these designs are similar in what they achieve but are labelled differently by varying researchers in the field (Green and Caracelli, 1997, Morgan, 2007, Patton, 1990, Tashakkori and Teddlie, 1998).

For the purposes of this study the terminology defined by Cresswell and Plano Clarke (2007) will be used to describe the design of the current study. The authors suggest that there are three explicit factors that should be considered when determining the most appropriate mixed methods study design. The stages include the timing of the quantitative and qualitative parts, the study weighting and the merging (or embedding) of the data or study findings.

The timing or sequencing of the quantitative and qualitative stages refers to the order in which the data are used in the study; it may be concurrent (quantitative and qualitative at the same time) or sequential (quantitative leading to qualitative or vice versa) (Creswell and Plano Clarke, 2007). The weighting involves deciding whether one method (i.e. quantitative or qualitative) should play a greater role. It also indicates whether the quantitative or qualitative phase is of more importance or of equal standing in addressing the research questions (Morse, 1991). The mixing of quantitative and qualitative methods can include merging, embedding or connecting of methods (Creswell and Plano Clarke, 2007).

Four main models of mixed methods designs are described by Creswell and Plano Clarke (2007) including triangulation, embedded design, explanatory design and exploratory design. All four models incorporate the timing, weighting and mixing of methods and can be seen in Table 4-1.
<table>
<thead>
<tr>
<th>Type of model</th>
<th>Timing</th>
<th>Purpose</th>
<th>Weighting</th>
<th>Mixing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triangulation</td>
<td>Concurrent-quantitative and qualitative</td>
<td>To collect different but complimentary data on the same topic.</td>
<td>Equal</td>
<td>Merging data during analysis or interpretation phase.</td>
</tr>
<tr>
<td>Embedded</td>
<td>Concurrent or sequential</td>
<td>One data type is the main basis for the study. The other data type is supplementary.</td>
<td>Uneven-quantitative or qualitative</td>
<td>Mixed at the design level (e.g. quantitative data could be embedded in a qualitative study).</td>
</tr>
<tr>
<td>Explanatory</td>
<td>Sequential-two phases, qualitative then qualitative</td>
<td>Two phases-qualitative phase adds depth to or explains quantitative results.</td>
<td>Typically quantitative</td>
<td>Connect data at analysis or interpretation stage.</td>
</tr>
<tr>
<td>Exploratory</td>
<td>Sequential-two phases, qualitative then quantitative</td>
<td>Qualitative results may be an initial exploration and quantitative data adds additional information.</td>
<td>Typically qualitative</td>
<td>Connect data at analysis or interpretation stage.</td>
</tr>
</tbody>
</table>

Cresswell and Plano Clarke (2007)

### 4.7 Explanatory mixed methods design

An explanatory mixed methods model with emphasis on the quantitative results with connection of the quantitative and qualitative study findings was conducted (see Figure 4-1). This was to investigate re-presentation (due to potential IHD symptoms) to acute services, for STEMI patients within 6 months of PPCI with the use of quantitative methods. The participants’ experiences of re-presentation were also explored using qualitative methods.

The explanatory method was chosen because the quantitative study determined the participants who re-presented with potential IHD symptoms (phase 1 of the mixed methods design – see Figure 4-1). Two groups were therefore identified 1) re-presentation group 2) non-representation group. From the re-presentation group, participants were purposefully selected for inclusion in qualitative interviews. The quantitative study led to identification of those who re-presented and the diagnostic causes of re-presentation. It also set out to investigate the relationship between psychological health (anxiety and depression), angina symptoms and re-presentation within 6 months of STEMI with the use of self-report questionnaires.
Furthermore, with a large enough sample size there was the potential to use the numerical data to generalise to the wider population (see section 5.7.3).

The qualitative phase of the study (phase 2) was a pragmatic means of accessing the views of participants who had re-presented to acute services. The participants experiences of symptoms and re-presentation were elicited through interviews to gain additional insight and add explanation of the quantitative results (Creswell et al., 2003).

Convergence of the analysis (phase 3) from each method helped to inform interpretation of the findings, leading to greater depth of understanding of the factors and relationships of re-presentation, psychological health and angina symptom occurrence (Johnson and Onwuegbuzie, 2004). The use of explanatory mixed methods in this study, by combining quantitative and qualitative research, produced a more complete understanding of the data as a whole (Creswell and Plano Clarke, 2007).
Figure 4-1 The explanatory mixed methods model for the current study

Phase 1
Quantitative

Baseline

Recruitment
Recruit STEMI PPCI patients

Data Collection
Self Report instruments
Descriptive data

6 Months

Data Collection
Self Report instruments
Potential IHD events

Identify re-presenters

Analysis
Quantitative Data

Phase 2
Qualitative

6 Months

Selection
Purposefully identify participants

Interviewing

Analysis
Quantitative Data

Phase 3
Combining

Connecting Data

Interpretation
4.7.1 Advantages and disadvantages of explanatory mixed methods

One of the advantages of the explanatory mixed methods model is that it is good for multiphase research (Ivankova et al., 2006). The design is one of the more straightforward of the mixed methods due to the two distinct phases. The simple study design was of particularly benefit for the current academic study, as it was achievable for a single researcher to run the study rather than a team of researchers. For ease of reading it was also possible to present the final study report in two separate sections (quantitative and qualitative) and merge the findings during the discussion section (Ivankova et al., 2006, Creswell and Plano Clarke, 2007). In the case of the current study it was advantageous in identifying participants for the qualitative phase. Individuals for the qualitative study were then purposefully selected (May and Etkina, 2002).

A disadvantage of the explanatory model relates to the two sequential phases which may extend the time taken to complete data collection particularly when compared to concurrent data collection techniques. A possible disadvantage of all mixed methods research is the potential for conflicting quantitative and qualitative results (Creswell, 2003, Creswell and Plano Clarke, 2007). Creswell (2003) suggests the collection of further data to resolve the issue should it occur, yet other researchers suggest purely using the results as a lead for future research (Padgett, 2004, Bryman, 1988). For the purposes of this research it was decided to use any conflicting results as a basis for future recommendations for research.

4.8 Quantitative method

The quantitative design of this study was an exploratory prospective cohort study. Full description of the quantitative study design, methods and plan of analysis can be found in Chapter 5.

4.8.1 Prospective cohort study

A prospective cohort study design involves investigating a current phenomena by collecting information as time progresses (Parahoo, 1997). It may also look to determine a relationship that is an association or one that is causal (Euser et al., 2009). The term ‘cohort’ is described as “the same group of respondents who are followed over a period of time” (Parahoo, 1997, page 158). In this study the study cohort was STEMI patients who had received PPCI treatment.
The strengths of the prospective cohort design include the collecting of data prior to an event (e.g. re-presentation) allowing the rates of an event to be determined (Ernster, 1994). The time sequence for cause and effect may also be determined (Ernster, 1994). The accuracy of data is more certain than in the case of retrospective data collection because data are collected for the purposes of the research. The disadvantages of this type of study are that it is costly and time consuming.

The prospective design for this study was appropriate because it provided precise, numerical data in relation to the number of patients who re-presented to acute services. It enabled the causes and associated factors of re-presentation within 6 months of STEMI and PPCI, to be investigated. For these purposes, re-presentation data, clinical and treatment factors, co-morbidity and self-report questionnaire data were obtained at baseline and 6 months; comparisons were undertaken between the re-presentation and non-representation groups.

The levels of anxiety, depression and angina symptoms were determined at baseline and 6 months for the re-presentation and non-re-presentation groups. Previous prospective studies have established that changes occur in psychological health (in particular depression) during the recovery period for STEMI patients (Frasure-Smith et al., 1995a, Dickens et al., 2006). Comparison of psychological health measures over the course of 6 months, enabled assessment of change for the re-presentation and non-re-presentation groups (see study aims 2, 3, 4 and 5 in section 5.4). This has not previously been established for STEMI patients receiving PPCI treatment. Furthermore, the collection of longitudinal angina symptom data alongside psychological data enables the detection of any correlation between psychological health and angina symptoms (Frasure-Smith et al., 1993).

Six months was identified as a follow-up time point as other researcher have previously demonstrated 6 months as an appropriate time to assess anxiety and depression following an MI (Dickens et al., 2004, Frasure-Smith et al., 1993). Furthermore, 6 month follow-up was also adopted for pragmatic reasons, because the study was an academic piece of work.

**4.8.2 Exploratory study**

It was decided to undertake an exploratory study as STEMI patients treated with PPCI are a relatively under-investigated group in terms of re-presentation, psychological health and angina symptom occurrence. The number of patients re-presentation due to potential IHD events (both cardiac and non-cardiac cause) has
not previously been established and therefore this was one of the current study aims. Thus, the relationship between re-presentation, and anxiety, depression, psychological distress (psychological health) and angina symptoms has not previously been addressed for this group. As the incidence of re-presentation is not currently defined it was not possible to establish an accurate sample size to base the assessment of relationship and therefore an exploratory study was the most appropriate design. The quantitative phase of the study is discussed in more detail in section 4.8.4.

4.8.3 Exploratory prospective cohort study

An exploratory prospective cohort study conducted singly and not as a mixed methods study (i.e. without the addition of the qualitative study) would have led to important but somewhat superficial information relating to re-presentation (Ivankova et al., 2006, Miles and Huberman, 1994). As the re-presentation (due to potential IHD events) is an under investigated area it was decided that more in-depth information from the patients perspective was of benefit in understanding the phenomena. It was therefore decided to undertake a mixed methods study including the prospective cohort design as part of the quantitative phase of the study.

4.8.4 Quantitative data collection methods

The possible data collection methods that were considered for this study included postal questionnaires and psychiatric interviews.

4.8.4.1 Questionnaires

A survey methodology in the form of postal questionnaires was chosen to collect psychological health and angina symptom data (see section 5.8 and 5.10). An advantage of postal questionnaires is that sensitive data can be collected from large numbers of participants (Siemiatycki, 1979). Participants can also complete questionnaires within their own timescale and in their own homes. Self-report questionnaires related to psychological and angina symptom data collection have been used and validated in CHD patients previously and are an accepted means of data collection (Arnold et al., 2009, Herrmann, 1997). One disadvantage of this method is that participants may choose not to return the questionnaire leading to reduced response rates and the potential for a biased sample. Participants may also miss some items on the instrument leading to missing data (Clarke-Carter, 1997). In an attempt to address some of these issues a telephone interview was conducted within one week of posting the questionnaires. This was to offer participants
assistance in completing questionnaires (if required) and act as a prompt for those who had not already returned them. When missing items on instruments were identified, participants were telephoned to obtain their responses. Details of the questionnaires used in this study are discussed in detail in section 5.10.

4.8.4.2 Telephone interviews
Baseline telephone interviews were conducted as a means of data collection for demographic, co-morbidity and assessment of clinical parameters (see section 5.8.1). At 6 months telephone interviews were performed to collect re-presentation data. Telephone interviews are known to be advantageous in obtaining more complete information than survey methods (Brägger et al., 2002). Re-presentation data were also collected by checking the participants’ hospital records and the interviews were useful in ascertaining whether individuals had re-presented to a hospital other than their local one. A disadvantage of collecting data by telephone interview is that the participant may not remember events or dates associated with them correctly and therefore data may not be reliable (Siemiatycki, 1979). For these purposes a diary was given to participants to record re-presentation events; the participant’s GP was also contacted when additional corroboration of data was required.

4.8.5 Alternative quantitative methods considered
Several other quantitative methods to conduct this study were considered, including case-control and a retrospective study. A summary of the rationale for excluding these methods are presented.

4.8.5.1 Retrospective cohort study
A retrospective study was considered to establish the proportion of re-presentations over a set period of time. The factors relating to the re-presentations and the psychological and physiological health of the participant would have been reviewed through the participants’ hospital records.

A retrospective study sets out to explain current phenomena through associated factors gained from past information (Parahoo, 1997). The main advantage of a retrospective study is that there is little burden on the research participant, as data are generally collected from their records. This results in little or no attrition of the research sample over the course of the study unlike longitudinal studies (Santos-Eggimann et al., 1997). This method is also cheaper to run than prospective studies (Santos-Eggimann et al., 1997).
Retrospective data collection, by nature has implicit problems with accuracy and validation of data (Schulz and Grimes, 2002). In the case of this study the patients’ hospital records would have been required to collect demographic, physiological and psychological data. Hospital records may not have contained the relevant information, particularly relating to the patients’ psychological health. This may have led to either incomplete or missing research data (Parahoo, 1997, Bernard et al., 1984). Furthermore, the study design does not allow the collection of specific data related to psychological (anxiety and depression) and physiological (angina symptoms) health, experienced by participants at different time points unlike a longitudinal design (Ernster, 1994).

4.8.5.2 Case-control

A case-control study design was considered. This type of study sets out to compare the characteristics of a group of individuals of interest (case group) with a group who come from the same population (control group); the groups are defined by the outcome (Schulz and Grimes, 2002, Bowling, 2002). A case-control study may be prospective, although more commonly they are retrospective in design (Bowling, 2002).

The benefits of a case-control study are that cause and effect can be determined through investigating the factors that are related to the condition under investigation (Bowling, 2002). The frequency of the occurring factors can be compared between the case group and control group (Schulz and Grimes, 2002). It is helpful in comparing existing ‘risk factors’ between the groups in prospective designs or their past experiences or ‘exposures’ through a retrospective study (Bowling, 2002).

A disadvantage of case-control studies is that they are more susceptible to bias than cohort studies. The validity of the case-control study can be easily affected by methodological issues such as selection bias, particularly with retrospective data collection and a potential bias in the data collected (Ernster, 1994, Johnston, 2002). It can also be challenging to match controls with cases and can be time consuming.

For the purposes of the current study the case and control groups would have been determined from the population (i.e. patients who had experienced a STEMI and PPCI treatment within the previous 6 months). The case group would have been those who re-presented (with potential IHD symptoms) and the control group, those who did not re-present.
A case-control study would have been useful in determining the factors related to re-presentation in this study. However, Bowling (2002) suggests that estimating an ‘exposure’ (re-presentation) in ‘cases’ compared to ‘controls’ is more accurately achieved through longitudinal surveys. Furthermore, in the case of this study matching individuals who re-presented with controls would have been very time consuming. It was therefore decided not to further pursue a case-control study design.

4.8.6 Alternative data collection method considered

4.8.6.1 Psychiatric interviews

An alternative method of capturing psychological health data would have been by performing an in-depth face to face psychiatric interview. The advantage of psychiatric interviews is that a psychiatric diagnosis can be determined (Regier et al., 1998). Conversely with self-report questionnaires such as the Hospital Anxiety and Depression Scale (HADS), when scores are raised further clinical assessment is required to confirm a diagnosis of psychological distress (Herrmann, 1997).

The number of individuals who can be included using psychiatric interviews, is limited when a single researcher is capturing the data due to the time consuming nature of the interviews (de Jonge et al., 2006, Dickens et al., 2004). In addition, researchers need specific training in the administration and interpretation of these clinical style interviews, and the associated diagnostic taxonomy used (e.g. ICD; DSM manuals) Questionnaires are also less intrusive and time consuming for participants and they allow individuals to give less socially accepted responses than interviews (Parahoo, 1997). Individuals may also choose not to reply to postal questionnaires therefore giving them an opportunity to opt out of the study should they wish to do so.

4.9 Reliability and validity

The quality of the quantitative phase of the study was addressed by assessing the reliability and validity of appropriate data collection methods and instruments. The underlying purpose of a quantitative study is to be able to draw meaningful inferences from results to a chosen population (Brooks, 1996).

Reliability is when a method consistently measures a phenomenon (Parahoo, 1997). Validity is when a method measures what it sets out to measure and has been repeatedly tested within the population (Parahoo, 1997, Bowling, 2002). Details of
reliability and validity of the instruments and justification for their inclusion in the study can be found in sections 4.9.1, 4.9.2 and 5.9.

4.9.1 Reliability

Reliability for the current study was assessed in a number of ways. The appropriateness of the variables included in the study was appraised and justified (see section 3.2.1 and 5.8). Furthermore, the reliability of the questionnaires chosen for inclusion in the study was also judged (section 5.10).

In the case of questionnaires, reliability relates to how consistently participants interpret and respond to the questions (Parahoo, 1997). It is possible for a questionnaire to be reliable but not valid (Coolican, 2004). The specific reliability assessment ‘internal consistency’ of measures was used in the current study.

Internal consistency refers to whether the items of a test are all consistent with each other. An estimate of internal consistency is Cronbach’s alpha, which is based on correlations between the items within the scales (Bowling, 2002). There is no agreed minimum level of internal consistency, but the higher the alpha the more correlated the items are: Cronbach (1951) accepts >0.50, whereas Nunnally (1978) considers 0.70 as an acceptable reliability coefficient. Wherever possible internal consistency was presented using Cronbach’s alpha for the instruments used in this study (see section 5.10).

4.9.2 Validity

Two main types of validity apply to study findings, internal and external validity (Parahoo, 1997, Bowling, 2002). Furthermore, construct validity and sensitivity and specificity are also used to assess the quality of instruments (Bowling, 2002, page 152).

Internal validity is described by Parahoo (1997) as, “the extent to which changes, if any, in the dependant variable can be said to have been caused by the independent variable alone” (page 196). It relates to how confident the researcher is that conclusions can be drawn from the study findings and to what extent flaws in the research design may have biased the study results (Buckwalter et al., 1998). Threats to the internal validity of a study may occur due to bias or confounding factors.

At any stage of a study bias may occur; this can include aspects of participant selection and attrition, measurement bias, as well as interpretation and reporting of
study results (Tashakkori and Teddlie, 1998, Parahoo, 1997). Confounding factors are those that may have positively or negatively influenced the study outcome and may not have been controlled for in the study design or analysis (Bowling, 2002, Parahoo, 1997). For example the introduction of psychological interventions as part of the clinical care of anxious and depressed STEMI patients, may influence the outcome of a study investigating the levels of anxiety and depression in post STEMI patients. Authors purport the importance of recognising and implementing strategies to reduce threats to internal validity when conducting a study (Parahoo, 1997, Bowling, 2002, Buckwalter et al., 1998).

External validity refers to the extent that study results can be generalized to the wider population (Bowling, 2002, Bell, 1995). A number of factors can influence external validity the most important being sampling (Bell, 1995). Although the current study was of an exploratory design, the internal and external validity of the study was assessed to establish whether the quantitative results could be generalised to the wider STEMI population.

The risks to internal and external validity and how they were addressed in this study are described in Table 4-2 and Table 4-3 respectively.
Table 4-2 Potential threats to internal validity and strategies to reduce threats

<table>
<thead>
<tr>
<th>Potential threats to internal validity</th>
<th>Potential threat in this study</th>
<th>Actions taken</th>
</tr>
</thead>
</table>
| Participants (i.e. sampling, attrition, responder variance, small sample in representation group) | **Sampling bias:** Small sample size and poor sampling methods may lead to a biased sample (unrepresentative of the target population) and jeopardise the validity of the findings. | • Although the study was exploratory to address potential threats to internal validity a sample size for the study was conducted, and was based on the predicted number of participants to re-present with potential IHD events (see section 5.7.3).  
• Consecutive sampling at more than one PPCI centre, ensuring a mixed representative cohort. This enabled a wide spread of patients from different geographical and socioeconomic areas across Manchester (see section 5.6 for further details of participating centres).  
• All eligible patients were identified, approached and recruited wherever possible at all centres.  
• The identification of potential participants was undertaken by regularly checking the catheter lab and MINAP databases at both PPCI centres and additionally regularly contacting the coronary care units (CCU) at all participating hospitals.  
• To improve the recruitment rate wherever possible patients were approached whilst an in-patient. Patients who were missed during their in-patient admission were approached by letter. A follow-up telephone call was then made to establish whether they wished to participate.  
• Details of the sample, recruitment rate and demographics of the sample are presented descriptively for others to assess the overall study cohort (see section 7.2). |
<table>
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<tr>
<th>Potential threats to internal validity</th>
<th>Potential threat in this study</th>
<th>Actions taken</th>
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</table>
| **Attrition bias:**                  | Participant attrition (i.e. poor response rate at baseline and high dropout rate at 6 months). | - Once baseline and 6 month questionnaires were mailed if patients did not return their questionnaires within 7 days a follow-up telephone call was made to prompt the return of questionnaires. Additionally at 6 months re-presentation data was collected during the telephone call.  
- The telephone call was also an opportunity to maintain a relationship with the participants and to offer participants assistance in completing their questionnaires.  
- Assessment of the response rate at baseline and 6 months was made to assess the dropout rate (see section 7.2.4). |
<p>| <strong>Responder bias:</strong>                  | Responders may be different to the non-responders leading to a biased sample (response bias). <em>Responders and non-responders refer to those who gave consent and returned questionnaires and those who gave consent but did not return questionnaires respectively.</em> | - Comparison of responder and non-responder characteristics, to establish if there were any major differences between those who did and did not participated was undertaken (see 7.2.2). |
| <strong>Data collection</strong>                  | <strong>Measurement bias:</strong> The instrument does not measure what is required. | - Study instruments the HADS and SAQ were reviewed for validity in the STEMI population. This included review of prior studies which have incorporated these instruments in STEMI samples (see section 5.10). |</p>
<table>
<thead>
<tr>
<th>Potential threats to internal validity</th>
<th>Potential threat in this study</th>
<th>Actions taken</th>
</tr>
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</table>
| Recall bias: Participants may not have remembered re-presentation events accurately. | - Multiple methods of collecting re-presentation data were used.  
- Telephone interviews.  
- A diary was provided for participants to complete at the time of a re-presentation event.  
- Hospital records were also checked for re-presentation events at both the participants’ local district general hospital and the PPCI centre.  
- General practitioners were also contacted in some instances to verify re-presentation events for some participants when details of events were unclear. |
Table 4-3 Potential threats to external validity and strategies to reduce threats

<table>
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<tr>
<th>Potential threats to external validity</th>
<th>Potential threat in this study</th>
<th>Actions taken</th>
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</table>
| **Participants** (representative of the general PPCI population) | Exclusion criteria: diagnosis of severe cognitive impairment, severe physical or mental illness (at baseline) limiting participation in the study. | • Where ever possible participants with severe physical or psychological illness were reviewed on a number of occasions during index (due to STEMI) admission, to determine if their condition had improved, enabling participation.  
• The exclusion criteria used in this study was kept to a minimum to allow the inclusion of PPCI cohort that was more representative of the general PPCI population. |
| **Confounding factors** (psychological distress prior to STEMI, timing of the study) | Participants may have been suffering psychological distress prior to STEMI and recruitment to the study. | • It was not possible to establish if participants were already suffering anxiety and depression prior to STEMI through validated instruments. However, participants were asked during the baseline telephone interview if they had ever received treatment for anxiety and/or depression. |
|                                                                 | The timing of the study may influence the study cohort due to changes in clinical and treatment practices. | • Comparison and presentation of the current study cohort with those of other similar STEMI studies to determine whether the study cohort is similar to other representative cohorts (of the general PPCI population) (see section 9.5). |
4.10 Qualitative method

A modified version of grounded theory using relevant aspects of the approach for this study (Charmaz, 2006), was used to explore participant experiences of re-presentation to acute services. Data was collected during an in-depth semi-structured interview on one occasion, and data were analysed using Framework analysis (Ritchie et al., 2003).

The qualitative phase of the mixed methods study is discussed briefly in this chapter. A more detailed explanation of the qualitative study for this study are discussed in Chapter 6. A number of potential qualitative methods were explored during the design phase and are discussed in this section.

4.10.1 Modified grounded theory

Grounded theory was first introduced by Glaser and Strauss (1967) with the purpose of developing theory from data (Corbin and Strauss, 2008). The method is underpinned by a naturalistic world view that enables the researcher to become absorbed and routed within the rich data that is compiled from the social setting under investigation (Charmaz, 2006). Full grounded theory includes following certain processes such as theoretical sampling, concurrent data collection and analysis, and constant comparative analytical techniques (Glaser and Strauss, 1967). Details of the concepts underpinning grounded theory can be seen in section 4.11.2.1 and Table 4-5.

During development of grounded theory Glazer and Strauss (1967) suggested that researchers use the grounded theory method in a flexible manner to meet their research needs. Charmaz (2006) proposes that grounded theory is a set of principles and practices that may be used as a set of guidelines for the conduct of a qualitative study:

"the method (grounded theory) can complement other approaches to qualitative data analysis, rather than stand in opposition to them" (page 9).

Due to the applied nature of this study and the ‘explanatory’ mixed methods design (see section 4.7), it was not appropriate to follow the grounded theory design per se (Creswell and Plano Clarke, 2007). Justification for discounting a ‘pure’ version of grounded theory for this study can be found in section 4.11.2.1. However, the use of elements of grounded theory enabled exploration of new and emerging concepts and offered insight of participant experiences relating to re-presentation
to acute healthcare services (Morse, 2003). Adopting threads of grounded theory is helpful when seeking to understand a relatively unknown cohort such as PPCI patients, because the method is an adaptive, flexible and exploratory means of enquiry (Charmaz, 2006). A further advantage of using modified grounded theory for this study was that a structured means of data analysis Framework analysis could be incorporated to analyze and interpret the study findings (section 4.11.1) (Ritchie and Spencer, 1994). Table 4-4 shows the main concepts adopted to conduct the qualitative aspects of the study.
Table 4-4 Concepts used to conduct the qualitative part of the study

<table>
<thead>
<tr>
<th>Method used in current study</th>
<th>Details of method used</th>
</tr>
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<tbody>
<tr>
<td>Semi structured Interviews.</td>
<td>An interview schedule devised from the literature and clinical practice was initially used to guide data collection. Subsequently the interview schedules were adapted based on the emerging concepts during data analysis (Kvale, 1996). The characteristics of the interviewees and the contextual information relating to the interviews, was recorded to add depth and richness to the data collected. Further details of the interview method can be found in section 4.10.2.1 and Chapter 6.</td>
</tr>
<tr>
<td>Purposeful sampling.</td>
<td>Purposeful sampling in the form of 'maximum variation' sampling was conducted to access the greatest variance and diversity of participants (Patton, 1990). Initially individuals who re-presented to acute healthcare services and were able and willing to provide rich data on the phenomenon were selected. Subsequently, participants were selected based on a spread of other characteristics and factors of interest to achieve maximum variation of areas relating to the study questions (Hoepfl, 1997). Individuals who could give insight into new areas of interest that were developing were also chosen. Further details of the sampling strategy are described in (section 6.2.4).</td>
</tr>
<tr>
<td>Concurrent sampling, data collection and data analysis.</td>
<td>Aspects of grounded theory used included concurrent sampling, data collection and data analysis (Glaser and Strauss, 1967). These were adopted to explore and develop concepts rooted in the data. The flexibility and dynamic nature of the concurrent data collection and analytical processes allowed 'ideas', 'new leads' and 'hunches' to be followed up (Charmaz, 2006). Furthermore, concurrent sampling was used in part to facilitate maximum variation sampling. This was because it was difficult to indicate a priori what the variation of participants was likely to be until ‘the field’ was accessed, due to the new phenomenon under investigation. Data collection ended once no new information was emerging from the interviews and categories became saturated (Streubert and Carpenter, 1999).</td>
</tr>
<tr>
<td>Method used in current study</td>
<td>Details of method used</td>
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<tr>
<td>Constant comparative data analysis.</td>
<td>A component of grounded theory, constant comparison, was used to develop codes and concepts directly from data (Corbin and Strauss, 2008). Constant comparison with the use of induction enabled the similarities and differences between events, emotions, behaviours and participant characteristics to be acknowledged (Charmaz, 2006). Use of this technique enabled both description and abstraction of the factors involved with the phenomena, leading to new insight of the experiences and perspectives of the participant group (Pope et al., 2000).</td>
</tr>
<tr>
<td>Framework analysis.</td>
<td>Framework analysis is a structured approached that is used in applied policy and health services research to move through the stages of analysis whilst keeping sight of the original data (Ritchie et al., 2003). The systematic method enables others to view the steps that have been taken during analysis adding to the rigour or auditability of the study (Yonge and Stewin, 1988, Mason, 1994). Framework analysis enables the use of both deduction and induction during the 5 stages of analysis (Ritchie and Spencer, 1994). This lends itself to exploration, detection, definition, explanation and theorising of the concepts related to the phenomenon under investigation. Framework analysis is discussed further in section 4.11.1 and 6.3.</td>
</tr>
<tr>
<td>Maintaining a research journal.</td>
<td>A journal was used during the qualitative study to record the processes and decisions made during sampling, data collection and data analysis. The journal was used by the researcher to record thinking, and the judgments made during the development of concepts. It was also a useful tool in better understanding and defining the relationships between categories (Corbin and Strauss, 2008). Recording the steps undertaken during conceptualisation also acted as an audit trail (Ritchie et al., 2003).</td>
</tr>
<tr>
<td>Reflexivity.</td>
<td>Reflexivity was used at the outset of the study to determine any potential biases of the research, that may have influenced data collection and data analysis, and the overall rigour of the study (Snape and Spencer, 2003). The researcher’s beliefs and potential biases are declared in the rigour section of this chapter in section 4.12.</td>
</tr>
<tr>
<td>Method used in current study</td>
<td>Details of method used</td>
</tr>
<tr>
<td>-----------------------------</td>
<td>------------------------</td>
</tr>
<tr>
<td>Literature review conducted prior to start of the study.</td>
<td>The literature review for this study was conducted during the planning stage of the mixed methods study as a whole. In grounded theory the literature review is conducted at the end of the study to limit the influence that the researchers knowledge gained through the literature has on the study findings (Glaser and Strauss, 1967, Strauss, 1987, Glaser, 1978) (Table 4-5). However, this was not possible for the current study due to the applied nature and the mixed methods design (see section 4.7.1).</td>
</tr>
</tbody>
</table>
4.10.2 Qualitative data collection

Two common data collection methods are often used for qualitative studies, interviews and focus groups. In the qualitative phase of this study semi-structured interviews were used for selected individuals to explore the patients’ own unique perspective of re-presentation. Justification for the use of interviews is discussed below as is the reasons for not adopting focus groups.

4.10.2.1 Interviews

In-depth semi-structured interviews were chosen as the data collection method for this study because the subject matter was considered by the researcher to be a sensitive topic. Re-presentation due to IHD symptoms may have led to strong emotional responses relating to the initial STEMI and the re-presentation event for those who had experienced them. Individuals may have developed fears related to death and dying for instance or concerns regarding their current and future health or the implications for their loved ones. One of the advantages of interviews is the opportunity for the researcher to build rapport with the participant leading to the possibility of discussing sensitive subjects (Kvale, 1996). The disadvantage of conducting interviews is that they are time consuming and therefore relatively expensive.

Participants were given the choice of being interviewed in their home or at the researchers’ place of work (Central Manchester University Hospital Trust). Interviews and analysis were conducted concurrently enabling the researcher to identify new areas for exploration in future interviews. Initially, interviews were based on an a priori interview guide (see Appendix M), which was subsequently adapted to incorporate additional topics that had emerged during prior interviews (see section 6.2.6). Contextual information relating to the interview surroundings and the participant were also documented.

4.10.2.2 Focus groups

An alternative data collection method, focus groups was considered. The advantage of this method is that it is cheaper and quicker, due to the involvement of multiple individuals (usually between seven and nine) during one focus group. However, the practical aspects of the method can lead to difficulties in getting people together at a specific date and time. The need for individuals to attend rather than the researcher visiting them at home (in the case of interviews), may discourage them from participating particularly for those with mobility issues. The logistics surrounding arranging transport for participants and the associated costs may also
make the method problematic. Additionally, in this study sensitive issues may have arisen and participants may not have been willing to discuss them in a group situation. This data collection method was therefore discounted for the current study.

### 4.11 Analysis of qualitative data

Various types of qualitative data analysis are available, including content analysis and thematic analysis. Content analysis involves data being investigated for content and context as well as relationships and categories (Graneheim and Lundman, 2004, Bowling, 1997). It is inductive and themes develop through the recurring description from within the data, leading to trustworthiness.

Thematic analysis is also an inductive method that is commonly associated with grounded theory and is used to report patterns or themes in rich detail (Braun and Clarke, 2006). Thematic analysis differs from content analysis, because it occurs concurrently with sampling (theoretical sampling) and data collection. It also involves constant comparison, which comprises of the analyst asking questions of the data to develop concepts and subsequently theory (Corbin and Strauss, 2008).

The questions that the analyst may ask of the data using constant comparison are for example:

- What led to the behaviour?
- Why and what was happening at that time?
- What (and why) might the participant have been experiencing emotionally?

The themes emerging from the data, through constant comparison, guide subsequent sampling and data collection (Charmaz, 2006, page 42).

The conduct of this study was guided with components of grounded theory, including concurrent data collection and analysis, and also constant comparison (Table 4-4). Furthermore, due to the copious amounts of data that qualitative research generates and to aid the rigour of the study, it was decided to use a structured approach to managing and analysing the data. It was therefore decided to use analysis, which is described in detail in section 4.11.1.

Qualitative analysis has been criticised because of its subjectivity, which may lead to misrepresented findings. However, assessment of its rigour using a range of methods, discussed in section 4.12, may offer reassurance to the researcher’s
4.11.1 Framework analysis

Framework analysis is a structured data analysis method that was first used in applied policy research and has increasingly been used in applied health services research (Ritchie et al., 2003, Swallow et al., 2003, Pope et al., 2000). It enables large amounts of qualitative data, often collected by multiple researchers in a short time scale, to be analysed in an effective manner. More recently it has also been used in smaller scale studies (Furber et al., 2009). Ritchie et al (2003) describe Framework analysis as,

“a matrix based analytical method which facilitates rigorous and transparent data management such that all the stages involved in the ‘analytical hierarchy’ can be systematically conducted” (page 220).

An advantage of Framework analysis is that it is based on a methodical procedure, which may be attractive to researchers and their audiences who are not familiar with interpreting qualitative data (Ritchie et al., 2003). The method is initially based on deduction involving pre-set aims and objectives (Pope et al., 2000). However, analysis is closely linked to the ‘raw’ data and as concepts evolve the analysis often becomes inductive offering the analyst the possibility of exploring abstract concepts.

The analytical process related to Framework is underpinned with five stages that are interconnected. The stages include familiarisation, identifying a thematic framework, indexing, charting, mapping and interpreting; the researcher is able to move between stages throughout the process (Ritchie and Spencer, 1994). Although it is a structured process, Framework still requires the analyst to interpret meaning and use ‘logical and intuitive thinking’ to identify patterns, as well as categorise similarities and differences in the data. Furthermore, it also entails the analyst using their judgment in connecting ideas, theorising and defining concepts.

Framework analysis was chosen to conduct the analysis for this study, partly due to its validated use in applied health services research (Swallow et al., 2003, Finch, 1988). The systematic analytical procedures enabled a clear means of demonstrating the rigour of the study processes undertaken. Furthermore, the flexibility of the method allowed the study aims to be fulfilled through exploration of
initial concepts with the use of deduction and subsequently development of themes through induction.

The five stages of Framework are described in more detail in the following sections (Ritchie and Spencer, 1994). The way that the stages of Framework analysis were applied in practice to this study are discussed in section 6.3.

4.11.1.1 Familiarisation

The intention during the familiarisation stage of analysis was to assess the subject content and the richness and diversity of the interviews. To establish this the audio-recordings and the interview transcripts were repeatedly reviewed by the researcher (Ritchie and Spencer, 1994). Contextual information was also revisited at this stage to ascertain whether certain concepts are more difficult to discuss with participants or whether any specific issues needed to be taken into consideration during analysis (e.g. age, gender or sexuality). Furthermore, "the process of abstraction and conceptualization" was started by documenting 'key concepts' and 'recurring themes' that came to the fore during review of the data (Ritchie and Spencer, 1994, page 178).

4.11.1.2 Identifying a thematic framework

The original concepts identified a priori and included in the interview schedule were used to develop a thematic framework (Ritchie and Spencer, 1994). The key concepts and recurrent themes identified during familiarisation were also incorporated in the thematic framework.

4.11.1.3 Indexing

The thematic framework was applied to the subsequent transcripts in a systematic manner. The thematic framework evolved during this phase as new concepts emerged and judgments were made regarding the meaning and significance of the data (Ritchie and Spencer, 1994). Inferences were made of the data using deductive reasoning (Charmaz, 2006). When new concepts emerged, induction was used to elicit meaning and new codes (or indexes) were developed and added to the thematic framework. A journal was kept during the coding (or indexing) of the data to record the decisions made relating to inductive interpretation and the inferences made. The findings from the coding (or indexing) of interviews was then used to select the next interviewee and to adapt the interview schedule (see 6.2.4). Due to the subjective nature of this phase of the analysis the decisions made by the
analyst were reviewed and précised by the researcher’s supervisory team (Pope and Mays, 1996).

4.11.1.4 Charting
Once indexing was complete, data were rearranged into charts that catalogued the data according to the themes and the sub-themes. This allowed the researcher to view the data as a whole across all participants for each theme and allowed cross referencing between participants. Each participant (or ‘case’) was kept in the same order on every chart for each theme and sub-theme (Ritchie and Spencer, 1994). Data were summarized on the charts sometimes with the use of full quotes and on other occasions several words were used to represent the nature of the meaning. In each respect, the original source data was referred to for cases with the use of page and line numbers; this was to allow the analysis to be routed in the data and for ease of auditing.

4.11.1.5 Mapping and interpreting
The mapping and interpretation stage involved the analyst combining the main characteristics of the data across all cases and all themes. During previous phases of the analytical process patterns, associations and emergent categories within themes were documented. The mapping and interpretation stage goes much further, and Richie and Spencer (1994, page 186) suggest that this is the most difficult stage to encapsulate; they state that “each step requires leaps of intuition and imagination”. They also describe the core objectives and features of qualitative analysis that should be encompassed in this stage of the analysis (Ritchie and Spencer, 1994, page 186).

- Mapping range and nature of phenomena
- Creating typologies
- Finding associations
- Providing explanations
- Developing strategies

One of the main purposes of this stage was to define concepts, by systematically examining the charts and identifying the major elements and themes across all cases. Furthermore, this phase enabled mapping of the “range and nature of phenomena” with the opportunity to identify the extremes of a phenomena as well as the middle ground (Ritchie and Spencer, 1994, page 186). It was also possible
during this stage to unearth and explain emotional constructs and the attitudes and behaviours of participants.

4.11.2 Alternative qualitative methods

The alternative methods of conducting the qualitative study that were considered included full grounded theory and phenomenology.

4.11.2.1 Grounded theory

Grounded theory was considered for the current study because it is a useful method to explore new areas of investigation like PPCI (Bowling, 1997). An advantage of the methods is that areas where little is known about a phenomenon or the lived experiences of individuals can be explored to uncover new emergent theories and findings are routed within the data (Snape and Spencer, 2003). The main concepts of grounded theory are underpinned with a particular set of procedures described in Table 4-5 (Glaser and Strauss, 1967, Strauss, 1987, Glaser, 1978, Charmaz, 2006).

<table>
<thead>
<tr>
<th>Grounded theory concept</th>
<th>Purpose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Theory developed at all stages of data collection and analysis.</td>
<td>Developing theory through induction directly from data.</td>
</tr>
<tr>
<td>Concurrent data collection and analysis.</td>
<td>Data analysis guides the collection of subsequent data (i.e. adaptation of subsequent interview schedules and participant selection).</td>
</tr>
<tr>
<td>Theoretical sampling.</td>
<td>Participant selection is based on the need to explore emerging concepts. The sampling strategy is based on constructing theory rather than population representation.</td>
</tr>
<tr>
<td>Constant comparison.</td>
<td>To classify data by developing codes and concepts through questioning and comparing incidents within the data i.e. events, emotions, behaviours.</td>
</tr>
<tr>
<td>The use of ‘memos’.</td>
<td>To understand and define relationships between categories. Memos are used to develop ideas, direct further data collection and to record the process of constant comparison.</td>
</tr>
<tr>
<td>Conducting the literature review after completion of data collection and data analysis.</td>
<td>To limit the influence that the researchers knowledge, viewpoint and beliefs has on the data collection and analytical process.</td>
</tr>
</tbody>
</table>

The disadvantages of grounded theory are that it is time consuming and requires the researcher to be wholly immersed in all aspects of conducting the study. The method also often involves researchers collecting data by multiple methods, with the use of ‘triangulation’, such as observation, focus groups and interviews to ensure thorough, rich data collection (Corbin and Strauss, 2008).
For the purposes of the current study a pure version of grounded theory was not adopted. Partly this was due to the limited time scale available relating to the academic purposes of the study. The mixed methods explanatory study design also did not lend itself to full grounded theory, as the explanatory study design involves the quantitative phase being the more important aspect of the study and being conducted prior to the qualitative phase (Creswell and Plano Clarke, 2007). Conducting the quantitative study first necessitated completion of the literature review prior the onset of the study, a contradiction in terms of true grounded theory, which requires the literature review to be conducted following the data collection and analysis (Glaser and Strauss, 1967). Furthermore, multiple qualitative data collection methods (e.g. interviews and focus groups) recommended by Corbin and Strauss (2008, page 27) as a means of quality assurance, was not possible (see section 4.10.2). This was due to the sensitive nature of the phenomenon under investigation.

4.11.2.2 Phenomenology

A phenomenology study was considered for the conduct of the current study because the experiences of patients who re-presented were to be explored to uncover rich, in-depth information of the event re-presentation. Phenomenology is the study of the lived experience of an individual, with the acceptance and acknowledgment of consciousness (Giorgi, 1997). It is a means of accessing the phenomena of human experience and is a rigorous descriptive approach (Husserl, 1913/1983, Giorgi, 1997).

Phenomenology is not commonly used in mixed methods studies, as the researcher becomes completely immersed in the phenomenon under investigation to gain sufficient in-depth view of the lived experience of the participants (Giorgi, 1997). It was decided to discount phenomenology for conducting phase two of this explanatory mixed methods study, as achieving complete emersion in the data would have been extremely time consuming and outside the time available to conduct this academic piece of work.

4.12 Rigour of the qualitative study

Evaluation of the quality of a research study is equally essential whether it be qualitative or quantitative in design (Corbin and Strauss, 2008, Ritchie et al., 2003, Miles and Huberman, 1994). Corbin and Strauss (2008) believe that the quality of a study is related to the rigour or trustworthiness of the study findings:
“the value of any research study lies in the substance, depth and innovation of the product that is generated” (page 303).

Lincoln and Guba (1985) relate trustworthiness to how well an investigator is able to convince others that the study findings are worthy of consideration and are credible.

There is much debate in the literature relating to how the trustworthiness of qualitative research should be defined. The quality of quantitative research is judged in terms of validity and reliability and is based within the positivist paradigm which indicates that there is only one ‘truth’ and that evidence is objective and value free. Some theorists therefore believe that quantitative terminology is not appropriate to express the trustworthiness of qualitative research (Strauss and Corbin, 1990, Smith and Heshusius, 1986, Lincoln and Guba, 1985). This is due to the naturalistic ontology underlying qualitative enquiry which asserts that multiple realities exist and data are value laden.

Researchers such as Lincoln and Guba (1985) define trustworthiness using four categories credibility, dependability, confirmability and transferability to assess the value and robustness of a study (Lincoln and Guba, 1985). Alternative terms such as ‘credible’ and ‘plausible’ have been used to determine the trustworthiness of research findings by Glaser and Strauss (1967).

However, other theorists and researchers believe that terms such as validity and reliability can be used to address trustworthiness when redefined (Pope et al., 2000, Lewis and Ritchie, 2003, Kirk and Miller, 1986, Long and Johnson, 2000). Lewis and Ritchie (2003) believe that reference to ‘validity’ during naturalistic enquiry should relate to “the validity of representation, understanding and interpretation” (page 273). The authors use terms such as reliability, inter-rater reliability and validity to assess the goodness of their research. The authors believe that,

"in their broadest conception, reliability meaning ‘sustainable’ and validity meaning ‘well grounded’ will have relevance for qualitative research since they help to define the strength of the data” (page 270).

The debate continues in the literature as to what constitutes the most appropriate terminology (Sommer Harrits, 2011, Cohen and Crabtree, 2008, Corbin and Strauss, 2008, Rolfe, 2006). Rolfe (2006) argues that the exploration for an overarching group of criteria to appraise the validity of qualitative research is
misguided and that there would be more value in assessing each research methodology (or even each study) according to its own worth.

In this study the terminology advocated by Lewis and Ritchie (2003) has been adopted to describe the trustworthiness of the qualitative study. This is because the study involves mixed methods and the use of similar terminology across the two phases of the study is intended to aid assessment of the quality of the study as a whole.

4.12.1 Assessing rigour

An important feature of ensuring the rigour of a qualitative study is that it is a continuous process throughout the study. Careful thought and assessment of the methods, processes and application of study procedures should occur at all stages of the research (from the design to the final report). Key to demonstrating 'rigour' is 'laying bare' the procedures used in the study to enable 'auditability', that is for others to assess and evaluate the study (Bowling, 1997, page 353).

Pope and Mays (1996) devised a checklist for assessing the rigour of qualitative research. They recommend that the theoretical framework, the context of the research and the methods be clearly described. Description of the methods should include fieldwork, sampling and analytical techniques (Pope and Mays, 1996). The authors advocate that sufficient 'raw' data be presented to enable the reader to assess the interpretation made by the investigator (Pope and Mays, 1996). Furthermore, triangulation methods are suggested to test the validity of the findings. Good record keeping is also a vital step in assuring that the study processes and procedures are accessible for others to evaluate the rigour of the study (Bowling, 1997). Snape and Spencer (2003) advise that the external influences that the researcher has on the study findings due to their beliefs and biases should be minimised to aid the rigour of the study.

Good record keeping, clear definition of the study methods, declaration of the researchers beliefs and potential biases, and ‘validating’ the study findings were all used to define rigour of the study (Bowling, 2002, Ritchie et al., 2003, Pope and Mays, 1996). Furthermore, aspects of the checklist devised by Pope and Mays (1996) were used to demonstrate the efforts that the researcher made to ensure the rigour of this study.
4.12.1.1 Good record keeping

Good record keeping was maintained throughout the study procedures and in particular included the use of 1) Framework analysis, 2) the use of computer aided qualitative data analysis software (CAQDAS), 3) keeping a journal, 4) clear description of study methods and 5) recording the contextual information of the interviews.

Framework analysis was used as it is a structured data analysis method and enabled the reader to review the processes used during analysis (Ritchie and Spencer, 1994). It also allowed access to the 'raw' data through the use of the charts (see section 4.11.1). The charts were recorded electronically using Microsoft® Word documents.

Data analysis was organised and conducted with the use of CAQDAS 'NVivo 8®', which enabled easy access to interview transcripts, evidence of indexing and the thematic framework (see section 4.11.1 and 6.3). A journal was maintained to record the decisions made by the researcher throughout the concurrent sampling, data collection and analytical processes of the study (Table 4-4) (Corbin and Strauss, 2008). Description of the study methods were recorded in the study protocol at the outset of the study, and a more detailed description are contained in section 4.10 of this thesis.

Information relating to the context of the interviews and the characteristics of the interviewees were also recorded and stored in 'NVivo 8®'.

4.12.1.2 Defining the study methods

The study methods are clearly defined as a whole in Chapter 4 of this thesis. Detailed information relating to the procedures used in this study can be seen in Table 4-4.

4.12.1.3 Beliefs and potential biases

The experiences, beliefs and potential biases of a researcher may influence the way that they analyse, interpret and construct meaning, with the potential of enforcing concepts on the data rather than allowing meaning to be derived from the data. Snape and Spencer (2003, page 20) suggest that researchers should remain as objective and neutral as possible during the conduct of qualitative research.
However, Corbin and Strauss (2008, page 33) believe that the researcher's experiences and knowledge cannot be removed from the analytical process. The authors suggest that it increases the analyst's sensitivity to the data and aides their understanding of what is occurring.

In the current study the gender, ethnicity and age of the researcher may have influenced the way in which the research viewed and applied meaning to the data. Furthermore, the researcher has been in full-time employment as an experienced nurse, with a depth of cardiac knowledge for many years. All the potential influencing factors listed may have influenced the way that the researcher conducted this study. Many of the interviewees were of the opposite gender, were currently unemployed or on sick leave and were experiencing financial difficulties, it was therefore important for the researcher to be aware of their 'own status' (relating to the fore mentioned factors) during data collection and analysis.

4.12.1.4 Validating the study findings

A number of methods appropriate to qualitative research (Lewis and Ritchie, 2003, Pope et al., 2000) were used to depict the validity of the study findings with the use of 1) inter-rater reliability, 2) presenting sufficient raw data for others to review, 3) triangulation of methods.

Throughout the analytical phases of the study the development of concepts and meaning and the overall study findings were checked by another experienced qualitative researcher. This was to ensure that the findings were not influenced by the researcher’s views and potential biases (Bowling, 1997, Pope and Mays, 1996). Lewis and Ritchie (2003, pages 271) refer to this as inter-rater reliability.

It was the intention of the researcher to present sufficient raw data in the qualitative findings section of the thesis (Chapter 8), to enable others to 'validate' the study findings (Pope and Mays, 1996).

The use of triangulation has been suggested by numerous authors as one means of validating qualitative research (Miles and Huberman, 1989, Patton, 2002, Denzin and Lincoln, 1994). Triangulation is described by Lewis and Ritchie (2003) as, "the use of different sources of information” “to improve the clarity, or precision, of a research finding” (page 275). Denzin (1978) suggests that the use of ‘methods triangulation’ using quantitative and qualitative methods, as a useful form of validating study findings. The mixed methods design of the current study incorporating quantitative and qualitative methods adheres to Denzin’s view of
‘methods triangulation’ and therefore may add to the validity of study findings (Denzin, 1978).

4.12.1.5 Generalisability

The generalisability or transferability of qualitative research is an aspect that initiates much debate in the literature. Some authors believe that it is possible to apply inferences from a study to a population where similar conditions exist (Lewis and Ritchie, 2003, Cronbach, 1975, Patton, 2002). However, Kearney (2007) suggests that it is more appropriate to achieve depth with qualitative research rather than making inferences across populations.

Generalisability of the qualitative findings in this study will be expressed in terms of inferences made at the level of categories, concepts and explanation in accordance with the recommendations of Lewis and Ritchie (2003, page 269-270).

4.13 Ethical considerations

As the current study was a non-interventional study the ethical risks associated with participating in the study were relatively small. During the quantitative phase of the study it was possible that individuals could become distressed during the telephone interviews due to the collection of data related to the original STEMI and the re-presentation events. On the rare occasions (approximately five) when participants became distressed, they were asked if they wished to terminate the telephone interview. Additionally discussion took place with participants who appeared to be anxious, depressed, or who gave cause for clinical concern, regarding seeking help via their GP. Alternatively, participants unwilling to access their GP practice were offered re-referral to the Cardiac Rehabilitation (CR) service which offered the psychological support of a nurse trained in delivering cognitive behavioural therapy. The GP and CR referral strategies were devised and negotiated a priori and formed part of the Multi Research Ethics Committee (MREC) application.

Similarly, it was possible that individuals participating in the in-depth semi structured interviews may experience some distress during the interview due to discussing the critical illness (i.e. STEMI and re-presentation). Conversely, it was acknowledged that participants may benefit from the interview process, as they had the opportunity to talk about their heart attack and re-presentation event.
To prevent undue distress, at the beginning of the interview the interviewer gave the interviewee the option of terminating the interview at anytime. During the interviews two of the interviewees became distressed and asked for the interview to be stopped; both were already receiving treatment for psychological distress from their GP. One of these participants was also interviewed in the presence of his/her young children and did not want to appear distressed in front of the children. A third participant became distressed during the interview, but wished to continue. Four other interviewees appeared to be experiencing psychological health issues, the interviewer therefore suggested that they may benefit from contacting their GP to receive further assessment and potential treatment.

During the quantitative phase of the study the participant responses to questionnaires would identify those patients with raised levels of anxiety and/or depression. In the instance that patients were identified with raised anxiety and depression levels in the clinical range, patients were advised to contact their GP. Consent was also gained from the participants during the informed consent process (see section 5.7.4), for the researcher to contact their GP during the study when appropriate. However, throughout the conduct of the study maintaining participants’ autonomy was regarded as paramount. In particular participants experiencing psychological distress may have been vulnerable to reduced self governance and disrupted decision making. The researcher therefore discussed possible treatment options related to further care, to enable the participants to make an informed choice, whilst avoiding coercion.

Throughout the study procedures were in place to maintain confidentiality and the anonymity of the participants. At the outset of the study all participants were allocated a study identification (ID) number to protect their identity. The ID number was used on all written and electronic documentation including the quantitative database and interview transcripts. All participant identifiable information was stored and maintained in accordance with the Research Governance Framework (Department of Health, 2005). Identifiable data and ID numbers were stored separately on a main server within Manchester Heart Centre and all study records were password protected. Paper copies of documents were stored in a locked filing cabinet within a locked room. The researcher was the identified responsible person for safe keeping of identifiable data and study documentation.

Joint ethical approval was gained for the main study and the current study from Stockport Multi Research Ethics Committee (MREC) (see Appendix D). Under the
National Research Ethics Service (NRES) guidelines at the time of gaining ethical approval for this study site-specific assessment (SSA) was not required at each of the participating sites. However local Research and Development (R&D) approval was required and obtained for each site.

4.14 Summary

In this chapter of the thesis the rationale for choosing the explanatory mixed methods design is discussed. The quantitative and qualitative methods are also discussed along with alternative methods and data collection techniques that were considered.

The first phase of the mixed methods study was the quantitative phase. The quantitative methodology chosen for this study was an exploratory prospective cohort study. Data collection involved self-report questionnaires, telephone interviews and data collected from participants’ health records. The qualitative and second phase of the study was conducted using modified grounded theory. The experiences of participants relating to re-presentation and their earlier STEMI were explored using in-depth semi-structured interviews on one occasion. Data analysis was conducted using Framework analysis.
CHAPTER 5 QUANTITATIVE METHODS

5.1 Introduction

This chapter describes methods used in the quantitative element of this mixed methods study; details of the mixed methods study design can be found in Chapter 4 and methods for the qualitative study can be found in Chapter 6. Included in the current chapter are details of the purpose of the quantitative study, the research hypothesis and the study aims. The planned data collection methods for the quantitative study and the planned statistical analysis are also described.

This study was part of a larger study that set out to compare the treatment received by STEMI patients, i.e. thrombolytic therapy and PPCI. The PPCI group was the focus for the current study and the re-presentation and angina symptoms data were unique data-sets for the study presented in this thesis.

5.2 Purpose of quantitative study

The purpose of the quantitative study was to determine the proportion of participants who re-presented to acute services due to potential IHD symptoms and to investigate the relationship between anxiety, depression, psychological distress, angina symptoms and the primary outcome of re-presentation during 6 months post PPCI, after controlling for other factors.

5.2.1 Definition of study cohort

For the purposes of this study the study cohort was divided into two groups: those who re-presented and those who did not re-present to acute healthcare services with potential IHD symptoms within 6 months of STEMI and PPCI.

5.3 Research hypothesis

Patients, who re-presented with potential IHD symptoms, during the first 6 months following STEMI and PPCI treatment, would have higher levels of anxiety, depression, psychological distress and/or levels of angina symptoms and/or more severe physiological health at baseline, compared to those who did not re-present.

5.4 Quantitative study aims

In order to test the study hypothesis it was necessary to establish the associations and relationships between the key variables and the outcome (re-presentation and
non-representation). To facilitate this, the methodology and subsequent analysis were sub-divided into a number of aims related to re-presentation, anxiety, depression, psychological distress and angina symptoms (physical limitation, angina stability and angina frequency). The conceptual model in Chapter 3 together with the relationships and associations established through the aims (1 to 5) below informed the investigation of the research hypothesis described in section 5.3, which was also directly related to aim 6.

**Aim 1**
To identify the number and frequency of re-presentations due to potential IHD symptoms, by determining and categorising the reasons for re-presentation during the first 6 months post STEMI.

**Aim 2**
To determine and compare the level of anxiety, depression, psychological distress and angina symptoms (angina stability, angina frequency, physical limitation, treatment satisfaction and quality of life) between the two groups at baseline and at 6 months.

**Aim 3**
To determine the change in the level of anxiety, depression, psychological distress and angina symptoms within the groups from baseline to 6 months.

**Aim 4**
To compare the change in anxiety, depression, psychological distress and angina symptoms between the two groups at 6 months.

**Aim 5**
To determine the association between the levels of anxiety, depression, psychological distress and angina symptoms for both groups at baseline and at 6 months.

**Aim 6**
To determine the association of psychological health (anxiety, depression and psychological distress), angina symptoms (stability, frequency and physical limitation) and physiological health (severity of STEMI and comorbidity) with re-presentation due to potential IHD symptoms, adjusted for other expected contributing or confounding factors previously identified in the conceptual model.
5.5 Quantitative study design

The quantitative aspect of this mixed methods study was conducted using a prospective, cohort, exploratory study design.

5.6 Study setting

The study was set across the Manchester conurbation and included the involvement of STEMI patients admitted for PPCI at the two Manchester PPCI treatment centres. During the recruitment phase of the study (May 2007 to January 2009) the PPCI service in Manchester was in its infancy. At that time, STEMI patients were initially taken to their local A&E department to be assessed prior to being transferred to one of the PPCI centres for treatment. In most cases, transfer patients were repatriated following their PPCI to the original transfer hospital. To facilitate recruitment of participants and data collection it was decided to seek approval to access patient identifiable data, from both the PPCI treatment centres and also the transfer hospitals.

The hospitals participating in the study and whether they were a PPCI treatment centre or a referral centre are listed in Table 5-1.

Table 5-1 Participating Hospital Trusts

<table>
<thead>
<tr>
<th>Trust</th>
<th>Trust Name</th>
<th>PPCI treatment or referral centre</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Central Manchester University Hospitals NHS Foundation Trust (CMFT)</td>
<td>PPCI treatment</td>
</tr>
<tr>
<td>2</td>
<td>University Hospital of South Manchester NHS Foundation Trust (UHSM)</td>
<td>PPCI treatment</td>
</tr>
<tr>
<td>3</td>
<td>Salford Royal HNS Foundation Trust</td>
<td>Referral</td>
</tr>
<tr>
<td>4</td>
<td>North Manchester General Hospital (The Pennine Acute Hospitals NHS Trust)</td>
<td>Referral</td>
</tr>
<tr>
<td>5</td>
<td>Stockport NHS Foundation Trust</td>
<td>Referral</td>
</tr>
<tr>
<td>6</td>
<td>Tameside Hospital NHS Foundation Trust</td>
<td>Referral</td>
</tr>
</tbody>
</table>
5.7 Population

All STEMI patients who were admitted to either of the Manchester PPCI treatment centres, Central Manchester University Hospitals NHS Foundation Trust (CMFT) or University Hospital of South Manchester NHS Foundation Trust (UHSM) were screened for suitability (inclusion and exclusion criteria are described in 5.7.1 and 5.7.2).

Information regarding current and past medical history was taken from the patient notes to enable an assessment of eligibility to be made. Individuals were approached if they satisfied the following inclusion and exclusion criteria.

5.7.1 Inclusion criteria

- ≥18 years.
- Diagnosis of STEMI: ST-segment elevation ≥1mm in ≥2 contiguous leads, or new left bundle branch block with IHD symptoms or chest pain for <12 hours [in line with ESC/AHA guidance on delivery of PPCI (Wijns et al., 2010)].
- Received Primary Percutaneous Cardiovascular Intervention (PPCI).
- Able to understand written/spoken English.
- Able to provide informed consent.

5.7.2 Exclusion criteria

- Participation in another psychological study within the past 30 days.
- Diagnosis of severe cognitive impairment, for example dementia, Alzheimer’s, severe learning disabilities.
- Severe physical illness or severe mental illness (at baseline) limiting the individual’s ability to participate in the study. Severe mental illnesses included psychosis or a current acute psychiatric event requiring treatment through the acute psychiatric services. Individuals receiving treatment in the form of either medication or a psychological treatment (cognitive behavioural therapy, psychotherapy or counselling) for anxiety and depression were not excluded.
5.7.3 Sampling and sample size

It was planned that all eligible patients over a twelve month period would be approached consecutively (i.e. all patients who underwent PPCI and met the eligibility criteria). However, recruitment of participants took longer than expected and ran from May 2007 until January 2009; this is discussed further in section 9.7.1.

It was decided to undertake an exploratory study (see section 4.8 for full justification) and therefore a formal sample size calculation was not required. However as a guide, using Raosoft ® software (http://www.raosoft.com/samplesize.html) a sample size calculation was undertaken. This was to aid the planning of the study and set timelines for participant recruitment.

As previously discussed combined re-presentation rates (including both cardiac ischaemia and potential IHD events) have not previously been reported. The response distribution to be included in the sample calculation was therefore based on Major Adverse Cardiac Events (MACE) rates reported in a number of randomised controlled trials (RCTs) involving PPCI patients. MACE rates include physiological abnormalities such as re-infarction, revascularization and stroke; they also often include death but do not include diagnoses such as pulmonary, gastric or musculoskeletal problems. Two RCTs involving PPCI patients, the GUSTO IIb study (GUSTO IIb angioplasty substudy investigators, 1997) and the CADILLAC study (Stone et al., 2002) reported MACE rates of 9.6% and 11.3% at 30 days and 6 months respectively. It was expected that the re-presentation rate would be somewhat higher when including both cardiac and potential IHD re-presentations, thus a re-presentation rate of 15.0% was chosen. For estimating a re-presentation rate of 15.0%, with a predicted margin-of-error of 5.0% for a 95% Confidence Interval (CI), the necessary sample size was 195 patients.

During the planning stages of the study (from April 2006 to April 2007) the total available STEMI patient population treated with PPCI was 180 at CMFT (n=110) and UHSM (n=70) respectively. It was predicted that the number of PPCI patients would increase to >300 patients per year (25 per month) across the Manchester conurbation, during the subsequent 12 months due to the planned expansion of the PPCI service. Lane et al. (2000b) has previously reported a refusal rate of 34.0% for an MI cohort participating in a self-report questionnaire based study (Lane et al., 2000b). Based on a refusal rate of 35.0% three hundred participants were
required to reach the study sample size of 195 participants for the current study and recruitment was predicted to take 12 months.

5.7.4 Patient recruitment

Potential participants were to be identified through the catheter lab scheduling system at both PPCI centres. All patients treated with PPCI are recorded on these systems. The researcher also planned to contact the coronary care units (CCU) at both PPCI centres and transfer hospitals to identify the projected discharge times of PPCI patients. This was to ensure that, wherever possible, eligible patients could be approached face to face by the researcher whilst the patient was still an in-patient following PPCI treatment. The intention was to use a personal approach to recruitment to ensure a higher recruitment rate.

On approaching the patient a verbal description and patient information was offered. Patients were given time (at least 24 hours) to consider taking part, and subsequently consent was taken before discharge (see Appendix E for a copy of the patient information sheet and consent form). Those who were missed during their admission were sent an information pack (including an introductory letter, a patient information sheet, consent form and stamped addressed envelope) through the post within several days of discharge. Prior to sending out postal packs the patients status was checked to ensure the patient had not died during their hospital admission. Patients who had been contacted by post were then contacted by telephone one week following discharge to ascertain whether they wished to take part. Those who expressed a wish to participate in the study were asked to complete the consent form and return it in the stamped addressed envelope.

5.8 Data collection

5.8.1 Data collection at baseline

Data collected as part of this study at baseline included sociodemographic data, PPCI admission details, clinical data, co-morbid conditions, medical history, psychological health and angina symptom occurrence (see Table 5-2). Data were collected by the researcher and a research assistant (employed to work on the main study) from medical records and with the use of a pro forma designed by the researcher and the researcher’s supervisors. Psychological health data and angina symptom occurrence were collected using self-report questionnaires the Hospital Anxiety and Depression Scale (HADS) (Zigmond and Snaith, 1983) and the Seattle Angina Questionnaire (SAQ) (Spertus et al., 1995) respectively at baseline and 6
months (see 5.10). Information regarding levels of social support was also collected using a self-report measure the ENRICHED Social Support Inventory (Mitchell et al., 2003) at baseline. The full list of variables, the sources of data and the time points that data was captured can be seen in Table 5-2.

Table 5-2 Description of study variables, data sources and time points of data collection

<table>
<thead>
<tr>
<th>Variable name</th>
<th>Source of data</th>
<th>Type of variable</th>
<th>Data collection time point</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sociodemographic variables</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>Patient records and Pro forma</td>
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<td>✓</td>
</tr>
<tr>
<td>Gender</td>
<td>Patient records and Pro forma</td>
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<td>✓</td>
</tr>
<tr>
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<td>Pro forma</td>
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<tr>
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<td><strong>Index PPCI admission variables</strong></td>
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<td>1Interview</td>
<td>categorical</td>
<td>✓</td>
</tr>
<tr>
<td><strong>Co-morbidity variables</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CCI</td>
<td>Patient records and 1Interview</td>
<td>continuous</td>
<td>✓</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Patient records and 1Interview</td>
<td>categorical</td>
<td>✓</td>
</tr>
<tr>
<td>Diabetes type</td>
<td>Patient records and 1Interview</td>
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<td>✓</td>
</tr>
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<td>Pulmonary disease</td>
<td>Patient records and 1Interview</td>
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<td>✓</td>
</tr>
<tr>
<td>Previous cardiac history</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous cardiac event</td>
<td>Patient records</td>
<td>categorical</td>
<td>✓</td>
</tr>
<tr>
<td>Multiple MIs</td>
<td>Patient records</td>
<td>categorical</td>
<td>✓</td>
</tr>
<tr>
<td>CABG</td>
<td>Patient records</td>
<td>categorical</td>
<td>✓</td>
</tr>
<tr>
<td>PCI</td>
<td>Patient records</td>
<td>categorical</td>
<td>✓</td>
</tr>
<tr>
<td>Heart Failure</td>
<td>Patient records</td>
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<td>✓</td>
</tr>
<tr>
<td><strong>Ischaemic Heart Disease risk factor variables</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable name</th>
<th>Source of data</th>
<th>Type of variable</th>
<th>Data collection time point</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of risk factors</td>
<td>Patient records</td>
<td>categorical</td>
<td>√</td>
</tr>
<tr>
<td><strong>Cardiac rehabilitation variables</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Offered cardiac rehab</td>
<td>1Interview</td>
<td>categorical</td>
<td>√</td>
</tr>
<tr>
<td>Attended cardiac rehab</td>
<td>1Interview</td>
<td>categorical</td>
<td>√</td>
</tr>
<tr>
<td><strong>Re-presentation variables</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of admissions (0-6 m)</td>
<td>Patient records, 1interview and diary card</td>
<td>continuous</td>
<td>√</td>
</tr>
<tr>
<td>Number of A&amp;E attendances (0-6 m)</td>
<td>Patient records, 1interview and diary card</td>
<td>continuous</td>
<td>√</td>
</tr>
<tr>
<td>Symptoms</td>
<td>Patient records, 1interview</td>
<td>descriptive</td>
<td>√</td>
</tr>
<tr>
<td>Initial diagnoses</td>
<td>Patient records</td>
<td>categorical</td>
<td>√</td>
</tr>
<tr>
<td>Discharge diagnoses</td>
<td>Patient records</td>
<td>categorical</td>
<td>√</td>
</tr>
</tbody>
</table>

1Telephone interview  
2ENRICHD Social Support Instrument (ESSI)  
3Global Registry of Acute Coronary Events (GRACE)  
4Hospital anxiety and depression scale (HADS)  
5Seattle Angina Questionnaire (SAQ)  
6New York Heart Association (NYHA)  
7Canadian Cardiovascular Society Angina score (CCSC)  
8Charlson Co-morbidity Index (CCI)

A patient pack, including the study pro forma and self-report questionnaires, was posted to participants within 14 days of discharge (baseline measure). It was decided to post the pro forma and all baseline questionnaires together to limit the burden on the responder (see 0 for a copy of the baseline patient pack). Posting the documents at the same time also allowed systematic study management, by the researcher keeping track of the dates that the documents were sent out and returned. The intention of the methodical study administration was to achieve high response rates.

The baseline time point of 14 days for mailing questionnaires was chosen as a compromise, because there were varying ideal data collection time points for the HADS and SAQ instruments for this study. Ideally HADS would have been mailed immediately following discharge, as previous researchers have reported and validated its use within 7 days of STEMI (Dickens et al., 2004). However, the SAQ is designed to capture the participants’ experiences of angina symptoms within the preceding 4 weeks of completion and it was the intention of this study to measure symptoms following rather than prior to STEMI (required for the multivariate analysis). Had the SAQ been mailed within 7 days of STEMI the majority of individuals may have reported symptoms prior to STEMI. In practice the mean time point of the baseline questionnaire completion (from STEMI occurrence) as indicated by participants, was 19.4 days (SD=10, range=5 to 44 days).
A baseline telephone interview was conducted by the author with the use of a telephone interview pro forma (see Appendix G), within one week of questionnaires being mailed. This was to collect details of co-morbidity (relating to a co-morbidity index) and to conduct clinical assessment of angina and heart failure classification. Angina and heart failure classification were established using the Canadian Cardiovascular Society’s Classification (CCSC) (Campeau, 1976) and the New York Heart Association (NYHA) functional classification system (see Appendix J and Appendix K for details of the classifications). Telephone interviews were also an opportunity to offer participants assistance in completing questionnaires (if required) and to act as a reminder for those who had not already returned their questionnaires.

5.8.2 Data collection at 6 months

At 6 months psychological health and angina symptom occurrence were again collected through self-report measures, which were posted to participants. A telephone interview was conducted by the author, within one week of questionnaires being mailed. This was to remind individuals to return their questionnaires (if they had not already done so) and to assist participants in completing questionnaires if needed.

The telephone interview was also an opportunity to collect other information including whether cardiac rehabilitation had been offered to participants and to what degree (if at all) they had attended (see Appendix G for a copy of the telephone interview pro forma). Participants were also asked if they had undergone any further revascularisation (CABG or PCI) since their PPCI. The clinical assessment of angina and heart failure classification using the CCSC and NYHA classifications respectively were also repeated (American Heart Association, 1994, Campeau, 1976). Telephone interviews were also used to collect 6 month re-presentation data. Details of re-presentation variables and data collection are discussed further in section 5.8.3.

Prior to telephoning patients at 6 months the strategic tracing service was accessed to identify deceased patients; this was to prevent causing unnecessary distress to relatives of deceased patients.

5.8.3 Re-presentation data

The number and frequency as well as categorisation of re-presentations during the 6 months following STEMI and PPCI were collected.
5.8.3.1 Collection and completeness

The collection of the number, frequency and reason for each re-presentation was conducted through review of patient records, the completion of a diary card by the participant (from baseline to 6 months) and telephone contact with participants at 6 months.

To facilitate completeness of data collection and ensure that a re-presentation was not missed all participants’ A & E and hospital records were reviewed at both the transfer (for transfer patients) and the PPCI hospital. In most cases the participant’s local hospital was also the transfer hospital and it was deemed to be possible that the participant would re-present to their local hospital in the first instance.

During the 6 month telephone call participants were asked whether they had either attended A and E or been admitted to hospital due to chest pain (or symptoms that they believed to be related to their heart) since their PPCI. To help participants to remember events and the dates that events had occurred, participants were given a diary card at baseline and asked to record all hospital admissions and visits to acute services. The diary card was collected at 6 months post STEMI (see 0 for an example of the diary card). Review of participant notes, the telephone interviews and data assimilation from the diary cards were conducted by the author (a trained cardiac nurse).

5.8.3.2 The categorisation of potential IHD events

The identification of participants with potential IHD events was conducted with the use of an a priori definition (Table 5-3); this was developed with the use of The National Institute for Health and Clinical Excellence (NICE) chest pain (2010a) and NICE Unstable angina and NSTEMI (2010b) guidelines, and in consultation with Dr Fath-Ordoubadi, Consultant Cardiologist. If participants experienced a re-presentation that fitted with any of the criteria 1 to 4 (and also criteria 5) of the a priori definition at any point during the first 6 months post STEMI, the participant was included in the re-presentation group. The re-presentation group consisted of individuals who suffered one or more potential IHD events during the 6 month follow-up. In the non-representation group a participant may have experienced an acute re-presentation not related to a potential IHD event (according to the a priori definition, Table 5-3). Patients were excluded from the re-presentation group and included in the non-representation group if there was a clear non-cardiac reason for attending A & E that was not considered to be related to potential myocardial
ischaemia (e.g. sprained ankle). Validation of participant inclusion in the re-presentation group was undertaken by the author’s supervisory team.

Table 5-3 a priori criteria for inclusion of participants to the potential IHD re-presentation group.

<table>
<thead>
<tr>
<th>Criteria number</th>
<th>Criteria for the inclusion of potential IHD re-presentation group</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Initial admission diagnosis as possibly IHD ischaemic event.</td>
</tr>
<tr>
<td>2.</td>
<td>Symptoms that could possibly be IHD related i.e. chest pain,</td>
</tr>
<tr>
<td></td>
<td>breathlessness, jaw pain, left arm pain, feeling unwell along</td>
</tr>
<tr>
<td></td>
<td>with nausea and/or vomiting, collapse.</td>
</tr>
<tr>
<td>3.</td>
<td>Participant believed that they were having a cardiac event.</td>
</tr>
<tr>
<td>4.</td>
<td>The participant’s relative, or a healthcare professional attending to them believed that the participant was having a possible cardiac event.</td>
</tr>
<tr>
<td>5.</td>
<td>Attended A&amp;E either by self referral or by ambulance or direct referral by GP falling within the above categories.</td>
</tr>
</tbody>
</table>

5.8.3.3 Final diagnostic categorisation

The A&E and medical records of each participant included in the re-presentation group received further review to establish a final diagnosis for every re-presentation. The data in Table 5-4 were collected for each re-presentation to aid identification of a final diagnosis.

Table 5-4 Data collected to establish a final discharge diagnosis for each re-presentation.

<table>
<thead>
<tr>
<th>Data collected to establish final diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Final discharge diagnosis.</td>
</tr>
<tr>
<td>2. Initial diagnosis.</td>
</tr>
<tr>
<td>3. Symptoms associated with re-presentation.</td>
</tr>
<tr>
<td>4. Investigations performed during A&amp;E attendance and/or hospital admission.</td>
</tr>
<tr>
<td>5. Route of admission.</td>
</tr>
</tbody>
</table>

Information relating to re-presentations was reviewed by the author and wherever possible a final decision was confirmed. In some instances a final diagnosis was not clearly documented in the patient notes and it was necessary for the researcher to use the additional information described in Table 5-4 (points 2 to 5) to determine the final diagnosis. Additionally, for some participants the documented symptoms and results of investigations were discussed with Dr Fath-Ordoubadi, Consultant Cardiologist to come to a final diagnosis. All decisions in relation to diagnosis were validated by the author’s supervisory team.

In the case of individuals who re-presented more than once, all acute re-presentations were recorded, including possible non-IHD re-presentations (e.g. participants who suffered a broken bone, eye problems or gastric problems). This
was to ensure that events were fully reviewed and to ensure that gastric, musculoskeletal or psychological related events were not missed. In the case of an inconclusive diagnosis, the final diagnosis was categorised as ‘no diagnosis’.

5.9 Reliability and validity of study questionnaires

Reliability and validity of measures were assessed in accordance with procedures discussed in section 4.9 and are further described in sections 5.9.1 and 5.9.2).

5.9.1 Reliability

Evidence to support the use of the chosen instruments in this study was sought from the literature including internal consistency (when available). The justification for inclusion of instruments is presented in section 5.11. Wherever possible the Cronbach’s alpha is presented for the instruments chosen for inclusion in this study (Cronbach, 1951) see section 4.9.1.

5.9.2 Validity

The internal validity of instruments used in this study was assessed by reviewing the literature for evidence that measures had been well used and tested in similar populations to that of the study cohort (Parahoo, 1997). Construct validity and sensitivity and specificity of measures are also presented where possible (Hoepfl, 1997).

Assessment of external validity was conducted for the current study despite the exploratory study design, to establish to what degree the study results could be generalised to the wider STEMI population (see section 4.9.2). As discussed in section 5.7.3a sample size was based on the re-presentation rates reported in previous clinical trials involving STEMI patients.

5.10 Study instruments

The validity of instruments chosen for inclusion in this study cohort is presented along with the reliability data in this section. A range of techniques and evidence were used by various researchers during the development or evaluation of the measures, to demonstrate reliability and validity of the instruments.
5.10.1 Psychological health assessment

Psychological health assessment was undertaken with the use of a self-report measure. The justification for the use of this methodology is set out in section 4.8.4.

A number of instruments are available to assess psychological health status (anxiety and depression); numerous tools assess anxiety and depression separately.

5.10.1.1 Anxiety

One instrument that measures anxiety is the State Trait Anxiety Inventory (STAI) (Spielberger, 1970). The STAI can be used as a clinical diagnostic tool and aids differentiation between anxiety and depression (Spielberger, 1970). It has been used by numerous researchers in MI populations (Frasure-Smith et al., 1995b, Cossette et al., 2001). It is a lengthy questionnaire with 40 questions measuring two sub-scales (state and trait anxiety) (Tilton, 2008) and due to its length it was discounted for use in the current study. A further anxiety related measure is the Anxiety Sensitivity Index (ASI), which has previously been used in an investigation of cardiac and non-cardiac chest pain (Keogh et al., 2004). The ASI has commonly been used to assess sensitivity to anxiety (believing that anxiety symptoms have harmful affects) and fearfulness in groups suffering anxiety disorders (Peterson and Heilbronner, 1987). The measure is useful in determining panic disorder from other general anxiety disorders, though it has less commonly been used in groups with medical conditions (Reiss et al., 1986). The measure focuses mainly on somatic aspects of anxiety and therefore, due to the underlying CHD of the study cohort, it was decided to exclude the use of ASI (Deacon et al., 2003).

5.10.1.2 Depression

The Beck Depression Inventory (BDI) is commonly used by researchers to assess depression (Beck et al., 1988). It is most frequently used in the diagnosis of depression rather than assessing overall psychological health status in participants with physical medical conditions (Beck, 1967). The BDI has been validated in numerous medical populations including cardiac and MI patients (Grace et al., 2005a, Frasure-Smith et al., 1995b). A further measure commonly used as a diagnostic tool is the Zung Self-Rating Depression Scale (SDS) (Zung et al., 1983); this measure is often used in the general population and has rarely been used in cardiac groups. The BDI and SDS were both discounted for this study, due to their use for diagnostic purposes and their high level assessment of somatic symptoms.
Furthermore, the inclusion of instruments that measure anxiety and depression separately consequently leads to higher burden on the respondent and therefore a measure which included both anxiety and depression was preferred.

5.10.1.3 Hospital Anxiety and Depression Scale
The Hospital Anxiety and Depression Scale (HADS) was chosen to assess psychological health in this study because it was developed specifically for use in medically ill populations and excludes bodily symptoms such as sleep disturbance, fatigue and pain that may be due to physical illness (Zigmond and Snaith, 1983, Dickens et al., 2004). A copy of the instrument can be found in 0.

The HADS is a self-rated 14 item scale which measures the symptoms of anxiety and depression. Seven items of the scale relate to anxiety and seven to depression. The sub-scales (anxiety and depression) are each measured on a 4-point scale from 'no not at all' (score=0) to 'yes definitely' (score=3). Two of the HADS anxiety items are reverse scored and 'yes definitely' (score=0) and 'no not at all' (score=3) and this also applies to four of the HADS depression items. The total scores for each sub-scale are 21, with higher scores indicating higher levels of anxiety or depression symptoms. The range of scores on each subscale can be classified as normal (0-7), mild (8-10), moderate (11-14) and severe (15-21) (Zigmond and Snaith, 1983). This classification was used to interpret HADS results in the current study. The HADS anxiety and HADS depression scores may be combined (often referred to as psychological distress) an overall measure of psychological health (Herrmann, 1997).

The use of cut-offs and categorical HADS scores are commonly applied by researchers, but there is not a single widely accepted cut-off score (Herrmann, 1997). Zigmond and Snaith (1983) in their original study recommend two cut-off scores indicating ≥8 possible and ≥11 probable for both anxiety and depression. In their manual Snaith and Zigmond (1994) also suggest a further cut-off for total HADS of ≥14 as severe, yet they do not offer any data to support the use of this cut-off. However, the evidence presented in Strik et al (2001) and Dickens et al’s (2004) studies support the use of ≥11 and ≥17 total HADS cut-offs.

The HADS has commonly been used and validated in a range of physically ill populations (Snaith and Zigmond, 1994). Researchers have also validated its use in a range of ischaemic heart disease populations including STEMI and MI patients (Dickens et al., 2004, Herrmann, 1997, Strik et al., 2001, Mortensen et al., 2005, Snaith and Zigmond, 1994). Mortensen et al (2005) used the HADS in a cohort of
STEMI patients treated with PPCI. In an RCT the researchers randomized 1572 participants to receive either PPCI or thrombolytic therapy, and of those participants 1352 completed the HADS questionnaire. The reliability of the HADS was reported with Cronbach’s alphas of 0.88 for anxiety and 0.82 for depression. Strik et al (2001) demonstrated an internal consistency with Cronbach’s alphas for HADS sub-scales, anxiety 0.83, depression 0.82 and total HADS 0.88, in a study of 206 patients one month following first MI.

A cohort of German patients (n=203) who presented with suspected angina were investigated by Herrmann (1997) and internal consistencies of HADS using Cronbach’s alphas were 0.80 for anxiety and 0.81 for depression. The study showed that individuals with high anxiety HADS scores, compared to those with normal scores, had significantly fewer positive angiograms (p=0.01; n=60). This evidence suggests that the participants’ chest pain was not explained through clinical cardiac investigations, yet was discriminated by German HADS anxiety scores. Herrmann (1997) suggests that it is appropriate to apply the findings of studies involving the German HADS in German cohorts to English participants:

"Because formal characteristics are almost identical for the English and German versions, and HADS scores have also been found identical in comparable groups of cardiological patients from England and Germany, the German standard values should also apply to English heart patients“ (page 22).

The specificity and sensitivity of combined HADS (psychological distress) in an MI cohort (n=589) has been investigated by Dickens et al (2004). The participants were asked to complete the HADS retrospectively during admission to establish their mental state one week prior to their MI. In order to validate the HADS in their study cohort the authors assessed the participants mental health by conducting psychiatric interviews in a subset of participants (n=314) using the ‘Schedules for clinical assessment in neuropsychiatry’ (Anon., 1996). They found that HADS psychological distress of >17 had the best sensitivity (87.7%) and specificity (84.7%) in relation to depressive disorder based on the psychiatric interviews (Dickens et al., 2004). However, in Strik et al’s (2001) study the combined HADS optimal cut-off was reported as 11/12 with sensitivity 78.1% and specificity 85.0% using a ROC curve. The difference in the cut off’s between the studies of Dickens et al (2004) and Strik et al (2001) may have partly been related to the differing time points that the HADS was used (retrospectively to reflect the participants mental state one week prior to MI and one month post MI respectively).
A licence for use of HADS in this study was obtained through the main study. The HADS anxiety, depression and total HADS data were collected in this study at baseline and 6 months to address the study aims. Two separate dichotomised scores (cut offs) based on Zigmond and Snaith's (1994) original study, were also used in part to address the study aims. The first dichotomised cut off chosen was anxiety or depression scores \( \geq 8 \), indicating raised (mild or worse) levels. The second dichotomised cut off was scores \( \geq 11 \), indicating moderate as a minimum level of anxiety or depression (Zigmond and Snaith, 1983). Furthermore, it was decided to include \( \geq 17 \) as total HADS cut-off. This decision was based on the sensitivity (87.7%) and specificity (84.7%) (in relation to depressive disorder) demonstrated in Dickens et al (2004) study. Clinically, it is important to acknowledge that raised HADS scores are only indicative of probable anxiety or depression and prior to diagnostic confirmation individuals require clinical assessment for anxiety and depression (Herrmann, 1997).

### 5.10.2 Symptom assessment

Symptom assessment was undertaken with the use of a self-report measure. The full justification for the use of this methodology is set out in section 4.8.4.

#### 5.10.2.1 Generic Symptoms

There are a number of instruments that assess general symptoms such as the Brief Symptom Inventory (BSI) (Derogatis and Melisaratos, 1983) and the 90-item Symptom Checklist (SCL-90) (Derogatis, 1975). The BSI and SCL-90 have both previously been used in AMI patients (Moser and Dracup, 1996, Derogatis and Melisaratos, 1983, Ruz et al., 2010, Strik et al., 2001). However, symptom assessment in these instruments does not directly relate to ischaemic heart disease symptoms, a necessary focus for the present study, and therefore these tools were not considered further.

#### 5.10.2.2 Disease Specific

Few disease specific symptom assessment tools relating to cardiac ischaemia are currently available. A number of instruments have been developed for use with other cardiac groups such as the Minnesota Living with Heart Failure Questionnaire (LHFQ) for heart failure patients (Rector et al., 1987), the Heart Transplant Symptom Scale (Jalowiec et al., 1997) for individuals receiving a heart transplant and Specific Symptoms Scale for cardiac arrhythmia patients (Brignole et al., 1997). However, many of these measures focus on specific symptoms for the
groups concerned such as fatigue, dyspnoea and exercise intolerance and therefore were excluded for use in this study.

The Physical Symptom Incidence and Distress Scale is a more general cardiac symptom reporting scale and includes symptom severity and frequency of both cardiac (chest pain) and non-cardiac (headache) related symptoms (Glazer et al., 2002). However, this measure was developed and validated in a range of cardiac rehabilitation patients (including IHD and heart failure patients) and therefore is not specific to cardiac ischaemia. As the current study directly involves re-presentation due to potential IHD symptoms, and therefore relies on the participants’ experience of cardiac ischaemia symptoms such as angina, an instrument that focuses on IHD symptoms was deemed to be the most appropriate instrument. Two such instruments are available for use and have been validated in a variety of IHD patients, the Rose Angina Questionnaire (Rose et al., 1977) and the Seattle Angina Questionnaire (Spertus et al., 1995).

The Rose Angina Questionnaire was designed as a diagnostic aid and is one of the few instruments that are specifically designed to assess levels of angina (and discomfort) related to cardiac ischaemia (Rose et al., 1977). It is a short questionnaire including only 8 questions relating to pain and discomfort in the chest (Rose et al., 1977). A variety of means have been used in validating the instrument (e.g. ECG, expert clinical diagnosis, thallium scan), but there are no overall agreed means of validating the tool (Fischbacher et al., 2001).

A modified version of the Rose Angina Questionnaire (Mortensen et al., 2005) was used as a self-report instrument to measure angina in a study involving a cohort of STEMI patients treated with either PPCI or thrombolytic therapy. However, as previously discussed the instrument was designed as a diagnostic tool and the researchers (Mortensen et al., 2005) did not undertake reliability and validity checks for the use of this tool as a self-report measure. Due to the diagnostic nature of the instrument and the lack of reliability and validity measurements for use as a self-report measure, it was decided to exclude the use of this instrument for the current study. The most appropriate instrument for use in this study was the Seattle Angina Questionnaire (SAQ), details of which are described below (Spertus et al., 1995).

5.10.2.3 The Seattle Angina Questionnaire

The SAQ was chosen for use in this study because it allows quantification of the stability of angina symptoms, the frequency that symptoms occur and the degree of
limitation to activities (due to cardiac ischaemia symptoms). All three of these items are reliable measures of IHD symptoms from the participants’ perspective for the purposes of this study. The SAQ has also been widely used in cardiac populations to assess health status and IHD symptoms specifically related to coronary heart disease (Spertus et al., 1995). A copy of the instrument can be found in 0.

The SAQ has five subscales that measure physical limitation (due to Coronary Heart Disease (CHD)), angina stability, angina frequency, (cardiac) treatment satisfaction, and quality of life (QoL). Each subscale is scored separately and no total score is given. Scale scores are all normalized to a scale of 0 to 100 for ease of interpretation, with higher scores representing better health status. A 10 point change in score is considered clinically relevant (Spertus et al., 1995).

Initial validation studies were carried out in primarily male patients with stable coronary heart disease, and those undergoing angioplasty (Spertus et al., 1995). The individual scales were found to be valid, reproducible and responsive, with significant correlations between the sub-scales and external testing of the domains (Spertus et al., 1995, Dougherty et al., 1998). The SAQ has also been used more recently in numerous clinical studies, including those involving STEMI patients treated with PPCI (Rinfret et al., 2001, Norris et al., 2004, Oesterle et al., 2000).

A licence for the use of the SAQ in this study was purchased from the author of the instrument via CV Outcomes (http://cvoutcomes.org/). The SAQ was used at baseline and 6 months to determine levels of angina symptoms (according to the sub-scales) and also to ascertain the changes in the levels at 6 months. The SAQ is designed to capture the participants’ experiences of angina symptoms within the preceding 4 weeks of SAQ completion and this is stated on the questionnaire. The instrument was mailed to participants at 14 days post STEMI (see section 5.8.1 for full justification) and therefore it was possible that responses were a mixture of before and after STEMI, depending on when the participant completed the instrument. In an attempt to ascertain the variation of the timing of responses participants were asked to complete the date that they completed the questionnaires. However, on the whole, participants did not do so and it is therefore acknowledged that the actual timing of instrument completion (HADS, SAQ and ESSI) reported in section 5.8 is only a guide.
5.10.3 Social support assessment

Social support assessment was undertaken with collection of sociodemographic data and the use of a self-report measure (see section 4.8.4).

5.10.3.1 ENRICHD Social Support Inventory

Reduced social support has been reported as a precursor to depression, poor recovery as well as increased mortality in post MI patients (Dickens et al., 2004, ENRICHD Investigators, 2003). It was therefore decided to include this variable in the current study. See Chapter 3 for full justification of the variables included in the study.

The ENRICHD Social Support Inventory (ESSI) is a self-report instrument developed for use in cardiac patients to measure social support (Mitchell et al., 2003). The ESSI was used in the current study (and the larger study) to give an objective measure of social support; a copy of the instrument can be found in 0.

The ESSI includes whether an individual has a partner (structural support), tangible help (instrumental support) and whether someone offers care (emotional help) to the individual (Mitchell et al., 2003). It is a 7 item scale (a 5 item scale is also available) that was developed as part of the Enhancing Recovery In Coronary Heart Disease (ENRICHD) study to identify areas of social support that are reported to influence mortality in CHD patients (ENRICHD Investigators, 2003). Six of the 7 items (questions 1 to 6) have 5 categories ranging from ‘none of the time’ (score=1) to ‘all of the time’ (score=5). Question 7 ‘are you currently married or living with a partner’ was scored yes (score =4) or no (score=2). The total score for the instrument was 34 and higher scores indicated better social support. Low social support was determined by a total score <18, or 2 items with a score <3, or 2 items with a score <2 irrespective of the total score.

The validity of the ESSI was demonstrated with a modest correlation coefficient between the ESSI and the SF-36 mental component scores (MCS) (McHorney et al., 1993) and the SAQ QoL scale (at baseline) [SF-36 MCS 0.31 (p<0.001) and SAQ QoL 0.22 (p<0.001)] (Vaglio et al., 2004). Reliability was demonstrated with internal consistency of Cronbach’s α = 0.88 (Vaglio et al., 2004). A 5 item scale was also validated by the ENRICHD investigators (2003); both the 5 and 7 item scales have been shown to be reliable and valid and there are very few differences between the measures. The ESSI was chosen for use in the main study; it was
decided by the investigators of the main study to use the original 7 item scale (Mitchell et al., 2003).

## 5.10.4 Severity of STEMI assessment

The severity of STEMI was included in this study as the size of infarct and resulting morbidity may lead to further IHD symptoms and therefore possible re-presentation (see section 3.5.2.1 for justification).

### 5.10.4.1 Clinical Assessment

A number of methods can be used at different time points to assess severity of STEMI. These include Killip class, which is based on assessment of the lung fields and is conducted early during STEMI (Killip and Kimball, 1967), biochemical assessment with the use of Troponin T, a biomarker for myocardial necrosis (Thygesen et al., 2007) and categorisation of Left Ventricular (LV) function during the early recovery phase of STEMI (van de Werf et al., 2008, Wijns et al., 2010). The clinical history, including the amount of ST-elevation (and the number of affected ECG leads), as well of the area of myocardium affected by infarction is also helpful in building up a picture of the extent of damage to the myocardium (Antman et al., 2004). Furthermore the speed of treatment (in the case of this study PPCI) also may influence long term damage to the myocardium and therefore severity of the STEMI (Stone et al., 2002, Madsen et al., 1997).

### 5.10.4.2 The GRACE Score

Currently, no one measure is available to summarise the severity of STEMI; NICE recommends the use of a composite score such as The Global Registry of Acute Coronary Events (GRACE) to calculate the probability of survival from either admission to 6 months, or discharge to 6 months post Acute Coronary Syndrome (ACS) event (National Institute for Health and Clinical Excellence, 2010a, Fox et al., 2006). The GRACE score (predictive outcome from admission to 6 months) was chosen for inclusion in this study as a measure of ‘severity of STEMI’, because it is a continuous, composite, predicted outcome measure (Granger et al., 2003, Fox et al., 2006). The GRACE is presented as both a whole number (score) and as a percentage probability of i) death, and ii) death or myocardial infarction (MI).

The conduct of a large, prospective international, observational study (the GRACE study) of patients admitted to hospital with ACS (both STEMI and N-STEMI patients) was used to develop the GRACE score (Fox et al., 2006). The main aim of the GRACE study was to improve the care of ACS patients by identifying the
differences and relationships between treatment practices, patient characteristics and post ACS outcome across the world (Grace Investigators, 2001). The study involved 94 hospitals, over 14 countries and included 43,810 ACS patients (both STEMI and N-STEMI patients). The study generated multiple hypotheses and outcomes, one being the construction and validation of the GRACE score (Fox et al., 2006, Granger et al., 2003).

The GRACE score includes eight factors: age, clinical parameters (heart rate and systolic blood pressure at admission), a marker of renal function (serum creatinine), level of Killip class and on admission whether a cardiac arrest occurred, if ST-deviation was present and furthermore whether initial biochemical cardiac markers were raised.

Good discrimination of the GRACE model was found for STEMI patients when prospectively tested in a separate GRACE dataset (22,122) and additionally with an external ACS cohort (12,142) as part of the GUSTO IIb (global use of strategies to open occluded coronary arteries) study (GUSTO IIb angioplasty substudy investigators, 1997, Fox et al., 2006).

The GRACE ACS Risk Model calculator was used to calculate the GRACE scores and percentage probability of death or MI for the current study and can be found on the GRACE website (http://www.outcomes-umassmed.org/GRACE/acs_risk.aspx). Information for entry into the model calculator was collected at baseline from patient notes.

5.10.5 Co-morbidity assessment

Assessment of co-morbidity was undertaken with the use of a co-morbidity index. See section 3.5.2.2 for the full justification regarding the inclusion of co-morbidity as a variable in the study.

5.10.5.1 The Charlson Co-Morbidity Index

The Charlson Co-morbidity Index (CCI) was used in this study to measure both frequency and severity of co-morbid conditions at baseline (Charlson et al., 1987). The CCI was developed to enable a quantifiable means of assessing prognostic outcome due to co-morbidity for use in longitudinal studies. Three possible CCI scores can be calculated through the collection of information relating to co-morbidity. The three CCI scores include the weighted index of co-morbidity, the combined and age related score and the estimated ten year survival (estimated as a percentage) (Charlson et al., 1987, Charlson et al., 1994). A weighted index of
co-morbidity score of zero depicts the best possible outcome (death highly unlikely) and 33 is the worst possible outcome (almost certain death).

The CCI weighted index of co-morbidity was originally developed through a study involving the review of 559 medically ill patients’ hospital records. The number and seriousness of co-morbid conditions and the outcome of the patients within 12 months of their admission was used in combination to develop a weighted co-morbidity index (CCI) (Charlson et al., 1987). Nineteen co-morbid conditions were each assigned a weighted score; the weighted scores were summed to give an overall co-morbidity index score. The list of 19 co-morbidities and assigned weights, included in the CCI, can be seen in 0. The CCI was further evaluated in a cohort of 685 patients for its ability to predict death over a 10 year period. Age was also found to be predictive of mortality in the longer follow-up. The CCI estimated ten year survival (due to co-morbidity) score can also be calculated and is presented as a percentage. The combined condition and age-related score was evaluated in a study of 218 elective surgery patients who suffered from either hypertension or diabetes. Patients were followed for 5 years and their relative risk of death due to an increase of one on the CCI scale was equivalent to an increase in age of 10 years (Charlson et al., 1994).

The limitations of the CCI include some of the weighting assigned to conditions such as malignancies. In practice survival rates for lymphoma and leukemia have both low and high risk categories and these are not differentiated for in the CCI. Furthermore, treatment for AIDS and other conditions have now improved survival rates compared to when the CCI was first developed. However, at the time of conducting this study the version of the CCI used was the only version available.

For the purposes of this study it was decided to use the weighted index of co-morbidity score as it has been used widely as an indicator of severity of co-morbid illnesses in CHD cohorts even when mortality is not a primary outcome (Deaton and Thourani, 2009, Dickens et al., 2004, Cole et al., 2004). Furthermore, the CCI can be treated as a continuous variable. For the multiple regression model it was planned to include the CCI and also the GRACE score (see section 5.23.1.2 for details of the multiple regression model).

As part of the current study, information required to calculate the CCI was collected at baseline from the patients’ case notes and the data were verified with participants during the baseline telephone interview. An online CCI calculator designed by the Medical Algorithms Project (2001) at
http://www.medal.org/OnlineCalculators/ch1/ch1.13/ch1.13.01.php#result was used to calculate the scores for this study (Institute for Algorithmic Medicine, 2001).

5.11 Quantitative data analysis

5.11.1 Planned analysis

Data were analysed using SPSS™ statistical software package, release 15. Presentation and analysis of data using appropriate descriptive and inferential statistics were planned as described below.

5.11.2 Data handling

Data were collected according to the sources listed in Table 5-2 and were initially entered into an Excel spreadsheet (without identifying information). Categorical data were coded according to a priori coding and the values of continuous variables were entered into the spreadsheet. Once the data were cleaned (see section 5.11.3) the Excel spreadsheet was loaded into SPSS to undertake the analytical procedures.

A further Excel spreadsheet was maintained to manage study procedures (including participant identifiable data) relating to the date that STEMI occurred and also when consent was gained. Furthermore, the dates that questionnaires were posted and returned and telephone contact dates were also recorded; future dates for questionnaires to be sent out and telephone contact dates were also planned using this tool. All participant identifiable information was stored and maintained in accordance with the Research Governance Framework (Department of Health, 2005). The researcher was the identified responsible person for safe keeping of identifiable data and study documentation.

5.11.3 Data cleaning

Once data were entered into the Excel spreadsheet they were checked for missing items and inaccuracies. Patient notes, the sociodemographic pro-forma and patient questionnaires were revisited to answer data queries. A second stage of data cleaning took place once the Excel spreadsheet had been loaded into SPSS, checking that data were coded and value labels had been entered appropriately for use in SPSS.
5.11.4 Baseline and 6 month missing items

Missing demographic, baseline and 6 month data according to Table 5-2 were checked through multiple sources. Two sets of health records were checked for transfer patients and where data were still absent the patient’s GP was contacted and in some instances (where appropriate) information was checked directly with participants during the six month phone call.

When completed questionnaires were received back from participants they were immediately checked for missing items. When large areas of the questionnaire were missing the form was returned to the participant with the missing items marked and a request for the individual to complete the missing fields and to return the questionnaire once again. When only a few areas of the questionnaire were missing the participant was telephoned and asked for their responses to the questions over the phone. Telephone calls were completed by the researcher alone to ensure consistency of information interpretation.

The handling of missing data for the self-report questionnaires was dealt with where possible in accordance with the recommendations from the questionnaire manuals, the author or publisher of the questionnaire. The procedures used in this study for handling missing variables for the HADS and SAQ are detailed below.

5.11.5 HADS missing values

For the HADS questionnaire the manual did not give guidance on dealing with missing variables. However, recommendations on the publishers website (GL Assessment http://www.gl-assessment.co.uk/) identified that for a missing item from a sub-scale (anxiety or depression) the mean of the remaining six items (from that sub-scale) should be used for the missing item. The subscale should be treated as invalid if more than one item is missing (GL Assessment, 2011).

5.11.6 SAQ missing values

In the case of the SAQ sub-scales a detailed description of the scoring mechanism was obtained from the author of the questionnaire through the publishers’ website (CV outcomes http://cvoutcomes.org/) and was used for the purposes of this study (Spertus et al., 1995).

The missing SAQ values were dealt with differently for each sub-scale and the most complex scoring mechanism was for SAQ physical limitation, which is comprised of 9 questions (1a to 1i). The nine questions are grouped into 3 sets of 3 questions.
making up 3 levels of activity; the lowest level of activity includes showering, walking and dressing (questions 1a to 1c), medium activity involves walking round the block, gardening and climbing (questions 1d to 1f), the highest level of activity covers sports, running and lifting (1g to 1i). Each question is scored from 1 to 6 (6 relates to ‘limited or did not do for other reasons’ than angina) and all scores of 6 are classed as missing. When one answer is missing for an activity level, the mean of the other two scores is used for the missing answer. If two or more answers from either the lowest or highest activity scale, then the mean of the medium level activity score is used for all the missing answers. Where the medium level of activity has two or more missing answers then the mean of the lowest and highest scores are used for the missing answers. If there were four or more missing answers then the physical limitation score could not be calculated. It was therefore possible (due to the scoring mechanism counting all scores of 6 as missing for questions 1a to 1i) that participants could have a missing value for physical limitation.

The angina stability (question 2) sub-scale could not be calculated if the answer to the question was missing. For angina frequency (questions 3 and 4) if both answers to question 3 were absent it was classed as missing. Treatment satisfaction (questions 5, 6, 7 and 8) was classed as missing when less than 2 answers were present; the mean of ≥2 answers could be used to calculate a score when all answers were not present. Quality of life (QoL) questions 9, 10 and 11) is classed as a missing value when only one answer is available; the mean of 2 or more answers can be used to calculate the QoL score.

5.11.7 ESSI missing values

At the time of analysing the data for this study a manual was not available for scoring or dealing with missing values for the ESSI. Details of scoring for the ESSI were taken from the ENRICHD study publications (see section 5.10.3.1) (Mitchell et al., 2003, ENRICHD Investigators, 2003). However, a description of how to address missing values was not included in the ENRICHD publications. The author Mitchell, who was responsible for publishing details of the ESSI on behalf of the ENRICHD Investigators, was contacted to gain information on dealing with missing variables; she replied by email on 7th March 2011 recommending that missing items should be scored as zero.
5.12 Study cohort

To allow the findings of the study to be generalised to the wider population (STEMI patients receiving PPCI), it was important to obtain a detailed description of the study cohort. It was also necessary to establish whether the characteristics of the cohort reflected those of the wider population.

The study cohort (as a whole) characteristics were summarised using the baseline and 6 month descriptive and comparison results. Furthermore, the number of patients recruited from the total available population during the recruitment period was identified. Details of why participants were not approached or included in the study (including details of eligibility to participate) were also recorded. Ideally the characteristics of those who were recruited with those who were not would have been compared to establish any differences between the recruited and not recruited patients. However, this was not possible because access to the personal data of those who were not recruited was not permitted because consent had not been obtained.

5.12.1 Responders

The number of responders was defined by the number of individuals approached and consented at baseline who went on to return the baseline questionnaires. Those who gave consent but did not return baseline questionnaires were classed as non-responders. Baseline demographics, PPCI admission details and whether individuals re-presented (with a potential IHD event) were recorded descriptively for both groups. This enabled contextual information to be assessed for both groups.

5.13 Continuous variables

All continuous variables were presented as raw scores, means, standard deviations, medians and ranges. The distribution was also assessed in accordance with procedures outlined in section 5.14.1. The normality assumptions were used to establish the most appropriate statistical tests to assess the null hypotheses for the study aims.

5.13.1 Normality analysis for continuous data

The distribution of continuous data was assessed to establish if the data were normally distributed (Field, 2009). Initially the mean and median were compared; these should be similar when the distribution is symmetrical, which is a property of
a normal distribution. A similar mean and median do not guarantee normality, but substantially different values suggest that the distribution is not normal.

A histogram was used to assess normality graphically. The centres of the tops of the bars on a histogram should follow a bell shaped curve for normally distributed data; tails extending mainly to the right or mainly to the left indicate positively or negatively skewed data respectively. Outliers were identified using a box plot and all outliers were investigated to ensure that errors had not occurred in data transcription or data entry.

The sample skewness was used to estimate symmetry about the mean; the sample kurtosis was used to assess the clustering of scores in the tails together with the sharpness of a central peak (Field, 2009). Positively skewed distributions have the majority of scores at the low end of the scale with a longer tail pointing towards the higher scores (or more positive scores). Negatively skewed distributions have more scores at the higher end of the scale and a longer tail at the lower end (or more negative scores). Kurtosis indicates the occurrence of clustering of scores in the tails and also the sharpness of a central peak (Pett, 1997). A positive kurtosis is known as a leptokurtic distribution (heavy tailed distribution), has a high degree of scores in the tails and a sharper central peak than the bell curve of a normal distribution. A negative kurtosis corresponds to a platykurtic distribution (light tailed distribution) with thin tails and is flatter at the centre than a normal distribution (Field, 2009). When both skewness and kurtosis are close to zero then data are likely to be normally distributed. When either or both are further from 0, data are unlikely to be normally distributed. Furthermore, when the magnitude of the sample skewness is at least twice as large as its standard error, then the distribution can be considered to be non-normal (Pett, 1997). The same is true of kurtosis: if the magnitude of the sample kurtosis is at least twice as large as its standard error, then the distribution can be considered to be non-normal (Pett, 1997).

The Shapiro-Wilk (S-W) normality test was also used to check the distribution of the data. This test was chosen because the re-presentation group was small [<50 (n=37)] and the test is more powerful for small samples (Shapiro and Wilk, 1972). Pett (1997) advised that evidence from the histogram, box plot, skewness and kurtosis, and the Shapiro-Wilk test be considered together when deciding whether or not a distribution is likely to be normal.
5.13.2 Hypothesis testing for continuous variables

When it was assumed that the continuous data were at least approximately normally distributed in each group, a two-sided unpaired t-test was used to compare means in two independent groups (Bland, 2000). The t-test tests hypotheses about population means. A two-sided test allows the null hypothesis to be tested against an alternative hypothesis with either a positive or a negative difference. Furthermore, an unpaired t-test is conducted for two unrelated groups at the same point in time Table 5-5. Associated with the t-test, a 95% confidence interval for the difference between the two means was estimated (Bland, 2000). If it could not be assumed that the data were approximately normally distributed in each group, then a two-sided Mann-Whitney U test was used (Clarke-Carter, 1997). The Mann-Whitney U test tests hypotheses about population distributions rather than means (Table 5-5). However, Moore and McCabe (1993, page 538) suggest that if the total sample size is ≥40 it is acceptable to treat data as if it were normally distributed and use t-tests and 95% confidence intervals for differences between means. This is justified because the larger sample size leads to sample means behaving like normally distributed data as a result of the Central Limit Theorem (Moore and McCabe, 1993). Description of statistical tests in relation to the characteristics analysis and the study aims can be seen in Table 5-5.

Table 5-5 Tests of association between continuous variables for Aims 2 to 5

<table>
<thead>
<tr>
<th>Type of assessment</th>
<th>Type of variable</th>
<th>Measurement level</th>
<th>Parametric</th>
<th>Non-parametric</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preliminary analysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dependent</td>
<td>Dichotomous</td>
<td>Unpaired t test</td>
<td>Mann Whitney-U</td>
</tr>
<tr>
<td></td>
<td>Independent</td>
<td>Continuous</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Analysis of study aims 2 to 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aim 2</td>
<td>Between groups comparison</td>
<td>Independent</td>
<td>Continuous</td>
<td>Unpaired t test</td>
</tr>
<tr>
<td>Aim 3</td>
<td>Within group</td>
<td>Independent</td>
<td>Continuous</td>
<td>Paired t test</td>
</tr>
<tr>
<td>Aim 4</td>
<td>Change between groups</td>
<td>Independent</td>
<td>Continuous</td>
<td>Analysis of covariance</td>
</tr>
<tr>
<td>Aim 5</td>
<td>Association of variables within groups</td>
<td>Independent</td>
<td>Continuous</td>
<td>Pearson’s product-moment correlation</td>
</tr>
<tr>
<td>Aim 6</td>
<td>Association multivariate analysis</td>
<td>Independent</td>
<td>Continuous</td>
<td>Logistic regression</td>
</tr>
</tbody>
</table>

In the instance that data in this study were strongly skewed, both parametric and non-parametric analyses were conducted. The purpose of conducting both
parametric and non-parametric analysis would enable cross referencing of analytical results leading to a more thorough analysis. Results of the analyses were then interpreted in relation to clinical relevance.

5.14 Categorical variables

Categorical data were described in terms of counts and percentages.

5.14.1 Hypothesis testing for categorical variables

To conduct hypothesis testing for a dichotomous or nominal variable between groups Pearson’s Chi-Squared test was used (Bland, 2000). However if the percentage of cells with expected counts (under the null hypothesis) <5 is 20% or more, or the expected count in one cell is <1.0, then Fisher’s exact test was used (Bland, 2000). Where categories were ordered the Chi-Square test for trend was. Hypothesis testing for dichotomous variables within groups but between time points was conducted using McNemar’s test, with results presented in a 2 X 2 contingency table (Clarke-Carter, 1997). Table 5-6 lists the tests of association for the categorical variables throughout the quantitative results presented in Chapter 7.

Table 5-6 Tests of association between categorical variables for the characteristics analysis and aims 1 to 3

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Type of variable</th>
<th>Measurement level</th>
<th>Test</th>
<th>Alternative test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preliminary analysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline comparison</td>
<td>Between groups</td>
<td>Independent</td>
<td>Categorical</td>
<td>Pearson’s Chi-Squared test</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dependent</td>
<td>Dichotomous</td>
<td>Fisher’s exact test</td>
</tr>
<tr>
<td></td>
<td>Independent</td>
<td>Categorical (ordered)</td>
<td>Chi-Square test for trend</td>
<td>n/a</td>
</tr>
<tr>
<td></td>
<td>Dependent</td>
<td>Dichotomous</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Analysis of study aims 1 to 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aim 1</td>
<td>Descriptive</td>
<td>Independent</td>
<td>Categorical</td>
<td>n/a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dependent</td>
<td>Dichotomous</td>
<td></td>
</tr>
<tr>
<td>Aim 2</td>
<td>Between groups (cut-offs)</td>
<td>Independent</td>
<td>Categorical</td>
<td>Pearson’s Chi-Squared test</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dependent</td>
<td>Dichotomous</td>
<td></td>
</tr>
<tr>
<td>Aim 3</td>
<td>Between groups (cut-offs)</td>
<td>Independent</td>
<td>Categorical</td>
<td>Pearson’s Chi-Squared test</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dependent</td>
<td>Dichotomous</td>
<td>McNemar’s test</td>
</tr>
<tr>
<td></td>
<td>Within groups (cut-offs)</td>
<td>Dependent</td>
<td>Dichotomous</td>
<td></td>
</tr>
</tbody>
</table>

Additionally, 95% confidence intervals (CI) were calculated for comparisons between groups for the dichotomised HADS scores anxiety and depression ≥8 and ≥11 and psychological distress ≥17. An Excel spreadsheet containing calculations prepared by Professor R Newcombe (2011) of The university of Cardiff, was used to calculate the 95% CI.
5.15 Analysis of baseline variables

A range of variables were analysed at baseline to identify the characteristics of the study cohort (a full list of variables can be found in Table 5.2). Analytical procedures for continuous and categorical variables are described in sections 5.13 and 5.14 respectively and were adhered to for the baseline analysis. Results for the study cohort as a whole and for the re-presentation and non-representation groups separately were presented accordingly.

5.16 Anxiety and depression

5.16.1 Measurement

Indicative levels of anxiety and depression were measured using the raw HADS anxiety and depression scores and two different dichotomised scores (HADS anxiety cut off and depression cut off) (Zigmond and Snaith, 1983). The two dichotomised scores for each sub-scale included 1) $\geq 8$ indicating raised (mild or worse) levels, 2) $\geq 11$ indicating moderate as a minimum level of anxiety or depression (Zigmond and Snaith, 1983). An indicative level of overall psychological distress was measured using the raw total HADS psychological distress score (HADS anxiety score + HADS depression score); a dichotomised score where a value $\geq 17$ is considered suggestive of caseness (Dickens et al., 2004) (see section 5.10.1.3 for details of HADS scoring).

5.16.2 Preliminaries

The HADS anxiety, depression and psychological distress (total) scores were calculated for each participant at baseline and at 6 months in SPSS by summing the appropriate items. SPSS was then used to calculate the corresponding dichotomised scores for each participant using the chosen cut-offs (see 5.10.1.3).

Change scores for the raw scores were calculated by subtracting the baseline score from the corresponding 6 month score for each participant. The distributions of the change scores for HADS anxiety, HADS depression and HADS psychological distress were assessed for normal distributions in accordance with procedures described in section 5.13.1.
5.17 Angina symptoms

5.17.1 Measurement

The raw scores and change scores for the five Seattle Angina Questionnaire (SAQ) (Spertus et al., 1995) sub-scales (physical limitation, angina stability, angina frequency, treatment satisfaction and quality of life related to CHD) were calculated to assess participants’ experiences of angina symptoms (see section 5.10.2.3). SAQ subscale scores were calculated using SPSS syntax prepared by Dr Malcolm Campbell, Lecturer in Statistics, The University of Manchester, from the scoring algorithms (CVoutcomes., 2010). The SAQ sub-scales are all scored separately, on the scale 0-100 with higher scores indicating better health status; there is no total SAQ score. Change scores were calculated as sub-scale scores at 6 months minus sub-scale scores at baseline.

The SAQ change scores were categorised for each participant according to SAQ cut-offs, i.e. whether scores remained essentially the same, or changes by at least (+/-) 10. This was where a change of at least +/-10 in the SAQ sub-scale score was considered to demonstrate a clinically important change. The three categories were: worsening of individual SAQ score (change of \( \leq -10 \)), SAQ score remains the same (change of \( > -10 \) to \( < +10 \)) or an improvement in SAQ score (change of \( \geq +10 \)) (Spertus et al., 1995).

5.17.2 Preliminaries

All SAQ sub-scale total scores were calculated for each participant at baseline and at 6 months in SPSS as described in section 5.17.1. Change scores were calculated for each participant by subtracting the baseline score for the participant from the corresponding 6 months score. The distributions of the change scores for each of the five SAQ sub-scales were assessed for normal distributions (see section 5.13.1).

The mean, standard deviation, median and interquartile range were estimated for the raw and change scores for the re-presentation and non-representation groups. The numbers and percentages whose categorised scores changed to a better category, stayed the same category or changed to a worse category from baseline to 6 months were estimated for all participants and in the re-presentation and non-representation groups.
5.18 **Aim 1 plan of analysis: Number and frequency of re-presentations**

Aim 1: To identify the number and frequency of re-presentations due to IHD symptoms, by determining and categorising the reasons for re-presentation during the first 6 months post STEMI.

The number and percentage of participants who re-presented or did not re-present during the 6 months since STEMI were identified. Individuals were categorised in relation to their final discharge diagnosis. A final list of categories relating to the cause of re-presentation was summarised.

From the final discharge diagnoses participants were allocated to the potential IHD 're-presentation group' or the 'non-representation group'. These will be referred to as 'presentation groups' in the remainder of the thesis. The number of acute re-presentations that each individual had experienced was reported.

5.19 **Aim 2 plan of analysis: Levels of psychological health and angina symptoms between presentation groups**

Aim 2: To determine and compare the level of anxiety, depression, psychological distress and angina symptoms (physical limitation, angina stability, angina frequency, treatment satisfaction and QoL) between the two groups at baseline and then at 6 months.

5.19.1 **Raw scores for HADS and SAQ**

The mean, standard deviation, median and the range of the HADS (anxiety, depression and psychological distress) and SAQ (physical limitation, angina stability, angina frequency, treatment satisfaction and QoL) sub-scale scores were estimated for the raw scores at baseline and 6 months for the groups. The distribution was also determined for the HADS and SAQ sub-scales for each group in accordance with procedures discussed in section 5.13.1. The null hypothesis was tested at the 5% significance level to assess whether there was a difference between the groups.

**Null hypothesis:** There is no difference in the individual HADS and SAQ sub-scale scores between the presentation groups at baseline. The null hypothesis was re-tested at 6 months.
Each HADS and SAQ sub-scale scores was compared at baseline and then at 6 months between the re-presentation and non-representation groups. When data were at least approximately normally distributed in each group, a two-sided unpaired t-test was used (Bland, 2000). If it could not be shown that data were normally distributed, then a two-sided Mann-Whitney U test was used additionally to the unpaired t-test in a sensitivity analysis (Clarke-Carter, 1997). The results of the analysis were then interpreted in relation to clinical relevance.

5.19.2 HADS cut offs

The numbers and percentage of individuals that fell above and below the HADS sub-scale cut-offs (anxiety and depression <8, ≥8 and <11, ≥11 and psychological distress <17, ≥17) were determined at baseline and 6 months for the groups. The null hypothesis was tested at the 5% significance level to assess whether there was a difference between the groups.

**Null hypothesis:** There is no difference in HADS sub-scale dichotomised scores (anxiety and depression <8, ≥8 and <11, ≥11 and psychological distress <17, ≥17), between the groups at baseline and 6 months.

Comparison between the groups for the dichotomised HADS scores (anxiety and depression <8, ≥8 and <11, ≥11 and psychological distress <17, ≥17) were conducted using Pearson’s chi-square test or Fisher’s exact test (Bland, 2000).

5.20 Aim 3 plan of analysis: Change in levels of psychological health and angina symptoms

Aim 3: To determine the change in the level of anxiety, depression and psychological distress and angina symptoms within the groups from baseline to 6 months.

The mean, standard deviation, median and range were estimated for each of the HADS and SAQ sub-scale change scores in the groups. The distributions of the HADS and SAQ sub-scale change scores (for both groups) were assessed for normal distributions. Comparison of HADS and SAQ sub-scale scores from baseline to 6 months were compared within each of the groups using the following null hypothesis at the 5% significance level.

**Null hypothesis:** There is no difference in HADS and SAQ sub-scale scores from baseline to 6 months within the groups.
When it was found that the baseline and 6 month HADS and SAQ sub-scale scores were at least approximately normally distributed in each group, a two-sided paired t-test was used, with an associated 95% confidence interval for the difference in means. If it could not be shown that the change scores were normally distributed in each group, then a Wilcoxon matched-paired signed-ranks test was used in addition to the paired t-test in a sensitivity analysis.

5.20.1 HADS and SAQ cut offs
The number and percentage of each HADS cut off (anxiety and depression <8, ≥8 and <11, ≥11 and psychological distress <17, ≥17) from baseline to 6 months for each group were estimated.

SPSS was used to determine whether SAQ sub-scale scores that changed by at least +/-10 or stayed the same (i.e. changed by less than 10). The numbers and percentages of the SAQ sub-scale categorised scores (changed to a better category, stayed the same category or changed to a worse category) from baseline to 6 months were estimated for the groups. Comparison of SAQ and HADS cut offs from baseline to 6 months were compared using the following null hypotheses.

**Null hypothesis:** There is no change in HADS and SAQ sub-scale cut off scores from baseline to 6 months within the groups.

Comparison within the groups for the dichotomised HADS scores (anxiety and depression <8, ≥8 and <11, ≥11 and psychological distress <17, ≥17) and categorical SAQ scores (changed +/-10 or stayed the same) were conducted using Mc-Nemar’s test for related categorical variables. The McNemar test is used when looking for change in a dichotomous variable for related data (Field, 2009). The McNemar-Bowker test is an extension to look for change in a nominal variable for related data (Bowker, 1948).

5.21 Aim 4 plan of analysis: Comparing levels of change in psychological health and angina symptoms between the groups

**Aim 4:** To compare the change in anxiety, depression, psychological distress and angina symptoms, between the two groups at 6 months.

The mean, standard deviation, median, range and distribution for the HADS and SAQ sub-scale change scores were established in aim 3 and used as a basis for the
null hypotheses. Changes in HADS and SAQ sub-scales at 6 months were compared for the groups using the following null hypotheses.

**Null hypothesis:** There is no difference in change in the HADS and SAQ sub-scale scores at 6 months between the re-presentation and non-representation groups.

The raw scores at 6 months were compared between groups (adjusted for raw scores at baseline) using analysis of covariance (ANCOVA). ANCOVA was chosen because it is a more powerful test than a two-sided unpaired t-test, adjusts for baseline imbalance and allows for testing of the time effect (Field, 2009).

### 5.22 Aim 5 plan of analysis: Associations of psychological health and angina symptoms within the groups

**Aim 5:** To determine the association between the levels of anxiety, depression, psychological distress and angina symptoms within each group at baseline and then at 6 months.

At baseline and 6 months correlations were estimated between the HADS sub-scales and the SAQ sub-scales; results were presented in the form of a correlation matrix for each group. All variables included in this analysis could be treated as continuous with correlation as an appropriate measure of association. The significance of individual correlations was assessed using a two-sided test of the following null hypothesis.

**Null hypothesis:** There were no associations in levels of anxiety, depression, psychological distress and angina symptoms for either group at baseline. The null hypothesis was also tested at 6 months.

When data were at least approximately normally distributed, the parametric Pearson’s product-moment correlation was estimated. However, if data were highly skewed, Kendall’s tau $\tau_b$ correlation was estimated. Correlations were estimated separately at baseline and 6 months.

The value of the correlation lies between -1 and +1. When the coefficient is zero, as one variable changes the other will stay the same because there is no relationship between the two variables. Conversely, a coefficient +1 indicates a perfect positive correlation (both values increase proportionately) and a coefficient of -1 signifies a perfect negative correlation (as one value increases the other value decreases).
proportionately) (Field, 2009). The correlation coefficient is a standardised measure and for Pearson’s correlation, the effect size can be measure with the values +/-0.1 indicating a small effect, +/-0.3 a medium effect and +/-0.5 a large effect (Field, 2009, page 170).

5.23 Aim 6 plan of analysis: Association of psychological health, angina symptoms, physiological health and re-presentation

Aim 6: To determine the association of psychological health (anxiety, depression and psychological distress), angina symptoms (stability, frequency and physical limitation) and physiological health (severity of STEMI and comorbidity) with re-presentation due to potential IHD symptoms, adjusted for other expected contributing or confounding factors previously identified in the conceptual model.

5.23.1 Regression modelling

Regression analysis sets out to estimate the association between one or more independent variables and a dependent variable. Galton (1886) developed the regression technique to investigate whether there was a relationship between heights of parents and their children (Galton, 1886). Regression models are used to investigate how well one or more variables predict another variable (Field, 2009). A model is fitted to data to predict the values of an outcome variable from one or more predictor variables (Bland, 2000).

5.23.1.1 Logistic regression modelling

Logistic regression modelling was chosen because the dependent or outcome variable re-presentation was dichotomous. The goal of logistic regression analysis is the same as any other model building technique in statistics, which is described by Hosmer and Lemeshow (1989):

“to find the best fitting and most parsimonious, yet biologically reasonable model to describe the relationship between an outcome (dependent or response) variable and a set of independent (predictor or explanatory) variables” (page 1).

The logistic regression model was used to address the following null hypothesis.

Null hypothesis: Patients who re-presented with potential IHD symptoms, during the first 6 months following STEMI and PPCI treatment, would not have higher levels of anxiety, depression, psychological distress and/or levels of angina
symptoms and/or levels of physiological burden at baseline, compared to those who did not re-present with potential IHD symptoms.

5.23.1.2 Inclusion of variables in the regression model

Previously the conceptual model has been discussed in detail in Chapter 3. It would have been most appropriate to include all the factors described in that model in a logistic regression model for this study. However, due to the reduced number of participants who both re-presented and returned baseline questionnaires (n=38) it was not possible to include all the conceptual variables in the final model.

It is recommended by Peduzzi et al (1996) that for reliable estimation, the ratio of individuals to predictor variables should be at least 10:1 for each of the outcome categories. The smaller outcome category, re-presentation, only had 38 individuals and therefore it is possible that the inclusion of more than 4 predictor variables may result in poor estimation in the model (Peduzzi et al., 1996). However, Vittinghoff and McCulloch (2007) suggest that the general rule of 10:1 (outcome variables per predictor variable) can be relaxed. They state that discounting statistically significant associations from models with 5 to 9 predictor variables is not warranted (Vittinghoff and McCulloch, 2007). Vittinghoff and McCulloch (2007) suggest a compromise between obeying the rule of Peduzzi et al (1996) strictly (including only 4 variables) and the inclusion of the most important variables from the conceptual model.

The conceptual model included three important concepts that underpin the model, symptoms, psychological health and physiological health; at least one variable from each category was chosen for inclusion in the final model. The final list of variables was chosen on theoretical grounds from the literature, and is described below in Table 5-7.
Table 5.7 Description of variables included in the logistic regression model.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Justification for inclusion in the model</th>
<th>Variable Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Re-presentation</td>
<td>Re-presentation due to potential IHD symptoms is the concept under investigation in this study.</td>
<td>Outcome</td>
</tr>
<tr>
<td><strong>Psychological variables at baseline</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety (HADS)</td>
<td>Anxiety has been reported as an important factor during A&amp;E attendances for chest pain and ACS events (Grace et al., 2004). Qualitative data (see Chapter 8) from the current study also demonstrated that participants felt a great deal of anxiety during recovery. Anxiety may therefore be a predictor for re-presentation.</td>
<td>Predictor</td>
</tr>
<tr>
<td>Depression (HADS)</td>
<td>Depression is a known phenomenon in patients post STEMI (Frasure-Smith et al., 1995b). Anxiety and depression are interlinked and symptoms can be similar to those of cardiac ischaemia (Dammen et al., 2004, Bech, 2006). Depression has therefore been incorporated as a predictor variable.</td>
<td>Predictor</td>
</tr>
<tr>
<td>Psychological distress (HADS)</td>
<td>Psychological distress is the sum of HADS anxiety and depression scores and is therefore useful in summarizing the participants psychological health (Dickens et al., 2004).</td>
<td>Predictor</td>
</tr>
<tr>
<td><strong>Symptom variables at baseline</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angina frequency (SAQ)</td>
<td>The occurrence of potential IHD symptoms (e.g. angina) drives the participants to re-present in this study. It is possible that in the current study more frequent occurrence of angina at baseline may lead to re-presentation (Grace et al., 2005b). Due to the unpredictable nature of the re-presentation it was not possible to measure angina frequency at the time of re-presentation in the current study. However, angina frequency was measured at baseline and could therefore be included as a predictor for re-presentation.</td>
<td>Predictor</td>
</tr>
<tr>
<td>Angina stability (SAQ)</td>
<td>Individuals with instability of their angina symptoms may be more likely to seek help through acute healthcare services (Grace et al., 2004, Spertus et al., 2002). For pragmatic reasons, SAQ angina stability was recorded at baseline rather than at the time of re-presentation.</td>
<td>Predictor</td>
</tr>
<tr>
<td><strong>Physiological variables at baseline</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STEMI outcome (GRACE)</td>
<td>It is known that some MI patients experience repeat cardiac events following MI (Jakobsen et al., 2010, Andersen et al., 2003). The GRACE score is used widely as a composite clinical risk prediction tool (for death or, death and MI) for ACS patients from admission to 6 months (Fox et al., 2006, National Institute for Health and Clinical Excellence, 2010a). It has been adopted as a measure of severity of STEMI in the model. The GRACE is treated as a predictor variable in the current study.</td>
<td>Predictor</td>
</tr>
<tr>
<td>Co-morbidity (CCI)</td>
<td>Certain symptoms related to some physiological comorbidities (e.g. of a pulmonary, gastroenterological or musculoskeletal nature) are similar to those associated with IHD (Dammen et al., 2004, Ros et al., 1997). Representation may therefore be due to co-morbidity (measured by the CCI) rather than IHD or psychological factors. An assumption that re-presentation was purely due to IHD or psychological factors may have led to biased findings. The CCI is a quantifiable, reliable measure of co-morbidity (Buntinx et al., 2002, Charlson et al., 1987) and was included in the model as a predictor variable.</td>
<td>Predictor</td>
</tr>
</tbody>
</table>
The evidence in Table 5-7 suggested the following list of independent variables for inclusion in the model: HADS anxiety, HADS depression, HADS psychological distress, SAQ angina frequency, SAQ angina stability, the GRACE score and the CCI. All were deemed to be predictor variables.

5.23.1.3 Assessing multicollinearity

Initially multicollinearity of variables to be entered into the model was assessed. It was possible for a variable included in the model to interfere with another variable if the two variables shared a strong linear association (Field, 2009). A strong linear association between variables can lead to an unstable model that is not robust to minor changes in the data (Stevens, 2009).

Multicollinearity can be assessed with the use of eigenvalues, the Variance Inflation Factor (VIF), the condition index and variance component (Field, 2009).

When all the variables included in the model are plotted on a matrix, the eigenvalues demonstrate the dimensions of the data and whether the variances of the matrix are distribution evenly (Field, 2009). When there is no correlation between variables then the eigenvalues will be the same and if the eigenvalues are similar then small changes in the variables are unlikely to affect the model.

Field (2009, page 224) states that the VIF indicates whether a predictor has a strong linear relationship with the other predictor(s). When the VIF for a variable in the model is >10 there is an indication of multicollinearity (Myers, 1990). Field (2009) describes the condition index as the square root of the ratio of the largest eigenvalue to the eigenvalue of interest. Norusis (2006) suggests that a condition index greater than 15.0 indicates a possible unstable model and an index greater than 30.0 indicates a serious problem with instability. The variance component (also known as variance proportion) indicates the proportion of variance of the regression coefficient for every predictor and its eigenvalue (Field, 2009). Norusis (2006) indicated that the variance components are not directly linked to the condition index, but if a high proportion of the variance of two or more coefficients (i.e. of different variables) is associated with the same eigenvalue and that eigenvalue is very small, there is some evidence of multicollinearity.

Further investigation showed that there was some suggestion of collinerarity between HADS anxiety and HADS depression in accordance with Field’s (2009) guidance. Field (2009) describes how the matrix of scaled uncentred cross-products of variables can be analysed using principal component analysis to give a series of
eigenvalues representing the standardised variances associated with the principal components. Field notes that if the eigenvalues are quite similar, then multicollinearity is unlikely, while if the eigenvalues are very different, small changes in the data may result in marked changes in the regression model. Differences between the eigenvalues are calculated in the form of a condition index, which is the square root of the ratio of the largest eigenvalue to the current eigenvalue. Norusis (2006) reports a rule-of-thumb that a condition index greater than 15.0 may suggest an unstable model while one greater than 30.0 indicates serious problems. Field (2009) and Norusis (2006) then recommend examining the proportions of variance of the regression coefficients associated with each principal component: variables that have high proportions of variance on the same component where the eigenvalue is very small are considered to show evidence of collinearity.

5.23.1.4 Univariable logistic regression models
The predictors HADS anxiety, depression and psychological distress and SAQ angina stability and angina frequency were all entered into univariate logistic regression models, to ascertain whether the models were significant and if there was significance in terms of association with re-presentation. This enabled identification of the most stable and appropriate predictor to be included in the model.

The earlier assessment of collinearity indicated some evidence of a relationship between HADS anxiety and HADS depression at baseline. As an additional assessment of the potential interaction between the predictors HADS anxiety and HADS depression, the two variables were included together in the same logistic regression model without the other predictor variables.

To then assess further the level of interaction between predictors HADS anxiety and HADS depression, the standard errors (SE) of the estimates of the regression coefficients of anxiety and depression (the Bs) were examined for models with the predictors considered separately and combined. An increase of the standard error when anxiety and depression were combined and a change in the coefficients (before and after combining the two variables) indicated an interaction between the variables.
5.23.2 **Multiple logistic regression models**

The predictor variables included in the logistic regression models (model 1, model 2 and model 3) discussed in this section of the thesis are described in Table 5-8.

Table 5-8 Description of predictor variables included in model 1, model 2 and model 3

<table>
<thead>
<tr>
<th>Model number</th>
<th>Predictors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td>HADS anxiety</td>
</tr>
<tr>
<td></td>
<td>HADS depression</td>
</tr>
<tr>
<td></td>
<td>SAQ angina stability</td>
</tr>
<tr>
<td></td>
<td>SAQ angina frequency</td>
</tr>
<tr>
<td></td>
<td>GRACE</td>
</tr>
<tr>
<td></td>
<td>CCI</td>
</tr>
<tr>
<td>Model 2</td>
<td>HADS psychological distress</td>
</tr>
<tr>
<td></td>
<td>SAQ angina stability</td>
</tr>
<tr>
<td></td>
<td>SAQ angina frequency</td>
</tr>
<tr>
<td></td>
<td>GRACE</td>
</tr>
<tr>
<td></td>
<td>CCI</td>
</tr>
<tr>
<td>Model 3</td>
<td>HADS anxiety</td>
</tr>
<tr>
<td></td>
<td>SAQ angina stability</td>
</tr>
<tr>
<td></td>
<td>SAQ angina frequency</td>
</tr>
<tr>
<td></td>
<td>GRACE</td>
</tr>
<tr>
<td></td>
<td>CCI</td>
</tr>
</tbody>
</table>

5.23.2.1 **Model 1**

The initial model (model 1) included six predictor variables listed in Table 5-8. The decision to include six variables was made in accordance with Vittinghoff and McCulloch (2007) suggestion of including the most important variables on conceptual grounds and compromising on Peduzzi et al (1996) rule of only including four variables. The theoretical justification for inclusion of the variables in model 1 can be seen in Table 5-7 and the theoretical underpinning of these variables is discussed in detail in the conceptual model (Chapter 3).

Assessment of multicollinearity did not overtly demonstrate that there was a problem with the inclusion of both HADS anxiety and HADS depression in the same model. The Nagelkerke R squared was assessed in terms of its closeness to zero, to check the goodness-of-fit of the model (zero indicates a poor fit of the model) and the amount of variation explained (Nagelkerke, 1991). The odds ratios, their significance and the overall significance of the model were also determined to assess whether anxiety or depression played a role in the re-presentation of participants.
5.23.2.2 Model 2
A second alternative model (model 2) was considered using HADS psychological distress in place of HADS anxiety and HADS depression and included the remaining four variables (see Table 5-7).

The Nagelkerke R squared was assessed in terms of its closeness to zero to check the goodness-of-fit of the model and the amount of variation explained (Nagelkerke, 1991). The odds ratios, their significance and the overall significance of the model were determined to assess whether psychological distress played a role in the re-presentation of participants.

5.23.2.3 Model 3
A final model (model 3) was developed using HADS anxiety as the psychological predictor with the additional inclusion of predictors SAQ angina stability, SAQ angina frequency, the GRACE and the CCI.

The Nagelkerke R squared was again assessed in terms of its closeness to zero to check the goodness-of-fit of the model and the amount of variation explained (Nagelkerke, 1991). The odds ratios, their significance and the overall significance of the model were determined to assess whether anxiety played a role in the re-presentation of participants.

5.24 Summary
In this quantitative methods chapter, the aims and hypotheses for the quantitative aspects of this mixed methods study are presented. Information relating to the sample size calculation and the inclusion and recruitment of participants are described.

The procedures for collecting re-presentation data are described. Furthermore, the justification for the choice of study instruments including reliability and validity information are also discussed. At baseline and 6 months post STEMI the Hospital Anxiety and Depression Scale (HADS) was chosen for measuring the psychological health of participants and the Seattle Angina Questionnaire (SAQ) score was selected to measure angina symptoms. Physiological health in the form of STEMI outcome (at 6 months) and baseline co-morbidity were measured using the ‘Global Registry of Acute Coronary Events’ GRACE score and the Charlson Co-morbidity Index (CCI) respectively.
The plan of analysis to answer the study aims included the use of descriptive, univariate analysis and multivariate analysis. Mean, median, standard deviation and normality tests were presented for all continuous data. Hypothesis testing for continuous variables within the groups (baseline to 6 months) included the use of two sided paired t-test for normally distributed data and Mann-Whitney U test for non-normally distributed data. Between group comparisons were conducted using the unpaired t-test for normally distributed data and the Mann-Whitney U test for non-normally distributed data. Categorical data were presented in terms of counts and percentages and hypothesis testing was undertaken using Pearson’s Chi-Squared test or Fisher’s exact test.

Logistic regression modelling was used to assess which predictor variables played a role in the re-presentation of participants. Preliminary analysis identified an interaction between HADS anxiety and HADS depression that led to the variables cancelling each other out when included together in the same model (without additional variables).

Three final logistic regression models were developed. The first (model 1) included HADS anxiety and HADS depression, SAQ angina stability and SAQ angina frequency, the GRACE and the CCI as predictors variables. A second model (model 2) included predictors HADS psychological distress, SAQ angina stability, SAQ angina frequency the GRACE and the CCI. A final model (model 3) included HADS anxiety as the psychological predictor with the additional inclusion of predictors SAQ angina stability, SAQ angina frequency, the GRACE and the CCI.
CHAPTER 6 QUALITATIVE METHODS

6.1 Introduction

This chapter describes the methods undertaken to conduct the qualitative aspect of this mixed methods study. The study purpose, study design and recruitment of participants are described. Methods used for data collection and data analysis are also discussed.

6.2 Qualitative study

6.2.1 Purpose of the qualitative study

The purpose of the qualitative study was to explore the experiences of patients who re-present with Ischaemic Heart Disease (IHD) symptoms within 6 months post STEMI. The patients’ perception of their symptoms which contributed to their re-presentation, and their reasons for seeking help were explored. Furthermore, their experiences of subsequent investigations and treatments during re-presentation were ascertained. Emotional responses associated with their experience were elicited.

6.2.2 Qualitative study design

The qualitative study design was based on modified grounded theory, which incorporated concurrent sampling, data collection and data analysis, and constant comparative analysis (Charmaz, 2006). Data collection was conducted using a single in-depth, semi-structured, face to face interview, with STEMI patients who had re-presented within the first 6 months post STEMI.

Modified grounded theory enabled exploration of the emerging concepts through both inductive and deductive reasoning for the relatively new phenomenon PPCI (Bowling, 1997). ‘Induction’ relates to the development of concepts directly from the data often leading to hypotheses or theory, whereas ‘deduction’ involves preconceived ideas or theory and may involve a priori hypothesis (Pope et al., 2000). Full details of the qualitative study design and how modified grounded theory was applied to this study can be found in sections 4.10.

6.2.3 Participants

Participants for the qualitative study were identified through the quantitative phase of this explanatory mixed methods study (Creswell and Plano Clarke, 2007).
mixed methods study design is discussed in section 4.6. Briefly, the quantitative part of the study was conducted prior to the qualitative study this process enabled the identification of individuals who had re-presented following their heart attack. Once identified, individuals from this cohort were then purposefully selected using ‘maximum variation’ sampling (Patton, 1990) for participation in the qualitative phase of the study (section 4.7).

6.2.4 Qualitative study sampling

The intention of using purposeful sampling in this study was to explore the experiences of individuals who had first hand knowledge of the phenomenon under study, in this case re-presentation. Purposeful sampling uses the varied characteristics of different individuals as a means of selecting participants (Wilmot, 2005). Patton (1990) suggests that purposeful sampling enables in-depth exploration of information rich cases.

Maximum variation sampling was used to identify participants for this study (Patton, 1990). This type of purposeful sampling sets out to access the greatest variance and diversity of participants who can share reports of the area of interest (Polit and Hungler, 1997). Maximum variation sampling has the advantage of highlighting common threads across all participant experiences whilst also providing detailed descriptions for individual encounters (Hoepfl, 1997, Patton, 1990).

The a priori characteristics for sampling in this study were patients who had experienced both STEMI and re-presentation to acute services due to potential IHD symptoms during the first 6 months following treatment for STEMI. The intention was to include a group that reflected the diversity of the study population. This allowed confirmation of important meaning and patterns that emerged from the data (Polit and Hungler, 1997). Qualitative data collection and analysis were conducted concurrently. This enabled the sampling strategy to evolve and to be adapted as data were collected, ensuring the inclusion of individuals who characterised the diversity of the group. Polit and Hungler (1997) state that, “who to sample next depends on who has been sampled already” (page 237).

Concurrent sampling, data collection and data analysis were conducted to aid selection of participants and to explore and develop concepts (Charmaz, 2006) (see Table 4-4). Sampling was regarded as complete once new information was no longer emerging during the interviews and analysis (Charmaz, 2006). This is known as data saturation and at this point no further participants were interviewed. Corbin
and Strauss (2008) use the following description to illuminate how researchers can be assured that data saturation as occurred:

"A researcher knows when sufficient sampling has occurred when the major categories show depth and variation in terms of their development" (page 149).

Clearly, there is a subjective element to the process of both achieving and recognising data saturation. It has been suggested that the involvement of more than one researcher can improve the reliability involved in the assessment of data saturation (Pope et al., 2000). Morse et al (2002) state that ensuring the rigor of a study should be a continuous process during the conduct of a study. Previously other researchers such as Guba and Lincoln (1989) have suggested that the assessment of trustworthiness is a post hoc evaluation. Morse et al (2002) determine that verification strategies used during the conduct of a study are a means of the researcher knowing when to stop collecting data and ensures rigor during the course of the study.

During the course of data collection and analysis one of the supervisory team worked with the author and acted as a verifier. The point at which data saturation was said to be present was jointly assessed in several discussions between the author and the supervisor.

6.2.5 Recruitment to the qualitative study

Previously, during the initial informed consent process for participation in the mixed methods study, an explanation had been given to participants regarding the interview aspect of the study. It was explained that should the participant re-present to acute services during the first 6 months following their heart attack they may be asked if they wish to be interviewed. During telephone contact at 6 months it was confirmed whether the participant had re-presented to acute services (with IHD symptoms) since STEMI. For individuals who had re-presented, a brief overview of the participant’s experience was elicited. This enabled the researcher to ascertain whether interviewing the individual would enable exploration of the emerging categories established during the concurrent data analysis. Those invited for interview, were given a further verbal description of the interview and it was established whether they were willing to be interviewed. Verbal consent was then taken to arrange an interview date; participants had already received a patient information sheet at the outset of the study 6 months earlier. However, a further copy of the information sheet was offered to participants.
and informed consent was taken at the beginning of the interview by the interviewer. As part of the consent process it was explained to participants that the interviews would be audio recorded and transcribed. Participants were informed that the recordings and transcripts would be stored in a locked cupboard and only the members of the research team and the transcriber(s) would have access to them.

Throughout the study, procedures were in place to maintain confidentiality and the anonymity of the participants. At the outset of the study all participants were allocated a study identification (ID) number to protect their identity. The ID number was used on all written and electronic documentation including the interview transcripts. Furthermore, all identifiable information including people and place names were removed from the interview transcripts. Electronic audio recordings, interview notes and transcripts were stored on a password locked computer and paper copies of documents were stored in a locked filing cabinet.

6.2.6 Qualitative data collection

6.2.6.1 Interview setting and conduct

Interviews took place in the patients’ choice of setting, and it was the intention of the author to limit interviews to participants alone. However, this was not always feasible as in most cases participants chose to be interviewed in their own home and when a partner or family member was present it was not possible or appropriate to ask them to leave. Due to the in-depth nature of the interviews the majority lasted between one and one and a half hours.

Although interviews were designed to be semi-structured, a conversational style was adopted to put the interviewee at ease and allow for probing of developing themes (Bowling, 1997). Avenues of enquiry that opened up during the interview were explored through flexible questioning, leading to increased clarity and detail (Kvale, 1996). Open ended questions were used to encourage patients to express themselves and share information (Kvale, 1996). Common language was adopted and technical jargon avoided enabling the interviewees to easily understand the interview dialogue (Pontin, 2000). Nonjudgmental responses to the interviewee’s responses helped to build rapport and encouraged individuals to share information (Johnson and Turner, 2003).

During the interviews audio recordings were made (using a digital recorder), to enable not only the dialogue to be captured but also pauses and the tone of speech
This helped interpretation relating to the participants’ emotions during the interview. Immediately following the interview a written record or ‘field notes’ were made to record the contextual information related to the interview (Hughes, 1994). For instance, the individuals’ body language during the interview, a description of where the interview took place including the type of living accommodation (for those interviewed at home) was recorded. Furthermore, information relating to demographics, employment, marital status and family situation were all recorded to contextualize the interviews.

6.2.6.2 Development of the interview schedule

An interview schedule was designed to meet the aims of the study at the start of the study (Kvale, 1996). The initial schedule covered the patients’ perception of the cause of symptoms which led to re-presentation. The participants’ reasons for seeking help and their emotional experiences of investigations and treatments received during admission were also explored. The initial interview schedule was piloted during an interview with a PPCI patient and the schedule was then adapted to include additional concepts such as lifestyle changes and attendance at cardiac rehabilitation. An example of the interview schedule can be found in Appendix M. During the interviews, the interview schedule was flexibly applied enabling issues and topics raised by the participants to be explored. Emergent themes and topics were then used as a basis for adapting and refining future interview schedules with subsequent patients (Kvale, 1996). Data collection was complete once data saturation occurred and no new data emerged, at this point no further patients were interviewed (see section 6.2.4).

6.2.7 Qualitative data analysis

In this study constant comparison, an aspect of grounded theory, was used to explore meaning and develop concepts and themes iteratively from the data (Creswell and Plano Clarke, 2007) (see section 4.10.2 and Table 4-4). Framework analysis was also used in managing the analytical procedures for this study (Ritchie and Spencer, 1994). The way in which framework was applied in this study is described in section 6.3.

6.2.7.1 Preliminary procedures

The interviews were transcribed verbatim. The transcriptions included colloquialisms and native speech as well as incorporating laughter and sighing to gain the true feeling of the participants’ emotions (Kvale, 1996). To assist the charting and mapping phases of framework analysis each page and lines of the
transcripts were numbered. Following transcription any reference of people or place names and other identifiable details were removed from the transcripts to protect the individual’s anonymity.

A software package, ‘NVivo 8’, was used to manage the data. NVivo is computer aided qualitative data analysis software (CAQDAS). Qualitative research involves the generation of large amounts of data and in the case of transcribed interviews copious pages of interview transcripts are generated. It has been shown that the analytical process can be aided with the use of CAQDAS (Fielding, 2002, Gibbs, 2002, Pope et al., 2000, Bazeley, 2007). These types of software have the advantage of enabling large amounts of data to be managed more easily; by allowing simple access to multiple transcripts, facilitating the coding of chunks of text and the retrieval of text (Bazeley, 2007).

6.3 Framework analysis

Framework analysis is described fully in section 4.11.1. In brief, it involves five interconnected stages including familiarisation, identifying a thematic framework, indexing, charting and mapping and interpreting (Ritchie and Spencer, 1994). This section of the thesis describes how Framework analysis was applied to the study data.

6.3.1 Familiarisation

The audio-recordings from the interviews were repeatedly listened to by the researcher to gain an understanding of the subject matter and the emotional content of the interviews. Furthermore, the interview transcripts were also read and re-read on numerous occasions, to enable the researcher to become immersed in the data. The background information documented following the interview was also reviewed to contextualise the interviews. Throughout the process of familiarisation, notes were made to capture recurring topics and important aspects that came to the fore (see section 4.11.1.1).

6.3.2 Identifying a thematic framework

A thematic framework was set-up in NVivo 8® (computer aided qualitative data analysis software (CAQDAS)), using the original concepts included in the interview schedule (Ritchie and Spencer, 1994). The thematic framework was developed and refined over time following the conduct and analysis of further interviews. Additionally, the key concepts and recurrent themes identified during familiarization were also used to draw up the framework (see section 4.11.1.2).
6.3.3 Indexing

Interview transcripts were indexed (or coded) line by line in NVivo 8 to elicit inferences through deductive reasoning (Charmaz, 2006). Indexes or codes are referred to as nodes, sub-nodes and free nodes (nodes which have not been assigned to a node or sub-node) in NVivo 8®. The nodes and sub-nodes are also equivalent to themes and sub-themes (respectively) and for the purposes of this analysis the terminology themes and sub-themes will be used.

Initially the indexing used broad concepts, including emotional, physical and disease related aspects of the participants’ experience (Ritchie and Spencer, 1994). As concurrent data collection and analysis progressed, indexing evolved and inductive reasoning was used to explore new topics and emergent themes. Inductive reasoning involves developing categories from within the data. In many cases sentences and paragraphs elicited multiple concepts and themes, as they included a combination of meaning, emotions and narrative descriptions (Charmaz, 2006). As indexing progressed some of the themes fitted well within the existing thematic framework. However, as new meaning or fresh concepts surfaced new themes or sub-themes were developed and were added to the existing framework. This led to refinement of the thematic framework over time.

As the analysis of more interviews advanced the number of sub-themes and free nodes had the potential to become unmanageable. The researcher in discussion with the supervisor decided at this point to rationalize the themes, sub-themes and free nodes by collapsing them into other appropriate themes and sub-themes where similar meaning or concepts could be found. When this was not possible a new relevant ‘sub-theme’ was used to incorporate a group of sub-themes or free nodes (or indexes). If it was believed that the meaning or concept would be lost by collapsing themes and sub-themes, they were left in their original form.

A journal was maintained throughout to capture the decisions made relating to the collapsing and combining of nodes, sub-nodes and the evolution of the themes and the overarching thematic framework (Charmaz, 2006). An excerpt from the diary can be found in Appendix P. This phase of framework analysis is discussed further in section 4.11.1.3.

6.3.4 Charting

Charts were drawn up for each of the themes and sub-themes, and the data for each participant was entered onto the relevant chart. These charts were
constructed under themes (or subject headings), some of which matched identically with the indexing codes (or nodes), whereas others reflected the themes that had developed during indexing (Ritchie and Spencer, 1994). Important quotes for participants and themes were entered onto the charts, to ensure that the context and ‘evidence’ remained rooted in the data. On other occasions several words were used to summarise the meaning. For all entries on the charts reference was made to the page and line numbers of the original source data (transcript), this was to ensure that the data could continue to be accessed and for ease of auditing and interpretation (Ritchie et al., 2003). Each participant (or case) remained in the same order on each chart and for every theme, this was to enable cross comparison during mapping and interpretation (Ritchie et al., 2003). For ease of storage and management, charts were arranged in Microsoft® Word tables. See section 4.11.1.4 for further details of the charting phase.

A chart was also drawn up containing contextual information including the participants’ sociodemographic and re-presentation data. Information from the interviews and from the quantitative dataset (stored in SPSS®) was used to indicate the participants’ demographic, employment status, the number of re-presentations due to potential IHD symptoms (for each participant) and the final re-presentation discharge diagnosis. Information related to the final discharge diagnosis was used to allow the researcher to gain further insight of the participants’ beliefs and understanding of the re-presentation event. In particular such information was helpful in establishing the participants’ misconceptions relating to diagnosis or in the case of a missing or indeterminate diagnosis the reason for their confusion and anxiety related to anxiety.

**6.3.5 Mapping and interpreting**

The mapping and interpretation stage involved looking across all charts, themes and cases to bring together the major concepts and characteristics running through the data relating to the participants experiences or re-presentation. The process of mapping and interpretation is the most difficult part of the analytical procedure to describe. Ritchie and Spencer (1994) consider that each step of this process requires “leaps of intuition and imagination” (page 186).

Large charts for each theme (and sub-themes) were drawn up to enable the researcher to visualise the concepts and to aid mapping and interpretation. Practical steps were implemented to assist the process, included using colour coding and Post-it notes to highlight and draw out linkages and associations on the
charts. The field notes and memos were reviewed again to ensure that the contextual information was not lost during this interpretive phase. Furthermore, the study purpose was revisited to remind the researcher of the focus of the study (Ritchie and Spencer, 1994).

Patterns in the data were explored to seek a more in-depth understanding of the participants’ perception of symptom cause; this was achieved by comparing and contrasting the accounts and experiences of the participants. Explanations were also sought regarding the reasons that participants had required additional help with their symptoms, and their construction of events that led to re-presentation. A systematic process was applied to further define emotional constructs and to establish linkages and associations between themes relating to the participants’ experiences of re-presentation and clinical investigations (Ritchie et al., 2003). Emergent themes that developed during data collection were reviewed for common threads and associations across cases. Behaviours of participants relating to symptoms and help seeking were also sought. Additionally, the beliefs to the original treatment and subsequent care were defined and explained. Throughout this phase the different stages of the analysis were often revisited, in some instances the raw data (i.e. the transcripts) was consulted, and on other occasions the coding and subthemes were re-reviewed (Ritchie et al., 2003).

This phase of the analysis led to the development of a final list of themes and thematic framework, which was presented in Table 8-2. The study findings were also described through a narrative incorporating participant quotes to elicit the lived experiences of the interviewees.

### 6.4 Rigour

The rigour (or quality) of the qualitative study was addressed throughout the conduct of the study. Good record keeping, clear definition of the study methods, declaration of the researchers beliefs and potential biases, and validating the study findings were all used to define rigour of the study. A full description of what constitutes rigour and how it is addressed as part of this study is described in detail in section 4.12. However, a summary of the methods used to address rigour are briefly presented below.

All aspects of the study are described in the thesis to enable the reader to ‘audit’ the research processes used and also to interpret the study findings (Bowling, 1997).
The researcher undertook good record keeping, including the use of computer aided qualitative data analysis software (CAQDAS), keeping a journal and recording contextual information. Furthermore, Framework analysis enabled the process of analysis to be clearly viewed by others.

It was the intention of the researcher to present sufficient raw data to enable the reader to validate the study findings. Excerpts of documents used during the data analysis process are provided as appendices for the reader to evaluate. These include copies of interview schedules, excerpts of interview transcripts and screenshots of indexing samples conducted in NVivo 8®. Additionally an extract of a Framework chart and photographs depicting the mapping and interpretation process are also presented in Appendix S and Appendix T respectively.

Moreover, the beliefs and potential biases of the researcher are declared to enable others to assess the influence that these may have had on the analysis. The potential biases were also addressed by the involvement of one of the supervisory team during the analysis. Furthermore, methods triangulation has been suggested by Denzin (1978) as a form of validating study findings. In this study the mixed methods study design may therefore have added to the validity of the study findings (Denzin, 1978).

6.5 Summary

The methods for the qualitative phase of this explanatory mixed methods study are described in this chapter of the thesis. The purpose of the study and study design are discussed in detail, along with the methods undertaken.

A modified version of grounded theory was used to conduct the study with the application of concurrent sampling, data collection and data analysis. Furthermore, the analysis was conducted using constant comparative analysis.

Purposeful sampling in the form of maximum variation sampling was used to select participants who had re-presented to acute healthcare services with potential IHD symptoms within 6 months of experiencing STEMI and PPCI treatment. Semi-structured interviews were conducted on one occasion and data collection was complete once data saturation was achieved. Data analysis was undertaken with the use of framework analysis. Ultimately a final thematic framework was devised with a final list of themes. The findings were presented in a table of themes (and sub-themes) and in narrative form through the words of the participants in a storyline.
7.1 Introduction

This chapter reports the results of the study, including descriptive information on identified and recruited patients. The characteristics of the study cohort including sociodemographic, clinical and psychological health results are presented in bivariate analyses. Results in relation to the study aims are also presented including the number and categorisation of re-presentation events. Additionally, the results of the self-report questionnaires at baseline and 6 months for the Hospital Anxiety and Depression Scale (HADS) and the Seattle Angina Questionnaire (SAQ) are also presented. The results of the multivariate analysis, relating to whether there is a relationship between psychological health, symptoms and re-presentation are presented towards the end of the chapter.

7.2 Sample Overview

In total 274 patients treated with PPCI for STEMI were screened between July 2007 and December 2008. A total of 43 patients were not recruited. Thirty-one of these patients fitted the study inclusion and exclusion criteria (see section 5.7) but were either too unwell at baseline to give consent, refused to participate or were missed, and one patient was a foreign visitor. A further 12 patients were not eligible. Reasons for non-participation for the 43 patients who were not recruited are described in Figure 7-1.

7.2.1 Study sample

A total of 231 patients gave written informed consent to participate in the study and of these individuals 202 returned baseline questionnaires (see Figure 7-1).
Figure 7-1 - Patient participation during the study

Total STEMI patients
n=274
- Treated with PPCI

Patients Recruited
n=231

Responders
n=202
(87.5% response rate)

Non-responders
n=29
• 4 died
• 3 withdrew
• 22 did not return baseline questionnaire

Re-presented (0-6m)
n=7
Non-represented (0-6m)
n=22

Patients Not Recruited
n=43

Eligible
n=31
• 19 Refused
• 6 Missed
• 4 Unwell at Baseline
• 1 Foreign visitor
• 2 No telephone
• 1 Alcoholism
• 2 Deaf and/or blind

Not – Eligible
n=12
• 2 Severe cognitive impairment
• 3 No English
• 2 Severe mental health problems
• 1 Died as in-patient
• 4 Severe physical illness

Re-presented (0-6m)
n=38
Non-represented (0-6m)
n=164
7.2.2 Addressing response bias

Individuals who gave informed consent and returned baseline questionnaires were categorised as responders (n=202) and individuals who gave consent but did not return their questionnaires or withdrew were classed as non-responders (n=29); indicating an 87.5% response rate. Response bias was assessed by comparing the descriptive statistics for specific group characteristics for responders and non-responders. Because the number of non-responders was small, the comparison was descriptive, details of which can be seen Table 7-1. The reasons for non-response are shown in Figure 7-1.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Responder (n=202)</th>
<th>Non-responder (n=29)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td></td>
<td>59.7 (13.9)</td>
<td>59.9 (11.3)</td>
</tr>
<tr>
<td></td>
<td>Median (range)</td>
<td>Median (range)</td>
</tr>
<tr>
<td></td>
<td>60.0 (26 to 87)</td>
<td>60 (27 to 83)</td>
</tr>
<tr>
<td>Gender (male)</td>
<td>N %</td>
<td>N %</td>
</tr>
<tr>
<td></td>
<td>153 75.7</td>
<td>19 65.5</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>191 94.5</td>
<td>26 89.7</td>
</tr>
<tr>
<td>Mixed race</td>
<td>0 0.0</td>
<td>3 10.3</td>
</tr>
<tr>
<td>Asian</td>
<td>9 4.5</td>
<td>0 0.0</td>
</tr>
<tr>
<td>Black</td>
<td>2 1.0</td>
<td>0 0.0</td>
</tr>
<tr>
<td>Marital status</td>
<td>Married(^1)</td>
<td>152 75.3</td>
</tr>
<tr>
<td></td>
<td>Single(^2)</td>
<td>35 17.3</td>
</tr>
<tr>
<td></td>
<td>Widowed</td>
<td>15 7.4</td>
</tr>
<tr>
<td>PPCI admission details</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PPCI length of stay (days)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td></td>
<td>11.2 (14.0)</td>
<td>6.3 (3.6)</td>
</tr>
<tr>
<td></td>
<td>Median (range)</td>
<td>Median (range)</td>
</tr>
<tr>
<td></td>
<td>5.0 (3 to 65)</td>
<td>5.0 (2 to 28)</td>
</tr>
<tr>
<td>Transfer PPCI</td>
<td>Yes</td>
<td>145 72.1</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>56 27.9</td>
</tr>
<tr>
<td>Index hospital admission</td>
<td>Trust 1</td>
<td>66 32.7</td>
</tr>
<tr>
<td></td>
<td>Trust 2</td>
<td>28 13.9</td>
</tr>
<tr>
<td></td>
<td>Trust 3</td>
<td>19 9.4</td>
</tr>
<tr>
<td></td>
<td>Trust 4</td>
<td>26 12.9</td>
</tr>
<tr>
<td></td>
<td>Trust 5</td>
<td>21 10.4</td>
</tr>
<tr>
<td></td>
<td>Trust 6</td>
<td>42 20.8</td>
</tr>
<tr>
<td>PPCI centre</td>
<td>Trust 1</td>
<td>156 77.2</td>
</tr>
<tr>
<td></td>
<td>Trust 2</td>
<td>46 22.8</td>
</tr>
</tbody>
</table>

\(^1\)Married includes Civil Partnership and living with partner
\(^2\)Single includes divorced and separated

There were small differences between the groups in relation to gender, marital status and transfer for PPCI. Compared to the non-responder group, a smaller percentage of responders were women (24.3% vs 34.5%) and more responders...
were married (75.3% vs 62.1%). Furthermore, a higher percentage of responders (72.1%) were transferred for PPCI than non-responders (55.2%).

During the index admission for STEMI, the mean length of stay was greater in the responder group (11.2 vs 6.3 days). However, this was due to outliers in the responder group with the longest stay being 65 compared to 28 days in the non-responder group; the median was the same for both groups (5.0).

Overall there appeared to be no major differences in characteristics between responder and non-responder groups (Table 7-2).

### 7.2.3 Re-presentation

Overall 45 /231 (19.5%) participants re-presented to acute healthcare services with symptoms of potential IHD during the first 6 months post PPCI. Seven of these individuals did not return questionnaires and were therefore excluded from subsequent analysis. The 38 individuals who did return questionnaires form the re-presentation group throughout the analysis in this study (further detailed analysis of re-presentations can be found under section 7.5). Those who did not re-present to acute healthcare services with potential IHD symptoms during the 6 months (either because they did not re-present at all or re-presented with symptoms other than those related to IHD) are referred to as the non-representation group.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Responder (n=202)</th>
<th>Non-responder (n=29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Re-presentation (potential IHD)</td>
<td>N</td>
<td>%</td>
</tr>
<tr>
<td>Yes</td>
<td>38</td>
<td>18.8</td>
</tr>
<tr>
<td>No</td>
<td>164</td>
<td>81.2</td>
</tr>
</tbody>
</table>

*95% CI confidence interval

### 7.2.4 Response rates at 6 months

A total of 184 individuals returned their 6 month HADS and SAQ questionnaires compared to 202 at baseline, indicating a response rate of 91.1%. In the re-presentation group one individual failed to return their 6 month questionnaire compared to 17 in the non-representation group. There was no evidence of a difference between the groups in the number of participants who returned their questionnaires at 6 months (Pearson’s $\chi^2=2.27$, df=1, p=0.132).
7.2.5 Missing values analysis

Data appeared to be missing at random and no real patterns of missing variables were detected. Data collection was complete for all 231 consented participants for age, gender, ethnicity, marital status, The Enhancing Recovery in Coronary Heart Disease (ENRICHD) social support instrument (ESSI) (ENRICHD Investigators, 2000, Vaglio et al., 2004), transfer PCI, diabetes and further revascularisation.

The largest number of missing values was seen under LV function. LV function is usually measured using echocardiography during the patients’ index admission for STEMI and recorded in the patients’ notes. It is recommended by NICE (2010b) and the European Society of Cardiology as an essential part of clinical care following STEMI (Thygesen et al., 2007). Despite these recommendations in 17.0% of participants in this study, the data were missing. As this investigation is usually part of routine clinical care, it was not initially thought necessary to conduct the test specifically for the study. Financial and time constraints due to the study being conducted as an academic piece of work also prevented the investigation being conducted specifically for the study.

In the case of responders who returned incomplete questionnaires, individuals were contacted by telephone to obtain answers to the missing questions. Complete HADS and SAQ data were collected for all 202 participants. However, for the SAQ physical limitation sub-scale when participants answered “limited or did not do for other reasons”, the algorithm treated the answer as missing data (see section 5.11.6). Consequently, this led to 17.3% missing data at baseline for the SAQ physical limitation sub-scale.

7.3 Baseline analysis

7.3.1 Characteristics

Sociodemographic characteristics are summarised in Table 7-3 and Table 7-4 for all participants and for the presentation groups. The theoretical underpinning and choice of statistical tests used are described in sections 3.4 and 5.11 respectively and will be referred to throughout the results chapter.

The mean age of the study cohort was 59.7 years and the age range was wide (from 26 to 87 years). The distribution for age was normal and there was no evidence of a difference in age between the presentation groups at baseline (Table
7-3). The mean age of the cohort is reflected in their employment status with almost 40.0% being retired.

Seventy-five percent of participants were male and the majority of individuals were either married, in a Civil Partnership or were living with a partner (75.3%) at the outset of the study. This was reflected by the high percentage of individuals living with someone (81.5%). Levels of social support (as measured by the The Enhancing Recovery in Coronary Heart Disease (ENRICHD) social support instrument (ESSI)) (ENRICHD Investigators, 2000, Vaglio et al., 2004) for the cohort as a whole and the two groups was high indicating good levels of social support. The distribution for ESSI was not normal for the cohort or for the groups (see Appendix L).

Overall relatively low levels of education were found across the study cohort, with 36.6% of participants not having any qualifications (including secondary education qualifications or certificates) and 36.1% having one or more of ‘O’ level, CSE, GCE, school leavers certificate or City and Guilds qualification.

At baseline gender, ethnicity, marital status, social support, occupation and education showed no significant differences between the presentation groups (Table 7-4).

Table 7-3 Continuous sociodemographic characteristics by presentation group

<table>
<thead>
<tr>
<th>Variable</th>
<th>All (n=202)</th>
<th>Re-presentation (n=38)</th>
<th>Non-representation (n=164)</th>
<th>Re-presentation vs Non-representation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>Mean (SD)</td>
<td>Median (range)</td>
<td>Mean (SD)</td>
<td>Median (range)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>59.7 (13.9)</td>
<td>60.0 (26-87)</td>
<td>58.2 (12.1)</td>
<td>58 (27-79)</td>
</tr>
<tr>
<td>ESSI</td>
<td>28.7 (6.4)</td>
<td>31.0 (8-34)</td>
<td>27.7 (7.1)</td>
<td>30.5 (11-34)</td>
</tr>
</tbody>
</table>

1Independent- t test
2Mann-Whitney U test
Table 7-4: Description of sociodemographic (categorical) variables by presentation group.

<table>
<thead>
<tr>
<th>Variable</th>
<th>All (n=202)</th>
<th>Re-presentation (n=38)</th>
<th>Non-representation (n=164)</th>
<th>Re-presentation vs Non-representation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N (%)</td>
<td>N (%)</td>
<td>N (%)</td>
<td>Test (df)</td>
</tr>
<tr>
<td>Gender (male)</td>
<td>153 (75.7)</td>
<td>25 (65.8)</td>
<td>128 (78.0)</td>
<td>$\chi^2=2.52$ (1)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>White</td>
<td>19 (94.5)</td>
<td>35 (92.1)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Asian</td>
<td>9 (4.5)</td>
<td>2 (5.3)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>2 (1.0)</td>
<td>1 (0.6)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>$\text{FE}$</td>
</tr>
<tr>
<td>Marital status</td>
<td>1Married</td>
<td>15 (75.3)</td>
<td>26 (68.4)</td>
<td>$\chi^2=2.77$ (2)</td>
</tr>
<tr>
<td></td>
<td>2Single</td>
<td>35 (17.3)</td>
<td>10 (26.3)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Widowed</td>
<td>15 (7.4)</td>
<td>2 (5.3)</td>
<td></td>
</tr>
<tr>
<td>Social support</td>
<td>Live alone</td>
<td>37 (18.3)</td>
<td>8 (21.1)</td>
<td>$\chi^2=4.96$ (1)</td>
</tr>
<tr>
<td></td>
<td>Living with</td>
<td>165 (81.7)</td>
<td>30 (78.9)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>someone</td>
<td></td>
<td>135 (82.3)</td>
<td></td>
</tr>
<tr>
<td>Occupation</td>
<td>Employed</td>
<td>58 (28.7)</td>
<td>11 (28.9)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>full-time</td>
<td></td>
<td>47 (28.7)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>part-time</td>
<td>13 (6.4)</td>
<td>3 (7.9)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Self</td>
<td>23 (11.4)</td>
<td>2 (5.3)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>employed</td>
<td></td>
<td>21 (12.8)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>unemployed</td>
<td>19 (9.4)</td>
<td>4 (10.5)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Retired</td>
<td>80 (39.6)</td>
<td>15 (39.5)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Housework</td>
<td>9 (4.5)</td>
<td>3 (7.9)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>6 (3.7)</td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td>Responders</td>
<td>n=194</td>
<td>n=37</td>
<td>n=157</td>
</tr>
<tr>
<td></td>
<td>None$^7$</td>
<td>71 (36.6)</td>
<td>12 (32.4)</td>
<td>59 (37.6)</td>
</tr>
<tr>
<td></td>
<td>GCSE$^7$</td>
<td>70 (36.1)</td>
<td>19 (51.4)</td>
<td>51 (32.5)</td>
</tr>
<tr>
<td></td>
<td>A Level$^7$</td>
<td>35 (18.0)</td>
<td>5 (13.5)</td>
<td>30 (19.1)</td>
</tr>
<tr>
<td></td>
<td>Degree</td>
<td>12 (6.2)</td>
<td>1 (2.7)</td>
<td>11 (7.0)</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>6 (3.1)</td>
<td>0 (0.0)</td>
<td>6 (3.8)</td>
</tr>
<tr>
<td></td>
<td>graduate</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

$^1$Married- includes Civil Partnership and living with partner
$^2$Single- includes divorced and separated
$^3$Fisher's Exact Test
$^4$Pearson's Chi-Square test
$^5$GCSE- includes 'O' levels, CSE's, school leavers certificate and City and Guilds
$^6$A Level- includes NVQ, diploma and HNC
$^7$No qualifications, including no secondary education qualifications or certificates.

### 7.3.2 Index PPCI admission variables

The total length of hospital stay (LOS) (including both the PPCI centre and the transfer hospital for those transferred) had a mean of 6.3 days (SD 3.6, median 5 days). The minimum admission time was 2 days and the longest stay was 65 days. There was no evidence of a difference between the presentation groups in length of hospital stay ($p=0.214$) (Table 7-5). The majority of individuals (72.1%) were directly admitted to a PPCI centre for treatment. Furthermore, 77.2% of participants were treated at PPCI centre, Trust 1; the reasons for this are discussed in section 9.7.1. Two thirds (66.0%) of individuals sought help at the point of
STEMI by telephoning the emergency ambulance service. The second most common route of accessing services was via self referral to the A&E department with 20.1% of individuals seeking help by this means (Table 7-6). There was no evidence of a difference between the groups in relation to the proportion of patients transferred for PPCI, treated at PPCI centre Trusts 1 and 2, and also admission route (Table 7-6).

Table 7-5 Continuous PPCI admission characteristics by presentation.

<table>
<thead>
<tr>
<th>Variable</th>
<th>All (n=202)</th>
<th>Re-presentation (n=38)</th>
<th>Non-representation (n=164)</th>
<th>Re-presentation vs Non-representation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Median (range)</td>
<td>Mean (SD)</td>
<td>Median (range)</td>
</tr>
<tr>
<td>PPCI length of stay (days)</td>
<td>6.3 (3.6)</td>
<td>5.0 (2.0-28.0)</td>
<td>5.5 (2.4)</td>
<td>5.0 (2.0-13.0)</td>
</tr>
</tbody>
</table>

*Mann-Whitney U test

Table 7-6 Categorical PPCI admission characteristics by presentation.

<table>
<thead>
<tr>
<th>Variable</th>
<th>All (202)</th>
<th>Re-presentation (n=38)</th>
<th>Non-representation (n=164)</th>
<th>Re-presentation vs Non-representation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N (%)</td>
<td>N (%)</td>
<td>N (%)</td>
<td>Test (df)</td>
</tr>
<tr>
<td>Transfer PPCI</td>
<td>Yes</td>
<td>56 (27.9)</td>
<td>9 (23.7)</td>
<td>47 (28.7)</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>145 (72.1)</td>
<td>29 (76.3)</td>
<td>116 (71.2)</td>
</tr>
<tr>
<td>Index hospital admission</td>
<td>Trust 1</td>
<td>66 (32.7)</td>
<td>16 (42.1)</td>
<td>50 (30.5)</td>
</tr>
<tr>
<td></td>
<td>Trust 2</td>
<td>28 (13.9)</td>
<td>5 (13.2)</td>
<td>23 (14.0)</td>
</tr>
<tr>
<td></td>
<td>Trust 3</td>
<td>19 (9.4)</td>
<td>2 (5.3)</td>
<td>17 (10.4)</td>
</tr>
<tr>
<td></td>
<td>Trust 4</td>
<td>26 (12.9)</td>
<td>4 (10.5)</td>
<td>22 (13.4)</td>
</tr>
<tr>
<td></td>
<td>Trust 5</td>
<td>21 (10.4)</td>
<td>2 (5.3)</td>
<td>19 (11.6)</td>
</tr>
<tr>
<td></td>
<td>Trust 6</td>
<td>42 (20.8)</td>
<td>9 (23.7)</td>
<td>33 (20.1)</td>
</tr>
<tr>
<td>PPCI centre</td>
<td>Trust 1</td>
<td>156 (77.2)</td>
<td>31 (81.6)</td>
<td>125 (76.2)</td>
</tr>
<tr>
<td></td>
<td>Trust 2</td>
<td>46 (22.8)</td>
<td>7 (18.4)</td>
<td>39 (23.8)</td>
</tr>
<tr>
<td>Admission route</td>
<td>Via GP</td>
<td>(n=201)</td>
<td>(n=38)</td>
<td>(n=163)</td>
</tr>
<tr>
<td></td>
<td>13 (6.5)</td>
<td>1 (2.6)</td>
<td>14 (9.0)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>123 (61.3)</td>
<td>26 (68.4)</td>
<td>102 (65.4)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>39 (20.1)</td>
<td>8 (21.1)</td>
<td>31 (19.9)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Already in hospital</td>
<td>10 (5.2)</td>
<td>1 (2.6)</td>
<td>9 (5.8)</td>
</tr>
</tbody>
</table>

1Pearson Chi-Square test
2Fisher’s exact test
3Dialing 999 for the emergency ambulance service

### 7.3.3 STEMI clinical variables

The mean levels of troponin ‘T’ for the groups were the same and were raised above the normal reference range (>0.01 at the time of this study) for troponin ‘T’ indicating myocardial infarction [re-presentation group 4.0 (SD 5.3) and non-representation group 4.8 (SD 5.2)] (Table 7-7). The range of troponin ‘T’ was from 0.01 to 23.4 in the re-presentation group and 0.01 to 37.8 in the non-
representation group. The upper limit of troponin 'T' was high in both groups indicating that some patients experienced high degrees of damage to the myocardium at the time of STEMI. One patient in each group (patients 254 and 270) had a troponin 'T' of 0.01 and this is considered to be a normal result indicating that a STEMI may not have recently occurred (Panteghini et al., 2002). However, according to both patients’ medical records the participants had suffered recent damage to the myocardial wall (due to STEMI) leading to the index admission. Patient 254 suffered an inferior wall STEMI (indicating the region of the myocardial wall damage) and patient 270 an anterior wall STEMI (see section 1.2.1 for the description of STEMI categorisation). It was therefore decided to continue to include the two patients in the dataset as they were treated with PPCI and received post PPCI discharge follow-up care. The unexpected 0.01 Troponin 'T' results may have been due to the blood being drawn at the point of admission. It is recommended that Troponin 'T' is measured 12 hours following the most intense pain to indicate raised Troponin 'T' levels (National Institute for Health and Clinical Excellence, 2010a, Antman et al., 2004). The troponin 'T' levels were not significantly different between the presentation groups (p=0.146) (Table 7-7).

Table 7-7 - Continuous STEMI clinical characteristics by presentation

<table>
<thead>
<tr>
<th>Demographics</th>
<th>All (n=186)</th>
<th>Re-presentation (n=38) vs Non- representation (n=148)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Troponin 'T'</td>
<td>Mean (SD)</td>
<td>Median (range)</td>
</tr>
<tr>
<td></td>
<td>4.6 (5.2)</td>
<td>3.1 (0.01-37.8)</td>
</tr>
<tr>
<td>GRACE predictor of death or MI at 6 months (%)</td>
<td>28.5 (9.8)</td>
<td>27.0 (12.0-70.0)</td>
</tr>
</tbody>
</table>

* Mann- Whitney U test

The GRACE results in Table 7-7 represent the probability of death or MI from admission to 6 months and show that overall more than a quarter of participants were at risk (mean 28.5%, SD=9.8; range 12.0% to 70.0%). There were no differences in the GRACE predicted outcome from admission to 6 months between the presentation groups (p=0.915).

The most common type of STEMI across the groups affected the inferior wall of the myocardium (re-presentation group 71.1% vs non-representation group 56.1%), with anterior being the second most common (26.3% vs 36.6%). Groups were the
same in terms of the number of patients suffering each type of STEMI (p=0.472) (Table 7-8).

Table 7-8 Categorical STEMI clinical characteristics by presentation.

<table>
<thead>
<tr>
<th></th>
<th>ALL (n=202)</th>
<th>Re-presentation (n=38)</th>
<th>Non-representation (n=164)</th>
<th>Re-presentation vs Non-representation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N (%)</td>
<td>N (%)</td>
<td>N (%)</td>
<td>Test (df) P value</td>
</tr>
<tr>
<td>Type of STEMI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior</td>
<td>70 (34.7)</td>
<td>10 (26.3)</td>
<td>60 (36.6)</td>
<td>FE</td>
</tr>
<tr>
<td>Inferior</td>
<td>119 (58.9)</td>
<td>27 (71.1)</td>
<td>92 (56.1)</td>
<td>FE</td>
</tr>
<tr>
<td>Posterior</td>
<td>6 (3.0)</td>
<td>0 (0.0)</td>
<td>6 (3.7)</td>
<td>FE</td>
</tr>
<tr>
<td>Lateral</td>
<td>6 (3.0)</td>
<td>1 (2.6)</td>
<td>5 (3.0)</td>
<td>FE</td>
</tr>
<tr>
<td>LBBB(^1)</td>
<td>1 (0.5)</td>
<td>0 (0.0)</td>
<td>1 (0.6)</td>
<td>FE</td>
</tr>
<tr>
<td>LV function</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Responders</td>
<td>(n=165)</td>
<td>(n=30)</td>
<td>(n=135)</td>
<td></td>
</tr>
<tr>
<td>&gt;50</td>
<td>94 (57.0)</td>
<td>18 (60.0)</td>
<td>76 (56.3)</td>
<td>FE</td>
</tr>
<tr>
<td>45-50</td>
<td>24 (14.5)</td>
<td>2 (6.7)</td>
<td>22 (16.3)</td>
<td>FE</td>
</tr>
<tr>
<td>30-44</td>
<td>36 (21.8)</td>
<td>5 (16.7)</td>
<td>31 (23.0)</td>
<td>FE</td>
</tr>
<tr>
<td>&lt;30</td>
<td>11 (6.7)</td>
<td>5 (16.7)</td>
<td>6 (4.4)</td>
<td>FE</td>
</tr>
<tr>
<td>Cardiac arrest O/A</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>12 (5.9)</td>
<td>3 (7.9)</td>
<td>9 (5.5)</td>
<td>FE</td>
</tr>
<tr>
<td>No</td>
<td>190 (94.1)</td>
<td>35 (92.1)</td>
<td>155 (94.5)</td>
<td>FE</td>
</tr>
<tr>
<td>STEMI complication</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Responders</td>
<td>(n=198)</td>
<td>(n=36)</td>
<td>(n=162)</td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>126 (63.6)</td>
<td>26 (72.2)</td>
<td>100 (61.7)</td>
<td>FE</td>
</tr>
<tr>
<td>Haematoma (^2)</td>
<td>9 (4.5)</td>
<td>1 (2.8)</td>
<td>0 (0.0)</td>
<td>FE</td>
</tr>
<tr>
<td>Bleed (NT)(^3)</td>
<td>6 (3.0)</td>
<td>0 (0.0)</td>
<td>8 (4.9)</td>
<td>FE</td>
</tr>
<tr>
<td>Bleed (T)(^4)</td>
<td>5 (2.5)</td>
<td>1 (2.8)</td>
<td>4 (2.5)</td>
<td>FE</td>
</tr>
<tr>
<td>PCI(^5)</td>
<td>7 (3.5)</td>
<td>2 (5.6)</td>
<td>5 (3.1)</td>
<td>FE</td>
</tr>
<tr>
<td>Allergy</td>
<td>5 (2.5)</td>
<td>0 (0.0)</td>
<td>5 (3.1)</td>
<td>FE</td>
</tr>
<tr>
<td>VF arrest</td>
<td>8 (4.0)</td>
<td>0 (0.0)</td>
<td>8 (4.9)</td>
<td>FE</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>6 (3.0)</td>
<td>1 (2.8)</td>
<td>5 (3.1)</td>
<td>FE</td>
</tr>
<tr>
<td>HF(^6)</td>
<td>2 (1.0)</td>
<td>1 (2.8)</td>
<td>1 (0.6)</td>
<td>FE</td>
</tr>
<tr>
<td>Balloon pump</td>
<td>8 (4.0)</td>
<td>2 (5.6)</td>
<td>6 (3.7)</td>
<td>FE</td>
</tr>
<tr>
<td>Other</td>
<td>16 (7.9)</td>
<td>2 (5.6)</td>
<td>14 (8.6)</td>
<td>FE</td>
</tr>
</tbody>
</table>

\(^1\)Left Bundle Branch Block, \(^2\)Haematoma PPCI puncture site, \(^3\)Bleed not requiring transfusion
\(^4\)Bleed requiring transfusion, \(^5\)PCI complication, \(^6\)Heart failure, \(^7\)Pearson Chi-Square test, \(^8\)Fisher’s exact test

In both groups over half the patients had normal LV function. However, more participants in the re-presentation group had severe LV dysfunction (LVEF <30) than in the non-representation group (16.7% vs 4.4%, Fisher exact test p=0.056). The majority of participants (94.1%) did not experience a cardiac arrest at the time of their STEMI (Table 7-8). Yet the complication rate at the time of STEMI and during admission was relatively high with almost 40.0% of all participants experiencing some kind of complication (Table 7-8).
7.3.4 Symptom variables

The New York Heart Association (NYHA) measure of heart failure prior to STEMI identified low levels of heart failure symptoms across the cohort (Table 7-9). More than three quarters of participants (77.5%) reported experiencing NYHA Class I and no participants reported Class IV. Likewise levels of angina prior to STEMI were also low with no pain being reported by 77.7% of participants and only 14.0% reporting Class 1 Canadian Cardiovascular Society Angina Grading Scale (CCSC) (Campeau, 1976), Table 7-9. See section 5.8.1, Appendix J and Appendix K for descriptions of NYHA and CCSC scores.

The Seattle angina Questionnaire scores for participants are discussed later in the results section.

Table 7-9 Categorical symptom characteristics by presentation

<table>
<thead>
<tr>
<th></th>
<th>All (n=202)</th>
<th>Re-presentation (n=38)</th>
<th>Non-representation (n=164)</th>
<th>Test (df)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>NYHA baseline</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class I</td>
<td>148 (77.5)</td>
<td>30 (83.3)</td>
<td>118 (76.1)</td>
<td>χ²=0.9</td>
<td>0.628</td>
</tr>
<tr>
<td>Class II</td>
<td>34 (17.8)</td>
<td>5 (13.9)</td>
<td>29 (18.7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class III</td>
<td>9 (4.7)</td>
<td>1 (2.8)</td>
<td>8 (5.2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CCSC baseline</td>
<td></td>
<td></td>
<td></td>
<td>FE</td>
<td>0.703</td>
</tr>
<tr>
<td>No pain</td>
<td>150 (77.7)</td>
<td>27 (73.0)</td>
<td>123 (78.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class 1</td>
<td>27 (14.0)</td>
<td>7 (18.9)</td>
<td>20 (12.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class 2</td>
<td>11 (5.7)</td>
<td>2 (5.4)</td>
<td>9 (5.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class 3</td>
<td>4 (2.1)</td>
<td>1 (2.7)</td>
<td>3 (1.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class 4</td>
<td>1 (0.5)</td>
<td>0 (0.0)</td>
<td>1 (0.6)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1 Pearson Chi-Square test
2 Fisher’s exact test
7.3.5 Comorbidity variables

The study cohort had very few co-morbidities at baseline with a predictive weighted index of comorbidity [Charlson Co-Morbidity Index (CCI)] mean 0.7 (SD=1.1, range 0 to 7). There was no significant difference between the groups in CCI scores at baseline (p=0.332) (Table 7-10). The CCI is discussed in detail in section 3.5.2.2 and 5.10.5. The occurrence of other specific co-morbidities was also low with only 19.8% of participants having diabetes and 15.8% having pulmonary disease (Table 7-11).

Table 7-10 Continuous comorbid condition characteristics by presentation

<table>
<thead>
<tr>
<th>Variable</th>
<th>All (n=202)</th>
<th>Re-presentation (n=38)</th>
<th>Non-representation (n=164)</th>
<th>Re-presentation vs Non-representation</th>
</tr>
</thead>
<tbody>
<tr>
<td>CCI</td>
<td>Mean (SD)</td>
<td>Median (range)</td>
<td>Mean (SD)</td>
<td>Median (range)</td>
</tr>
<tr>
<td></td>
<td>0.7 (1.1)</td>
<td>0.0 (0.0-7.0)</td>
<td>0.7 (1.0)</td>
<td>0.0 (0.0-3.0)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.6 (1.2)</td>
<td>0.0 (0.0-7.0)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Test</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Z=-0.97</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>p value 0.332</td>
</tr>
</tbody>
</table>

*Mann-Whitney U test

Table 7-11 Categorical comorbid condition characteristics by presentation

<table>
<thead>
<tr>
<th>Variable</th>
<th>All (n=202)</th>
<th>Re-presentation (n=38)</th>
<th>Non-representation (n=164)</th>
<th>Re-presentation vs Non-representation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes</td>
<td>N (%)</td>
<td>N (%)</td>
<td>N (%)</td>
<td>Test (df)</td>
</tr>
<tr>
<td>Yes</td>
<td>40 (19.8)</td>
<td>6 (15.8)</td>
<td>34 (20.7)</td>
<td>$^2$,$\chi^2$=0.48 (1)</td>
</tr>
<tr>
<td>No</td>
<td>162 (80.2)</td>
<td>32 (84.2)</td>
<td>130 (79.3)</td>
<td>0.491</td>
</tr>
<tr>
<td>Diabetes</td>
<td>(type)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>162 (80.2)</td>
<td>32 (84.2)</td>
<td>130 (79.3)</td>
<td></td>
</tr>
<tr>
<td>Type 1</td>
<td>1 (0.5)</td>
<td>1 (2.6)</td>
<td>0 (0.0)</td>
<td></td>
</tr>
<tr>
<td>Type 2</td>
<td>31 (15.3)</td>
<td>4 (10.5)</td>
<td>27 (16.5)</td>
<td></td>
</tr>
<tr>
<td>New$^1$</td>
<td>8 (4.0)</td>
<td>1 (2.6)</td>
<td>7 (4.3)</td>
<td></td>
</tr>
<tr>
<td>Pulmonary</td>
<td>N (%)</td>
<td>N (%)</td>
<td>N (%)</td>
<td>Test (df)</td>
</tr>
<tr>
<td>disease</td>
<td>Yes</td>
<td>32 (15.8)</td>
<td>7 (18.4)</td>
<td>$^2$,$\chi^2$=0.23 (1)</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>170 (84.2)</td>
<td>31 (81.6)</td>
<td>0.629</td>
</tr>
</tbody>
</table>

$^1$New diagnosis during index STEMI admission

$^2$Pearson Chi-Square test

$^3$Fisher’s exact test

7.3.6 Previous cardiac history variables

The majority of participants (76.1%) had not previously suffered a cardiac event, including angina, MI, cardiac arrhythmia or heart failure. For more than 85.0% of participants the index STEMI was their first heart attack, and only 10.9% had had one before and 4.0% two or more heart attacks previously. Previous cardiac revascularisation was also uncommon with only 8.4% having had a PCI and 4.0% a CABG. In the re-presentation group significantly more participants had previously been treated with PCI prior to their STEMI compared to the non-representation group (re-presentation group 21.1% vs non-representation group 5.5%; $\chi^2$=9.70,
Heart failure was only previously experienced by one participant and this was in the non-representation group (Table 7-12).

| Table 7-12 Categorical previous cardiac history characteristics by presentation |
|---------------------------------|-----------------|----------------|-----------------|-----------------|
|                                | ALL (n=202)     | Re-presentation (n=38) | Non-representation (n=164) | Re-presentation vs Non-representation |
|                                | N (%)           | N (%)         | N (%)           | Test (df)       | P value |
| Previous cardiac event | Yes | 48 (23.9) | 12 (31.6) | 36 (22.1) | $\chi^2=1.53$ (1) | 0.216 |
|                                | No  | 153 (76.1) | 26 (68.4)   | 127 (77.9)  |                |        |
| Multiple MIs                  | No  | 172 (85.1) | 31 (61.6)   | 141 (86.0)  |                |        |
|                                | 1   | 22 (10.9)   | 5 (13.2)    | 17 (10.4)   |                |        |
|                                | $\geq2$ | 8 (4.0)    | 2 (5.3)    | 6 (3.7)    |                |        |
| CABG                           | Yes | 8 (4.0)    | 2 (5.3)    | 6 (3.7)    | $\chi^2=9.70$ (1) | 0.002 |
|                                | No  | 194 (96.0) | 36 (94.7)   | 158 (96.3)  |                |        |
| PCI                            | Yes | 17 (8.4)   | 8 (21.1)    | 9 (5.5)    | $\chi^2=1.36$ (1) | 0.243 |
|                                | No  | 185 (91.6) | 30 (78.9)   | 155 (94.5)  |                |        |
| Heart Failure                  | Yes | 1 (0.5)    | 0 (0.0)     | 1 (0.6)    | $\chi^2=11.82$ (1) | <0.001 |
|                                | No  | 200 (99.5) | 38 (100)    | 162 (99.4)  |                |        |

*Pearson Chi-Square test

**7.3.7 Past psychological health variables**

Treatment for past anxiety was relatively uncommon and did not differ between the groups. In the re-presentation group 17.1% were treated for anxiety compared to 10.2% of the non-representation group (p=0.243). However, far more patients were treated for depression in the re-presentation group than the non-representation group. As many as 40.0% of the re-presentation group compared to 14.6% of the non-representation group received treatment for depression on one or more occasions prior to index STEMI (p<0.001) (Table 7-13)

| Table 7-13 Categorical previous psychological health characteristics by presentation group. |
|---------------------------------|-----------------|----------------|----------------|-----------------|
|                                | All (n=202)     | Re-presentation (n=35) | Non-representation (n=157) | Re-presentation vs Non-representation |
|                                | N (%)           | N (%)         | N (%)           | Test (df)       | P value |
| Treated for anxiety            | Yes | 22 (11.5) | 6 (17.1)  | 16 (10.2)  | $\chi^2=1.36$ (1) | 0.243 |
|                                | No  | 170 (88.5)| 29 (82.9)  | 141 (89.8) |                |        |
| Treated for depression         | Yes | 37 (19.3) | 14 (40.0)  | 23 (14.6)  | $\chi^2=11.82$ (1) | <0.001 |
|                                | No  | 155 (80.7)| 21 (60.0)  | 134 (85.4) |                |        |

*Pearson Chi-Square test

**7.3.8 Ischemic Heart Disease risk factors variables**

The number of risk factors experienced by participants was evenly spread between 1 and 3 risk factors and there was no significant difference in number of risk factors between the groups (Table 7-14). The risk factors included were smoking,
hypercholesterolaemia, family history of ischaemic heart disease, hypertension, diabetes and obesity.

Table 7.14 Categorical risk factor characteristics by presentation

| Number of risk factors | All (n=202) N (%) | Re-presentation (n=38) N (%) | Non-representation (n=163) N (%) | Test (df) | P value  \\
|------------------------|------------------|-----------------------------|---------------------------------|----------|---------  \\
| None                   | 9 (4.5)          | 1 (2.6)                     | 8 (4.9)                         | χ²(1) = 0.75 | 0.386    \\
| 1                      | 53 (26.4)        | 9 (23.7)                    | 44 (27.0)                       |          |          \\
| 2                      | 59 (29.2)        | 11 (28.9)                   | 48 (29.4)                       |          |          \\
| 3                      | 43 (21.3)        | 9 (23.7)                    | 34 (20.9)                       |          |          \\
| 4                      | 21 (10.4)        | 4 (10.5)                    | 17 (10.4)                       |          |          \\
| 5                      | 16 (1.9)         | 4 (10.5)                    | 12 (7.4)                        |          |          \\

1 Chi-Square test for trend

7.4 6 month analysis

7.4.1 Mortality

At 6 months there were a total of seven (3.0%) deaths for the total study cohort (231). In the responder group (202) there were three (1.5%) deaths, all of which occurred in the non-representation group. One of the deaths (patient 240) was related to a cardiac cause (0.5%) and the remaining two (1.0%), were non-cardiac related (both due to carcinoma; patients 31 and 251).
7.4.2 Cardiac revascularisation

Post PPCI revascularisation was low with only 20.0% of participants requiring either PCI (14.9%) or CABG (5.4%) between discharge and 6 month follow-up (Table 7-15). There was no significant difference between the groups in rates of revascularisation (p=0.599).

Table 7-15 Categorical cardiac revascularisation characteristics by presentation at 6 months.

<table>
<thead>
<tr>
<th></th>
<th>All (n=165)</th>
<th>Re-presentation (n=30)</th>
<th>Non-representation (n=135)</th>
<th>Re-presentation vs Non-representation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Further PCI / CABG</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>161 (79.7%)</td>
<td>29 (76.3%)</td>
<td>132 (80.5%)</td>
<td></td>
</tr>
<tr>
<td>Staged PCI</td>
<td>28 (13.9%)</td>
<td>5 (13.2%)</td>
<td>23 (14.0%)</td>
<td></td>
</tr>
<tr>
<td>CABG</td>
<td>11 (5.4%)</td>
<td>3 (7.9%)</td>
<td>8 (4.9%)</td>
<td></td>
</tr>
<tr>
<td>Acute PCI</td>
<td>2 (1.0%)</td>
<td>1 (2.6%)</td>
<td>1 (0.6%)</td>
<td></td>
</tr>
</tbody>
</table>

1 Fisher’s exact test

7.4.3 Symptom variables at 6 months

At 6 months the majority of the study cohort reported Class I heart failure symptoms measured using the NYHA (Table 7-16). Overall levels of heart failure were low and no participants reported NYHA Class IV.

Table 7-16 Categorical symptom characteristics by presentation at 6 months

<table>
<thead>
<tr>
<th></th>
<th>All (n=191)</th>
<th>Re-presentation (n=36)</th>
<th>Non-representation (n=155)</th>
<th>Re-presentation vs Non-representation</th>
</tr>
</thead>
<tbody>
<tr>
<td>NYHA 6 months</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class I</td>
<td>112 (55.4%)</td>
<td>20 (55.6%)</td>
<td>92 (59.4%)</td>
<td>χ²=6.05 (2) 0.109</td>
</tr>
<tr>
<td>Class II</td>
<td>53 (26.2%)</td>
<td>12 (33.3%)</td>
<td>41 (26.5%)</td>
<td></td>
</tr>
<tr>
<td>Class III</td>
<td>26 (12.9%)</td>
<td>4 (11.1%)</td>
<td>22 (14.2%)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>All (n=192)</th>
<th>Re-presentation (n=37)</th>
<th>Non-representation (n=155)</th>
<th>Re-presentation vs Non-representation</th>
</tr>
</thead>
<tbody>
<tr>
<td>CCSC 6 months</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No pain</td>
<td>139 (68.8%)</td>
<td>22 (59.5%)</td>
<td>117 (75.5%)</td>
<td>χ²=0.77 (3) 0.679</td>
</tr>
<tr>
<td>Class 1</td>
<td>26 (12.9%)</td>
<td>7 (18.9%)</td>
<td>19 (12.3%)</td>
<td></td>
</tr>
<tr>
<td>Class 2</td>
<td>19 (9.4%)</td>
<td>7 (18.9%)</td>
<td>12 (7.7%)</td>
<td></td>
</tr>
<tr>
<td>Class 3</td>
<td>8 (4.0%)</td>
<td>1 (2.7%)</td>
<td>7 (4.5%)</td>
<td></td>
</tr>
<tr>
<td>Class 4</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

1 Pearson Chi-Square test

Across the study cohort the majority of participants reported not experiencing any angina at all, as measured by the Canadian Cardiovascular Society Angina Grading Scale (CCSC) (Campeau, 1976), Table 7-9. The NYHA and CCSC scores are described in section 5.8.1, Appendix J and Appendix K.
The Seattle Angina Questionnaire scores for participants are discussed in section 7.4.5.2.

### 7.4.4 Cardiac rehabilitation variables

Cardiac rehabilitation was offered to more than 90.0% of participants and this was the same across the groups (Table 7-17). In the re-presentation group only 35.1% of participants attended all the cardiac rehabilitation sessions offered, compared to 56.8% of the non-representation group.

Table 7-17 Categorical cardiac rehabilitation characteristics by re-presentation

<table>
<thead>
<tr>
<th>Variable</th>
<th>All (n=202)</th>
<th>Re-presentation (n=37)</th>
<th>Non-representation (n=162)</th>
<th>Re-presentation vs Non-representation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N (%)</td>
<td>N (%)</td>
<td>N (%)</td>
<td>Test (df)</td>
</tr>
<tr>
<td>Offered CR $^1$</td>
<td>Yes</td>
<td>185 (93.0)</td>
<td>36 (97.3)</td>
<td>149 (92.0)</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>14 (7.0)</td>
<td>1 (2.6)</td>
<td>13 (8.0)</td>
</tr>
<tr>
<td>Attended CR $^1$</td>
<td>None</td>
<td>49 (24.6)</td>
<td>10 (27.0)</td>
<td>39 (24.1)</td>
</tr>
<tr>
<td></td>
<td>One</td>
<td>20 (10.1)</td>
<td>8 (21.6)</td>
<td>12 (7.4)</td>
</tr>
<tr>
<td></td>
<td>Some</td>
<td>18 (9.0)</td>
<td>6 (16.2)</td>
<td>12 (7.4)</td>
</tr>
<tr>
<td></td>
<td>All</td>
<td>105 (52.8)</td>
<td>13 (35.1)</td>
<td>92 (56.8)</td>
</tr>
</tbody>
</table>

$^1$Cardiac rehabilitation

$^2$ Fisher’s exact test

$^3$ Chi-Square test for trend
7.4.5 Self-report measure variables

7.4.5.1 HADS Anxiety, Depression and Psychological Health

The mean HADS anxiety scores for all participants improved at 6 months (Table 7-18). However, the mean HADS depression scores were relatively low and did not change at 6 months. The mean HADS psychological distress scores improved at 6 months (baseline mean 13.0 and 6 months 11.9).

Table 7-18 Levels of HADS sub-scales at baseline and 6 months and mean change for all participants.

<table>
<thead>
<tr>
<th>Variable</th>
<th>All baseline (n=202)</th>
<th>All 6 months (n=184)</th>
<th>0-6m change</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD) Median (range)</td>
<td>Mean (SD) Median (range)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>HADS Anxiety</td>
<td>7.5 (5.1) 7.0 (0-21)</td>
<td>6.7 (5.2) 6.0 (0-20)</td>
<td>-0.8 (3.4)</td>
</tr>
<tr>
<td>HADS Depression</td>
<td>5.6 (4.5) 4.0 (0-21)</td>
<td>5.2 (4.9) 3.0 (0-17)</td>
<td>-0.4 (3.2)</td>
</tr>
<tr>
<td>HADS Psychological distress</td>
<td>13.0 (9.0) 11.0 (0-42)</td>
<td>11.9 (9.7) 8.5 (0-37)</td>
<td>-1.2 (5.8)</td>
</tr>
</tbody>
</table>

The HADS cut offs at baseline and 6 months determined that close to half (44.1%) of participants at baseline reported HADS anxiety ≥8, which only improved slightly by 6 months. Furthermore, almost a third of patients (30.7%) reported baseline HADS depression scores ≥8; these also improved at 6 months for the total study cohort, Table 7-19.

Table 7-19 - HADS anxiety and depression scores, cut offs ≥8 and ≥11 and psychological distress scores cut offs ≥17 at baseline and 6 months for all participants

<table>
<thead>
<tr>
<th>Measure</th>
<th>All baseline n=202</th>
<th>All 6 months n=184</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety</td>
<td>N (%)</td>
<td>N (%)</td>
</tr>
<tr>
<td>HADS anxiety ≥8</td>
<td>89.0 (44.1)</td>
<td>76.0 (37.6)</td>
</tr>
<tr>
<td>HADS anxiety ≥11</td>
<td>52 (25.7)</td>
<td>46 (22.8)</td>
</tr>
<tr>
<td>Depression</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HADS depression ≥8</td>
<td>62.0 (30.7)</td>
<td>46.0 (22.8)</td>
</tr>
<tr>
<td>HADS depression ≥11</td>
<td>31.0 (15.3)</td>
<td>35.0 (17.3)</td>
</tr>
<tr>
<td>Psychological distress</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HADS psychological distress ≥17</td>
<td>67.0 (33.2)</td>
<td>56.0 (27.7)</td>
</tr>
</tbody>
</table>

7.4.5.2 Seattle Angina Questionnaire

The results of the Seattle Angina Questionnaire (SAQ) for angina stability, physical limitation scores, angina frequency and treatment satisfaction did not change from
baseline to 6 months. However, mean SAQ quality of life scores for all participants improved from baseline to 6 months, Table 7-20.

Table 7-20 Description of levels of SAQ at baseline and 6 months and mean change at 6 months for all participants

<table>
<thead>
<tr>
<th>Variable</th>
<th>All baseline (n=202)</th>
<th>All 6 months (n=184)</th>
<th>0-6m change</th>
<th>Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAQ angina stability</td>
<td>Mean (SD)</td>
<td>Median (range)</td>
<td>Mean (SD)</td>
<td>Median (range)</td>
</tr>
<tr>
<td>75.0 (32.0)</td>
<td>100.0 (0-100)</td>
<td>74.6 (28.5)</td>
<td>75.0 (0-100)</td>
<td>-0.4 (36.6)</td>
</tr>
<tr>
<td>SAQ physical limitation</td>
<td>66.4 (28.8)</td>
<td>100.0 (0-100)</td>
<td>69.0 (26.9)</td>
<td>75.0 (14-100)</td>
</tr>
<tr>
<td>SAQ angina frequency</td>
<td>81.9 (21.4)</td>
<td>90.0 (10-100)</td>
<td>84.2 (23.0)</td>
<td>100 (0-100)</td>
</tr>
<tr>
<td>SAQ treatment satisfaction</td>
<td>89.0 (13.8)</td>
<td>93.8 (38-100)</td>
<td>88.6 (15.6)</td>
<td>93.6 (25-100)</td>
</tr>
<tr>
<td>SAQ quality of life</td>
<td>54.6 (26.9)</td>
<td>50.0 (0-100)</td>
<td>66.5 (26.3)</td>
<td>75.0 (0-100)</td>
</tr>
</tbody>
</table>

^Paired- t test

7.4.6 Summary for descriptive results

At baseline the study groups were very similar. They varied significantly in three ways: more participants in the re-presentation group had severe LV dysfunction (LVEF <30), had previously been treated for depression prior to their index STEMI and had previously been treated with PCI. Furthermore, at 6 months fewer of the re-presentation group had also attended the entire cardiac rehabilitation programme.

The findings of the HADS measures indicated that anxiety and psychological distress improved from baseline to 6 months for the study cohort. Mean depression scores were relatively low and remained the same at 6 months. The improvement in HADS psychological distress scores from baseline to 6 months were mainly influenced by the reduction in anxiety scores.

The measurement of angina symptoms using the Seattle Angina Questionnaire showed good stability of angina and infrequency of symptoms across the cohort both at baseline and 6 months. However, participants reported some physical limitations and also reduced quality of life; quality of life significantly improved at 6 months.
7.5 Aim 1: Number and frequency of re-presentations

Aim 1: To identify the number and frequency of re-presentations due to potential IHD symptoms, by determining and categorising the reasons for re-presentation during the first 6 months post STEMI.

7.5.1 Introduction

The number, frequency and reason for acute re-presentations due to potential IHD events are presented for the study cohort. In the re-presentation group all acute re-presentations including non-potential IHD events were recorded and are presented.

The categorisation of reasons for re-presentation was undertaken in accordance with the patients final discharge diagnosis. The process of categorising the reasons for re-presentation are discussed in detail in section 5.18.

7.5.2 The number of re-presentations

The re-presentation group consisted of 38 (18.8%; 95% CI 14.0% to 24.8%) individuals who suffered one or more potential IHD events requiring attendance at acute healthcare service during 6 month follow-up. At 30 days, 14 (6.9%) participants had attended with their first re-presentation, which constituted 29.0% of the re-presentation group. Time to first re-presentation was mean 76.5 days (median 51.0, SD 79.8 days).

Conversely, the non-representation group included 164 individuals who had not accessed acute healthcare services for a potential IHD event during the 6 month study follow-up, although three of the group had died by 6 months. Ten individuals in the non-representation group had re-presented to acute healthcare services but none of the events were identified as potential IHD events according to a priori definition (see section 5.8.3).

7.5.3 The frequency of re-presentations

A total of 74 events occurred across the re-presentation group during the 6 month follow-up. In the re-presentation group, the majority (28) of the 38 participants had either one (16) or two (12) attendances at acute healthcare services (Table 7-21).
Table 7-21 Details of acute re-presentations for the groups at 6 months

<table>
<thead>
<tr>
<th>Number of acute re-presentations</th>
<th>Re-presentation group N (%)</th>
<th>Non-representation group N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>0</td>
<td>154 (93.9)</td>
</tr>
<tr>
<td>One</td>
<td>16 (42.1)</td>
<td>9 (5.5)</td>
</tr>
<tr>
<td>Two</td>
<td>12 (31.6)</td>
<td>1 (0.6)</td>
</tr>
<tr>
<td>Three</td>
<td>7 (18.4)</td>
<td>0</td>
</tr>
<tr>
<td>Four</td>
<td>2 (5.3)</td>
<td>0</td>
</tr>
<tr>
<td>Five</td>
<td>1 (2.6)</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>38</td>
<td>164</td>
</tr>
</tbody>
</table>

The largest number of re-presentations recorded was five and this occurred for only one individual (participant 13). Participant 13 suffered one non-cardiac event (an allergic reaction) and four events with symptoms of chest pain. Two of the chest pain events were diagnosed as severe episodes of depression (requiring admission to a psychiatric hospital), the further two chest pain events were not assigned a final diagnosis. Two individuals had four re-presentations (participants 374 and 421). Participant 374 had four chest pain events: one was a cardiac ischaemic event, two were due to musculoskeletal pain and one was not assigned a final diagnosis. Participant 421 suffered one non-cardiac event (bleed due to antiplatelet therapy) and three episodes of chest pain. One of the chest pain events was diagnosed as N-STEMI and the other two without a final diagnosis; the final attendance was recorded as an A&E attendance and no initial or discharge diagnosis was assigned.

In the non-representation group only 6.1% of individuals suffered one or more acute re-presentations (only one individual suffered more than one acute event). All were non-cardiac and did not involve potential IHD events.

### 7.5.4 Categorising the reasons for re-presentation

Twenty-two (57.9%) of the 38 participants who re-presented during the 6 month follow-up did not receive a clear diagnosis on at least one occasion and were categorised as ‘no diagnosis’; this amounts to 10.9% of the 202 responders. The category ‘no diagnosis’ included comments such as “not likely to be cardiac”, “chest pain of uncertain cause”, “non-specific chest pain” or “troponin T’ negative chest pain” and in a number of cases there was an absence of any diagnosis.

A diagnosis of cardiac ischaemia or cardiac cause was received at least once by 16 (42.1%) of the re-presentation group, which is 7.9% of the 202 respondents. The category cardiac ischaemia or cardiac cause included angina, ACS, N-STEMI, further STEMI and cardiac arrhythmia. Nineteen (9.4%) of the 202 respondents either died
(3) or experienced a cardiac ischaemic or cardiac cause re-presentation event (16) on at least one occasion. A cardiac ischaemic event was experienced by 13 of the 38 re-presenters (34.2%); the three others (patients 258, 408, 422) suffered a cardiac arrhythmia.

Across the 74 re-presentation events, the highest percentage (39.2%) were categorised as ‘no diagnosis’. The second most common discharge diagnosis was cardiac ischaemia or cardiac cause (24.3%) (Table 7-22).

Table 7-22 Final diagnostic categories for events in the re-presentation group (all acute attendances included)

<table>
<thead>
<tr>
<th>Diagnosis category name</th>
<th>First * (%)</th>
<th>Second * (%)</th>
<th>Third * (%)</th>
<th>Fourth * (%)</th>
<th>Fifth * (%)</th>
<th>Total N ** (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Did not re-present</td>
<td>0</td>
<td>16 (42.1)</td>
<td>28 (73.7)</td>
<td>34 (89.5)</td>
<td>37 (97.4)</td>
<td>29 (39.2)</td>
</tr>
<tr>
<td>No diagnosis</td>
<td>10 (26.4)</td>
<td>12 (31.6)</td>
<td>3 (7.9)</td>
<td>3 (7.9)</td>
<td>1 (2.6)</td>
<td>18 (24.3)</td>
</tr>
<tr>
<td>Cardiac ischaemia or</td>
<td>12 (31.6)</td>
<td>4 (10.5)</td>
<td>2 (5.3)</td>
<td>0</td>
<td>0</td>
<td>18 (24.3)</td>
</tr>
<tr>
<td>cardiac cause</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart failure</td>
<td>4 (10.5)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>4 (5.4)</td>
</tr>
<tr>
<td>Psychological</td>
<td>0</td>
<td>2 (5.3)</td>
<td>1 (2.6)</td>
<td>0</td>
<td>0</td>
<td>3 (4.1)</td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>3 (7.9)</td>
<td>1 (2.6)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>4 (5.4)</td>
</tr>
<tr>
<td>Gastric</td>
<td>2 (5.3)</td>
<td>1 (2.6)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3 (4.1)</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>2 (5.3)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2 (2.6)</td>
</tr>
<tr>
<td>Non-cardiac</td>
<td>5 (13.2)</td>
<td>2 (5.3)</td>
<td>4 (10.5)</td>
<td>0</td>
<td>0</td>
<td>11 (14.9)</td>
</tr>
<tr>
<td>Total</td>
<td>38</td>
<td>38</td>
<td>38</td>
<td>38</td>
<td>38</td>
<td>74</td>
</tr>
</tbody>
</table>

*percentage of 38 participants to have this discharge diagnosis, **percentage of the total number of representation events 74 (100%)

7.5.5 Summary for aim 1

Thirty-eight (18.8%; 95% CI 14.0% to 24.8%) of the 202 responders re-presented to acute healthcare services with at least one potential IHD event during the 6 month study follow-up. Re-presentation due to non-cardiac events in the non-representation group was relatively uncommon (10 participants, 6.1%).

In the case of 57.9%) of the re-presentation group a clear diagnosis relating to their re-presentation attendance was not evident in their healthcare records on at least one occasion. Sixteen (42.1%) of the re-presentation group sought help via acute services for a cardiac ischemic or cardiac event at least once. Additionally a total of 74 events were reported for the re-presentation group with the highest proportion (32.9%) being categorised as ‘no diagnosis’ and nearly a quarter (24.3%) due to cardiac ischaemia or cardiac cause.
7.6 Aim 2: Levels of psychological health and angina symptoms

Aim 2: To determine and compare the level of anxiety, depression, psychological distress and angina symptoms (angina stability, angina frequency, physical limitation, treatment satisfaction and quality of life) between the two groups at baseline and at 6 months.

7.6.1 Introduction

Levels of HADS anxiety and depression were interpreted according to Zigmond and Snaith’s (1983) classification, normal (0-7), mild (8-10), moderate (11-14) and severe (15-21). HADS psychological distress was also measured using a total HADS score ≥17 to indicate probable major depressive disorder (Dickens et al., 2004). Further details of the HADS measures can be found in section 5.10.1.3. The HADS anxiety and depression score cut offs (≥8 and ≥11), along with the HADS psychological distress cut off (≥17) are also presented for both groups. The HADS anxiety, depression and psychological distress distributions are presented in Appendix L.

Comparison of results between the groups at baseline and 6 months for angina symptoms (SAQ sub-scales angina stability, angina frequency, physical limitation, treatment satisfaction and quality of life) are presented in the tables in sections 7.6.2 and 7.6.3. In accordance with the recommendations of Spertus et al (1995), SAQ scale scores (0 – 100 range) were interpreted with higher scores representing better health status. A ten point change in score was also considered clinically important (SAQ change scores can be seen under aim 3 in section 7.7). The distribution of the two groups for each SAQ scale at baseline and 6 months are presented in Appendix L.

7.6.2 Hospital Anxiety and Depression Scale results

7.6.2.1 HADS anxiety

Baseline: The mean HADS anxiety scores at baseline in the re-presentation group demonstrated mild anxiety according to the subscales defined by Zigmond and Snaith (1983) (see section 7.6.1). The mean score for the non-representation group demonstrated normal levels of anxiety. There was a significant difference between the groups at baseline with the re-presentation group having higher mean scores (9.53 and 7.05, p=0.004) (Table 7-23). At baseline a significantly higher
percentage of individuals in the re-presentation group reported HADS anxiety scores ≥8 (re-presentation group 65.8% vs non-representation group 39.0%, p=0.003) and ≥11 (39.5% vs 22.6%, p=0.032), Table 7-25.

6 months: Mean HADS anxiety scores were higher in the re-presentation compared to the non-representation group (means 9.4 and 6.0, p<0.001) at 6 months. Mean scores had remained raised in the re-presentation group but had decreased in the non-representation group (Table 7-24). The percentage of individuals with ≥8 and ≥11 HADS anxiety scores continued to be higher at 6 months in the re-presentation than non-representation group (≥8, p=0.003 and ≥11, p=0.032), Table 7-25.

7.6.2.2 HADS depression

Baseline: The HADS depression mean score was higher in the re-presentation than non-representation group (means 7.5 and 5.3, p=0.004) at baseline. Mean scores demonstrated mild depression in the re-presentation group and no evidence of depression in the non-representation group (Table 7-23). At baseline a higher percentage of individuals in the re-presentation group reported HADS depression scores ≥8 (47.4% re-presentation group and non-representation group 26.8%, p=0.013) and ≥11 (28.9% vs 12.2%, p=0.010) (Table 7-25).

6 months: Mean HADS depression scores increased at 6 months and continued to demonstrate mild depression for the re-presentation group; however in the non-representation group mean scores decreased and remained within the normal range (means 8.4 and 4.4). There was a significant difference in HADS depression mean scores between the groups (p<0.001) (Table 7-24). At 6 months a significantly higher percentage of individuals in the re-presentation compared to non-representation group reported higher HADS depression scores ≥8 (51.4% vs 18.4%, p<0.001) and ≥11 (43.2% vs 12.9%, p<0.001) (Table 7-25).

7.6.2.3 HADS psychological distress

Baseline: Higher mean HADS psychological distress scores were seen in the re-presentation compared to non-representation group (means 17.1 and 12.4, p=0.003) at baseline. This was due to the higher levels of both anxiety and depression at baseline for the re-presentation group compared to the non-representation group (Table 7-23). At baseline, a higher percentage of individuals in the re-presentation group had HADS psychological distress scores ≥17 than in the non-representation group (50.0% vs 29.3%, p=0.014) (Table 7-25).
6 months: Mean HADS psychological distress scores remained higher in the re-presentation group than the non-representation group (means 17.8 and 10.4, \( p<0.001 \)) at 6 months (Table 7-24). At 6 months, the percentage of individuals in the re-presentation group with HADS psychological distress scores \( \geq 17 \) increased and in the non-representation group the percentage of individuals with \( \geq 17 \) decreased, with a highly significant difference between the groups (54.1% vs 24.5%, \( p<0.001 \)) (Table 7-25).

Table 7-23 Comparison of HADS anxiety, depression and psychological distress means at baseline between the groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Re-presentation group baseline (n=38)</th>
<th>Non-representation group baseline (n=164)</th>
<th>Re-presentation vs Non-representation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Median (range)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>Х HADS anxiety</td>
<td>9.5 (4.8)</td>
<td>9.5 (0-18)</td>
<td>7.1 (5.0)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>∞ HADS depression</td>
<td>7.5 (4.7)</td>
<td>7 (0-19)</td>
<td>5.3 (4.3)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>¥ HADS psychological distress</td>
<td>17.1 (8.9)</td>
<td>16 (0-36)</td>
<td>12.3 (8.7)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Х HADS anxiety distribution normal for re-presentation group and non-normal for non-representation group.

∞ HADS depression scores distribution for both groups was non-normal at baseline.

¥ HADS psychological distress distribution for re-presentation group was normal and for non-representation group non-normal.

\(^1\)Mann-Whitney U test
\(^2\)Independent- \( t \) test
### Table 7-24 Comparison of HADS anxiety, depression and psychological distress means at 6 months between the groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Re-presentation group 6 months (n=37)</th>
<th>Non-representation group 6 months (n=147)</th>
<th>Re-presentation vs Non-representation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Median (range)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>Ж HADS anxiety</td>
<td>9.4 (5.0)</td>
<td>9.0 (0-20)</td>
<td>6.0 (5.0)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>∞ HADS depression</td>
<td>8.4 (5.3)</td>
<td>9.0 (0-17)</td>
<td>4.4 (4.5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>¥ HADS psychological distress</td>
<td>17.8 (9.8)</td>
<td>20 (0-37)</td>
<td>10.4 (9.1)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Ж HADS anxiety distribution for re-presentation group normal and for non-representation group non-normal.
∞ HADS depression distribution for re-presentation group was normal and for non-representation group non-normal.
¥ HADS psychological distress distribution was normal for the re-presentation group and non-normal for the non-representation group.
^Mann-Whitney U test
^Independent- t test

### Table 7-25 HADS anxiety and depression scores, cut offs at ≥8 and ≥11 and psychological distress scores cut offs ≥17 at baseline and 6 months.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Re-presentation cut offs (%)</th>
<th>Non-representation cut offs (%)</th>
<th>*95% CI for difference</th>
<th>χ²(df)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>≥8 &lt;8</td>
<td>≥8 &lt;8</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HADS anxiety baseline</td>
<td>25 (65.8)</td>
<td>13 (34.2)</td>
<td>64 (39.0)</td>
<td>100 (61.0)</td>
<td>9.1%, 41.6%</td>
</tr>
<tr>
<td>HADS anxiety 6 months</td>
<td>25 (67.6)</td>
<td>12 (32.4)</td>
<td>51 (34.7)</td>
<td>96 (65.3)</td>
<td>14.9%, 47.6%</td>
</tr>
<tr>
<td></td>
<td>&gt;11 &lt;11 ≥11 &lt;11</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HADS anxiety baseline</td>
<td>15 (39.5)</td>
<td>23 (60.5)</td>
<td>37 (22.6)</td>
<td>127 (77.4)</td>
<td>1.4%, 33.7%</td>
</tr>
<tr>
<td>HADS anxiety 6 months</td>
<td>16 (43.2)</td>
<td>21 (56.8)</td>
<td>30 (20.4)</td>
<td>117 (79.6)</td>
<td>6.6%, 39.7%</td>
</tr>
<tr>
<td>Depression</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HADS depression baseline</td>
<td>18 (47.4)</td>
<td>20 (52.6)</td>
<td>44 (26.8)</td>
<td>120 (73.2)</td>
<td>4.0%, 37.1%</td>
</tr>
<tr>
<td>HADS depression 6 months</td>
<td>19 (51.4)</td>
<td>18 (48.6)</td>
<td>27 (18.4)</td>
<td>120 (81.6)</td>
<td>16.0%, 49.1%</td>
</tr>
<tr>
<td></td>
<td>&gt;11 &lt;11 ≥11 &lt;11</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HADS depression baseline</td>
<td>11 (28.9)</td>
<td>27 (71.1)</td>
<td>20 (12.2)</td>
<td>144 (87.8)</td>
<td>3.4%, 33.1%</td>
</tr>
<tr>
<td>HADS depression 6 months</td>
<td>16 (43.2)</td>
<td>21 (56.8)</td>
<td>19 (12.9)</td>
<td>128 (87.1)</td>
<td>14.4%, 46.8%</td>
</tr>
<tr>
<td>Psychological distress</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HADS psychological distress baseline</td>
<td>19 (50.0)</td>
<td>19 (50.0)</td>
<td>48 (29.3)</td>
<td>116 (70.7)</td>
<td>3.9%, 37.2%</td>
</tr>
<tr>
<td>HADS psychological distress 6 months</td>
<td>20 (54.1)</td>
<td>17 (45.9)</td>
<td>36 (24.5)</td>
<td>111 (75.5)</td>
<td>12.2%, 45.7%</td>
</tr>
</tbody>
</table>

*95% Confidence Interval for difference in percentage of abnormal scores (i.e. ≥8, ≥11 and ≥17)
7.6.3 Seattle Angina Questionnaire (SAQ) Results

7.6.3.1 SAQ Angina Stability

**Baseline:** Angina stability was relatively good at baseline and did not differ between the re-presentation and non-representation groups (mean 68.9 and 75.0, p=0.148) (Table 7-26). Scores closer to 100 demonstrate increased stability (Spertus et al., 1995).

**6 months:** Mean SAQ angina stability scores at 6 months remained relatively good and again there was no difference between the groups (re-presentation group mean 69.1 and non-representation group 76.4, p=0.450) (Table 7-27).

7.6.3.2 SAQ Angina Frequency

**Baseline:** The mean SAQ angina frequency results demonstrated that angina occurred infrequently for both groups at baseline (re-presentation group mean 78.7 and non-representation group 82.6, p=0.159). This was demonstrated by the high scores in both groups; a high score indicates less frequent angina (Table 7-26).

**6 months:** Angina frequency remained infrequent with relatively high mean scores (re-presentation group 74.7 vs non-representation group 86.5) at 6 months (Table 7-27). However, angina occurred more frequently in the re-presentation group than the non-representation group at 6 months (p=0.011).

7.6.3.3 SAQ Physical Limitation

**Baseline:** Physical limitation was slightly reduced in both groups (means 63.2 and 65.7, p=0.688), Table 7-26. Scores close to 100 demonstrate no physical limitation and zero indicates severe limitation. Mean scores in both groups were closer to 50 demonstrating moderate physical limitation.

**6 months:** Physical limitation remained slightly reduced in the re-presentation group, but improved slightly for the non-representation group; however, there was no significant difference between the groups (means 61.8 and 70.4, p=0.069) (Table 7-27).

7.6.3.4 SAQ Treatment Satisfaction

**Baseline:** Treatment satisfaction in both groups was high at baseline (representation group mean 86.7 and non-representation group mean 89.1, p=0.750). Scores of 100 demonstrate high levels of satisfaction (Table 7-26).
6 months: SAQ treatment satisfaction mean scores remained high for both groups (representation 86.0 and non-representation group 89.2, p=0.247) at 6 months (Table 7-27).

7.6.3.5 SAQ Quality of Life

Baseline: Quality of life in both groups was moderately reduced with mean scores being close to 50 (52.0 and 55.8, p=0.411) (Table 7-26). Scores of 100 demonstrate good quality of life, where as zero indicates poor quality of life.

6 months: There was a slight improvement in SAQ quality of life for the representation group, although mean scores (56.8) remained moderately reduced. In the non-representation group mean scores improved (69.7) at 6 months. SAQ quality of life was slightly different between the groups (p=0.003) (Table 7-27).

Table 7-26 Comparison of SAQ scales at baseline between the groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Re-presentation group baseline (n=38)</th>
<th>Non-representation group baseline (n=164)</th>
<th>Re-presentation vs Non-representation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Median (range)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>* Angina stability</td>
<td>68.9 (32.0)</td>
<td>75.0 (0-100)</td>
<td>75.0 (32.8)</td>
</tr>
<tr>
<td>† Angina frequency</td>
<td>78.7 (20.4)</td>
<td>80.0 (30-100)</td>
<td>82.60 (21.8)</td>
</tr>
<tr>
<td>Ж Physical limitation</td>
<td>63.2 (29.0)</td>
<td>64.0 (3-100)</td>
<td>65.7 (28.7)</td>
</tr>
<tr>
<td>‡ Treatment satisfaction</td>
<td>86.7 (14.0)</td>
<td>93.8 (56-100)</td>
<td>89.1 (13.6)</td>
</tr>
<tr>
<td>◊ Quality of life</td>
<td>52.0 (29.4)</td>
<td>50.0 (0-100)</td>
<td>55.8 (26.3)</td>
</tr>
</tbody>
</table>

*Angina stability distribution in both groups was non-normal
† Angina frequency distribution in both groups was non-normal
Ж Physical limitation distribution in both groups was non-normal
‡ Treatment satisfaction distribution in both groups was non-normal
◊ Quality of life distribution was normal in both groups
1Mann-Whitney U test
2Independent- t test
Table 7-27 Comparison of SAQ scales at 6 months between the groups.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Re-presentation group 6 months (n=37)</th>
<th>Non-representation group 6 months (n=147)</th>
<th>Re-presentation vs Non-representation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Median (range)</td>
<td>Test (df)</td>
</tr>
<tr>
<td>Angina stability</td>
<td>69.1 (35.4)</td>
<td>88 (0-100)</td>
<td>MWU</td>
</tr>
<tr>
<td></td>
<td>76.4 (26.4)</td>
<td>100 (0-100)</td>
<td></td>
</tr>
<tr>
<td>Angina frequency</td>
<td>74.7 (29.4)</td>
<td>90 (0-100)</td>
<td>MWU</td>
</tr>
<tr>
<td></td>
<td>86.5 (21.0)</td>
<td>100 (0-100)</td>
<td></td>
</tr>
<tr>
<td>Physical limitation</td>
<td>61.8 (25.8)</td>
<td>63 (14-100)</td>
<td>MWU=19</td>
</tr>
<tr>
<td></td>
<td>70.4 (26.9)</td>
<td>78 (14-100)</td>
<td>Z= -1.82</td>
</tr>
<tr>
<td>Treatment satisfaction</td>
<td>86.0 (17.3)</td>
<td>87.5 (25-100)</td>
<td>MWU=19</td>
</tr>
<tr>
<td></td>
<td>89.2 (15.6)</td>
<td>100.0 (25-100)</td>
<td>Z= -1.16</td>
</tr>
<tr>
<td>Quality of life</td>
<td>56.8 (24.5)</td>
<td>58.3 (0-100)</td>
<td>t=2.71 (182)</td>
</tr>
<tr>
<td></td>
<td>69.7 (26.3)</td>
<td>75.0 (0-100)</td>
<td>MWU=2.98</td>
</tr>
</tbody>
</table>

* Angina stability distribution in both groups was non-normal
† Angina frequency distribution in both groups was non-normal
Ж Physical limitation distribution in both groups was non-normal
‡ Treatment satisfaction distribution in both groups was non-normal
◊ Quality of life distribution for the re-presentation group was normal and non-representation group non-normal
1Mann-Whitney U test
2Independent- t test

7.6.4 Summary for aim 2

At both baseline and 6 months mean HADS anxiety scores were higher in the re-presentation than non-representation group. In the re-presentation group there were mild levels of anxiety (baseline mean 9.5 and 6 month mean 9.4), yet in the non-representation group mean scores were in the normal range (baseline 7.1 and 6 month 6.0).

Likewise, mean HADS depression scores at baseline and 6 months demonstrated mild levels of depression in the re-presentation group (7.5 and 8.4). Depression scores were within the normal range for the non-representation group (mean 5.3 and mean 4.4). Interestingly at 6 months, depression scores increased in the re-presentation group but decreased in the non-representation group. HADS psychological distress mean scores mirrored those of HADS anxiety and depression with higher scores in the re-presentation compared to non-representation group.

There was overall low burden of angina symptoms reflected by low frequency and high stability of symptoms as measured by the SAQ sub-scales. In particular, for both groups at baseline and 6 months angina stability was relatively good. Likewise the occurrence of angina across the groups was infrequent (as measured by SAQ angina frequency) at both baseline and 6 months. Despite infrequent angina, at 6
months the re-presentation group did experience angina more often than the non-representation group.

Physical limitation due to angina was moderate for both groups at baseline. Physical limitation worsened slightly at 6 months for the re-presentation group but improved for the non-representation group; however there was not a statistically significant difference between the groups.

Treatment satisfaction was high across the groups at baseline and 6 months. However, quality of life at baseline across the groups was moderately reduced. At 6 months there was a slight improvement for the re-presentation group, but quality of life remained moderately reduced. A larger improvement was seen in the non-representation group leading to a significant difference between the groups and only a slight reduction in quality of life for the non-representation group.

7.7 Aim 3: Change in levels of psychological health and angina symptoms

Aim 3: To determine the change in the level of anxiety, depression, psychological distress and angina symptoms within the groups from baseline to 6 months.

7.7.1 Introduction

The analysis for aim 3 (comparison from baseline to 6 months) included cases that had both baseline and 6 month measures; cases that did not have a 6 month measure were excluded. This resulted in small differences in mean values between aim 2 and aim 3. The change distributions for HADS anxiety, depression and psychological distress and SAQ scales are presented in Appendix L.

Details relating to the interpretation of HADS scores (and whether scores indicate normal or raised levels of anxiety, depression and psychological distress) are presented in sections 5.10.1.3 and 7.6.1. Details of SAQ scale interpretation can also be seen in sections 5.10.2.3 and 7.6.1. A change in SAQ sub-scale score of 10 point is considered clinically relevant (Spertus et al., 1995). Therefore SAQ change results from baseline to 6 months are presented as both whole numbers and as 10 point cut offs (including the categories no change, ≤-10 or ≥+10).
7.7.2 Hospital Anxiety and Depression Scale results

7.7.2.1 HADS Anxiety change scores

There was no change in the mean HADS anxiety scores for the re-presentation group from baseline to 6 months (9.4 vs 9.4, p=0.964) (Table 7-28). The non-representation group mean HADS anxiety score significantly improved at 6 months (using a paired-t test, mean change -0.97; baseline mean 7.0 and 6 month mean 6.0, p<0.001) [using Wilcoxon matched-pairs signed-ranks (WMPSR) test p<0.001] (Table 7-28).

Table 7-28 HADS change scores for the re-presentation group

<table>
<thead>
<tr>
<th>Variable</th>
<th>Time point</th>
<th>Change</th>
<th>Baseline vs 6 months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 Mean (SD)</td>
<td>6 Mean (SD)</td>
<td>Change mean (SD)</td>
</tr>
<tr>
<td>Ж HADS anxiety</td>
<td>9.4 (4.9)</td>
<td>9.4 (5.0)</td>
<td>0.0 (3.7)</td>
</tr>
<tr>
<td>※ HADS depression</td>
<td>7.4 (4.6)</td>
<td>8.4 (5.3)</td>
<td>1.1 (3.5)</td>
</tr>
<tr>
<td>¥ HADS psychological distress</td>
<td>16.8 (8.8)</td>
<td>17.8 (9.8)</td>
<td>1.1 (6.1)</td>
</tr>
</tbody>
</table>

Ж HADS anxiety change scores distribution was normal for the re-presentation group.
※ HADS depression change scores distribution was normal for the re-presentation group.
¥ HADS psychological distress change scores distribution was normal for the re-presentation group.

1 Paired-t test

7.7.2.2 HADS depression change scores

In the re-presentation group there was a non-significant worsening in the mean HADS depression scores at 6 months with a mean change of 1.1 (using a paired-t test) (baseline mean 7.4 vs 6 month mean 8.4, p=0.073), Table 7-28. In the non-representation group there was a significant improvement in the mean HADS depression scores at 6 months with a mean change of -0.77 (using a paired-t test, 5.1 vs 4.4, p=0.003), (using a WMPSR test, p<0.001), Table 7-29.

7.7.2.3 HADS psychological distress change scores

In the re-presentation group there was a non-significant worsening of mean HADS psychological distress scores from baseline to 6 months with a mean change of 1.1 (using a paired-t test) (16.8 vs 17.8, p=0.287), Table 7-28. In the non-representation group there was a significant improvement in mean HADS psychological distress scores at 6 months, with a mean change of -1.7 (using a paired-t test, baseline mean 12.1 vs 6 month mean 10.4, p<0.001), (using a WMPSR test, p<0.001), Table 7-29.
Table 7-29 - HADS change scores for the non-representation group

<table>
<thead>
<tr>
<th>Variable</th>
<th>Time point</th>
<th>Change</th>
<th>Baseline vs 6 months</th>
<th>Test result</th>
<th>df</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 m mean (SD)</td>
<td>6 m Mean (SD)</td>
<td>Change mean (SD)</td>
<td>Change median (range)</td>
<td>Test result</td>
<td>df</td>
</tr>
<tr>
<td>Anxiety</td>
<td>7.0 (5.0)</td>
<td>6.0 (5.0)</td>
<td>-0.97 (3.29)</td>
<td>-1.0 (-12, 8)</td>
<td>t = 3.59</td>
<td>146</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>WMPSR Z = -3.34</td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>5.1 (4.4)</td>
<td>4.4 (4.5)</td>
<td>0.77 (3.05)</td>
<td>-1.0 (-11, 11)</td>
<td>t = 3.05</td>
<td>146</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>WMPSR Z = -3.50</td>
<td></td>
</tr>
<tr>
<td>Psychological distress</td>
<td>12.1 (8.8)</td>
<td>10.4 (9.1)</td>
<td>-1.7 (5.6)</td>
<td>-2.0 (-23, 17)</td>
<td>t = 3.77</td>
<td>146</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>WMPSR Z = -4.17</td>
<td></td>
</tr>
</tbody>
</table>

* HADS anxiety change score distribution was non-normal for the non-representation group.
* HADS depression change score distribution was not normal for the non-representation group.
* HADS psychological distress change scores were non-normal for the non-representation group.
1 Paired-t test
2 Wilcoxon matched-pairs signed-ranks test

Table 7-30 - HADS anxiety and depression scores, cut offs ≥8 and ≥11 and psychological distress scores cut offs ≥17 baseline to 6 months for the re-presentation group

<table>
<thead>
<tr>
<th>Measure</th>
<th>Re-presentation group cut offs (n=37)</th>
<th>McNemar Test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>P value</td>
</tr>
<tr>
<td>HADS anxiety 6 months</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td>&lt;8</td>
<td>≥8</td>
</tr>
<tr>
<td>HADS anxiety baseline</td>
<td>&lt;8</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>≥8</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>&lt;11</td>
<td>19</td>
</tr>
<tr>
<td></td>
<td>≥11</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.687</td>
</tr>
<tr>
<td>HADS depression 6 months</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td>&lt;8</td>
<td>≥8</td>
</tr>
<tr>
<td>HADS depression baseline</td>
<td>&lt;8</td>
<td>17</td>
</tr>
<tr>
<td></td>
<td>≥8</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>&lt;11</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>≥11</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.070</td>
</tr>
<tr>
<td>Psychological distress</td>
<td>&lt;17</td>
<td>≥17</td>
</tr>
<tr>
<td>HADS psychological distress 6 months</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 7-31 - HADS anxiety and depression scores, cut offs ≥8 and ≥11 and psychological distress scores cut offs ≥17 baseline to 6 months for the non-representation group

<table>
<thead>
<tr>
<th>Measure</th>
<th>Non-representation group cut offs (n=147)</th>
<th>McNemar Test P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HADS anxiety 6 months</td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HADS anxiety baseline</td>
<td>&lt;8</td>
<td>0.238</td>
</tr>
<tr>
<td></td>
<td>≥8</td>
<td></td>
</tr>
<tr>
<td>HADS anxiety baseline</td>
<td>&lt;11</td>
<td>0.629</td>
</tr>
<tr>
<td></td>
<td>≥11</td>
<td></td>
</tr>
<tr>
<td>HADS depression 6 months</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>&lt;8</td>
<td>0.013</td>
</tr>
<tr>
<td>HADS depression baseline</td>
<td>&lt;11</td>
<td>0.791</td>
</tr>
<tr>
<td></td>
<td>≥11</td>
<td></td>
</tr>
<tr>
<td>Psychological distress</td>
<td>&lt;17</td>
<td>0.238</td>
</tr>
<tr>
<td>HADS psychological distress</td>
<td>≥17</td>
<td></td>
</tr>
</tbody>
</table>

7.7.3 Seattle Angina Questionnaire results

7.7.3.1 SAQ Angina Stability change scores

In the re-presentation group there was a non-significant improvement in the mean SAQ angina stability scores at 6 months with a mean change of 2.0 (using a paired-t test) (baseline mean 68.2 vs 6 month mean 70.3, p= 0.791), Table 7-32. In the non-representation group there was a very small non-significant improvement in the mean SAQ angina stability scores at 6 months with a mean change of 0.68 (using a paired-t test, 76.2 vs 76.9, p=0.805) (using a WMPSR test, p=0.815), Table 7-33.

The number of participants experiencing a relevant clinical 10 point change (no change or ≤ -10 or > +10) in their SAQ angina stability scores at 6 months was the same across the groups (p=0.105), Table 7-34.

7.7.3.2 SAQ Angina Frequency change scores

In the re-presentation group there was a non-significant worsening of the mean SAQ angina frequency scores at baseline and 6 months (mean change -4.41) (79.7 vs 75.4, p=0.213) using a paired-t test (using a WMPSR test p= 0.239), Table
7-32. In the non-representation group there was a significant improvement in the mean SAQ angina frequency scores at 6 months with a mean change of 4.01 (using a paired-t test, baseline mean 83.0 and 6 month mean 86.7, p=0.024) (using a WMPSR test, p=0.020), Table 7-33.

The number of participants experiencing a clinically relevant 10 point change (no change or ≤ -10 or ≥ +10) in their SAQ angina frequency scores at 6 months is the same across the groups (p=0.258), Table 7-34.

7.7.3.3 SAQ Physical Limitation change scores

In the re-presentation group there was a non-significant decrease in mean SAQ physical limitation scores at 6 months with a mean change of -2.13 (using a paired-t test, baseline mean 63.9 and 6 month mean 61.8, p=0.617), (using the WMPSR p=0.543), Table 7-32. In contrast, the non-representation group had a significant improvement in mean SAQ physical limitation scores (mean change 3.74) between baseline and 6 months (67.0 vs 70.7, p=0.045) by paired-t test, and WMPSR (p=0.022), Table 7-33.

At 6 months fewer individuals in the re-presentation compared to the non-representation group, had a clinically relevant 10 point change in their SAQ physical limitation scores (cut offs). Furthermore, fewer individuals in the non-representation group had worsening SAQ physical limitation (p=0.054), Table 7-34.

7.7.3.4 SAQ Treatment Satisfaction change scores

In the re-presentation group there was a non-significant worsening of mean SAQ treatment satisfaction scores at 6 months (mean change -1.65) (baseline mean 88.7 and 6 month mean 86.0), using a paired-t test (p=0.282) and WMPSR test (p=0.264), Table 7-32. In the non-representation group there was no change at baseline and 6 months for mean SAQ treatment satisfaction (using a paired-t test) (89.1 vs 89.2, p=0.871) (using a WMPSR test, p=0.655), Table 7-33.

There was no difference between the groups in the cut offs for SAQ treatment satisfaction; over half the patients in both groups had no change in their treatment satisfaction, Table 7-34.

7.7.3.5 SAQ Quality of Life change scores

In the re-presentation group there was a non-significant improvement in the mean SAQ quality of life scores at baseline and 6 months (mean change 5.9, 52.0 vs 56.8, p=0.309) using a paired-t test, Table 7-32. In the non-representation group
there was a significant improvement in mean SAQ quality of life scores with a mean change of 13.38 (using a paired-t test, baseline mean 55.8 and 6 month mean 69.7, p<0.001), Table 7-33. There was no difference at 6 months between the groups in terms of cut offs, Table 7-34.

Table 7-32 SAQ sub-scale change scores for the re-presentation group.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Time point</th>
<th>Change</th>
<th>Baseline vs 6 months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 Mean (SD)</td>
<td>6 Mean (SD)</td>
<td>Change mean (SD)</td>
</tr>
<tr>
<td>* Angina stability</td>
<td>68.2 (32.6)</td>
<td>70.3 (34.8)</td>
<td>2.0 (46.2)</td>
</tr>
<tr>
<td>† Angina frequency</td>
<td>79.7 (19.4)</td>
<td>75.4 (28.6)</td>
<td>-4.4 (21.4)</td>
</tr>
<tr>
<td>Ж Physical limitation</td>
<td>63.9 (28.6)</td>
<td>61.8 (25.8)</td>
<td>-2.1 (24.6)</td>
</tr>
<tr>
<td>‡ Treatment satisfaction</td>
<td>88.7 (14.0)</td>
<td>86.0 (17.3)</td>
<td>-1.7 (14.0)</td>
</tr>
<tr>
<td>◊ Quality of life</td>
<td>52.0 (29.4)</td>
<td>56.8 (24.5)</td>
<td>5.9 (26.5)</td>
</tr>
</tbody>
</table>

*Angina stability change scores distribution was normal for the re-presentation group.  
† Angina frequency change scores distribution was non-normal for the re-presentation group.  
Ж Physical limitation change scores distribution was non-normal for the re-presentation group.  
‡ Treatment satisfaction change scores distribution was non-normal for the re-presentation group.  
◊ Quality of life change scores distribution was normal for the re-presentation group.  
1 Paired-t test  
2 Wilcoxon matched-pairs signed-ranks test

Table 7-33 - SAQ sub-scale change scores for the non-representation group

<table>
<thead>
<tr>
<th>Variable</th>
<th>Time point</th>
<th>Change</th>
<th>Baseline vs 6 months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 Mean (SD)</td>
<td>6 Mean (SD)</td>
<td>Change mean (SD)</td>
</tr>
<tr>
<td>* Angina stability</td>
<td>76.2 (32.4)</td>
<td>76.9 (26.4)</td>
<td>0.7 (33.4)</td>
</tr>
<tr>
<td>† Angina frequency</td>
<td>83.0 (21.0)</td>
<td>86.7 (20.9)</td>
<td>4.0 (20.2)</td>
</tr>
<tr>
<td>Ж Physical limitation</td>
<td>67.0 (29.0)</td>
<td>70.7 (26.9)</td>
<td>3.7 (21.7)</td>
</tr>
<tr>
<td>‡ Treatment satisfaction</td>
<td>89.1 (13.6)</td>
<td>89.2 (15.6)</td>
<td>-0.1 (13.9)</td>
</tr>
<tr>
<td>◊ Quality of life</td>
<td>55.8 (26.3)</td>
<td>69.7 (26.3)</td>
<td>13.4 (25.1)</td>
</tr>
</tbody>
</table>

*Angina stability change scores distribution was non-normal in the non-representation group.  
† Angina frequency change scores distribution was non-normal for the non-representation group.  
Ж Physical limitation change scores distribution was non-normal.  
‡ Treatment satisfaction change scores distribution was non-normal.  
◊ Quality of life change scores distribution was normal for the non-representation group.  
1 Paired-t test  
2 Wilcoxon matched-pairs signed-ranks test
Table 7.34 SAQ change scores cut offs (≤-10, no change or ≥+10)

<table>
<thead>
<tr>
<th>Measure</th>
<th>Re-presentation change cut offs (%)</th>
<th>Non-representation cut offs (%)</th>
<th>***χ² (df)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>≤-10 *No change</td>
<td>≥+10 *No change</td>
<td>≤-10 *No change</td>
<td>≥+10 *No change</td>
</tr>
<tr>
<td>SAQ angina stability</td>
<td>11 (29.7)</td>
<td>11 (29.7)</td>
<td>15 (40.5)</td>
<td>37 (25.2)</td>
</tr>
<tr>
<td>SAQ angina frequency</td>
<td>12 (32.4)</td>
<td>15 (40.5)</td>
<td>10 (27.0)</td>
<td>30 (20.4)</td>
</tr>
<tr>
<td>SAQ physical limitation</td>
<td>12 (35.3)</td>
<td>11 (32.4)</td>
<td>11 (32.4)</td>
<td>24 (17.5)</td>
</tr>
<tr>
<td>SAQ treatment satisfaction</td>
<td>10 (27.0)</td>
<td>22 (59.5)</td>
<td>5 (13.5)</td>
<td>27 (18.4)</td>
</tr>
<tr>
<td>SAQ QoL</td>
<td>9 (24.3)</td>
<td>14 (37.8)</td>
<td>14 (37.8)</td>
<td>19 (12.9)</td>
</tr>
</tbody>
</table>

*No change indicates no clinical change (i.e. not ≥-10 or +10)
**Pearson's chi-square test for trend

7.7.4 Summary for aim 3

There were no significant differences in the mean HADS scores, from baseline to 6 months, for the re-presentation group. There was some weak evidence that depression increased slightly at 6 months but this was not significant (p=0.073). There were also some small non-significant increases in the SAQ sub-scales, angina stability and quality of life. Conversely, there was a slight decrease at 6 months for the SAQ physical limitation, angina frequency and treatment satisfaction scales, but none of the SAQ sub-scales changed significantly at 6 months for the re-presentation group.

In contrast, HADS anxiety, depression and psychological distress all significantly improved at 6 months for the non-representation group. Likewise, the SAQ sub-scales mean scores also significantly improved at 6 months for physical limitation, angina frequency and quality of life. There was no change in SAQ angina stability or treatment satisfaction at 6 months for the non-representation group.
7.8  **Aim 4: Comparing levels of change in psychological health and angina symptoms between the groups**

*Aim 4:* To compare the change in anxiety, depression, psychological distress and angina symptoms between the groups at 6 months.

### 7.8.1 Introduction

Analysis of covariance (ANCOVA) was used to test whether there were differences between the re-presentation group and the non-representation group in terms of mean scores on psychological health and angina symptom variables at 6 months, adjusted for baseline values of the variables. Table 5-32 demonstrates the results for ANCOVA including a number of tests: a test for group effect, Levene’s test and a test of equal slopes. The group effect is a test of the null hypothesis that the difference in means of 6 month scores adjusted for baseline scores has a population value of 0. This hypothesis should be rejected in order to demonstrate that a group effect is present. Levene’s test is a test of homogeneity of variance (the level of variance in the outcome variable between the groups). This hypothesis should not be rejected in order to demonstrate that a group effect is present, as it is an underlying assumption of ANCOVA. The test of equal slopes is a test of the hypothesis that the relationship between the 6 month score and the baseline score is the same in each group. This hypothesis should also not be rejected as it is another underlying assumption of ANCOVA. The 95% CI is for the difference in means of 6 month scores between the groups adjusted for baseline scores.

### 7.8.2 Hospital Anxiety and Depression Score ANCOVA results

#### 7.8.2.1 HADS Anxiety comparison of the change between the groups

ANCOVA indicated that there was no difference between the groups at 6 months for the change in HADS anxiety scores \((p=0.139)\), Table 7-35. There was no evidence that the assumptions of equality of variance and equal slopes were being violated.

#### 7.8.2.2 HADS Depression comparison of the change between the groups

No difference was demonstrated between the groups at 6 months for the change in HADS depression using ANCOVA \((p=0.096)\), Table 7-35. This finding should be
viewed with a little caution as there is a suggestion from Levene’s test (p=0.049) that there may be some difference in variance between the groups.

7.8.2.3 HADS Psychological distress comparison of the change between the groups

There were no significant differences between the groups for the change in HADS psychological distress scores at 6 months using ANCOVA (p=0.194), Table 7-35. There was no evidence that the assumptions of equality of variance and equal slopes were violated.

Table 7-35 ANCOVA HADS results for comparison of change between the groups.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F</td>
<td>df</td>
</tr>
<tr>
<td>Anxiety</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group effect</td>
<td>2.21</td>
<td>1, 180</td>
</tr>
<tr>
<td>Levene’s test</td>
<td>2.15</td>
<td>1, 182</td>
</tr>
<tr>
<td>Test of equal slopes</td>
<td>0.09</td>
<td>1, 180</td>
</tr>
<tr>
<td>Depression</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group effect</td>
<td>2.81</td>
<td>1, 180</td>
</tr>
<tr>
<td>Levene’s test</td>
<td>3.92</td>
<td>1, 182</td>
</tr>
<tr>
<td>Test of equal slopes</td>
<td>0.46</td>
<td>1, 180</td>
</tr>
<tr>
<td>Psychological distress</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group effect</td>
<td>1.70</td>
<td>1, 180</td>
</tr>
<tr>
<td>Levene’s test</td>
<td>2.45</td>
<td>1, 182</td>
</tr>
<tr>
<td>Test of equal slopes</td>
<td>0.20</td>
<td>1, 180</td>
</tr>
</tbody>
</table>

7.8.3 Seattle Angina Questionnaire (SAQ) ANCOVA results

7.8.3.1 SAQ Angina Stability comparison of the change between the groups

ANCOVA indicated that there was no evidence of a difference between the groups at 6 months for change in SAQ angina stability scores (p=0.325), Table 7-36. The result should be viewed with caution as Levene’s test is significant suggesting that there is variance between the groups (p<0.001).

7.8.3.2 SAQ Angina Frequency comparison of the change between the groups

ANCOVA indicated that there was a difference between the groups at 6 months for SAQ angina frequency scores (p<0.001), although the test of unequal slopes was violated suggesting that there may be a difference between the groups in the level of change from baseline to 6 months (p=0.006). In section 7.7.3.2 it was found that at 6 months the re-presentation group demonstrated a non-significant worsening of mean change for SAQ angina frequency, whereas a significant
improvement was found for the non-representation group (-4.41 [SD 21.4] and 4.01 [SD 20.2]), Table 7-32 and Table 7-33.

7.8.3.3 SAQ Physical Limitation comparison of the change between the groups
There was no significant difference between the groups at 6 months for change in SAQ physical limitation scores as indicated by ANCOVA (p=0.983), Table 7-36. There was no evidence that the assumptions of equality of variance and equal slopes were violated.

7.8.3.4 SAQ Treatment Satisfaction comparison of the change between the groups.
There was no significant difference between the groups at 6 months for SAQ treatment satisfaction scores using ANCOVA (p=0.689), Table 7-36. Again, there was no evidence that the assumptions of equality of variance and equal slopes were violated.

7.8.3.5 SAQ Quality of Life comparison of the change in between the groups.
ANCOVA indicated that there was no evidence of a difference between the groups at 6 months for SAQ quality of life scores (p=0.720), Table 7-36. Again, there was no evidence that the assumptions of equality of variance and equal slopes were being violated.

Table 7-36 ANCOVA SAQ results for comparison of change between the groups.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F</td>
<td>df</td>
</tr>
<tr>
<td>Angina stability</td>
<td>Group effect</td>
<td>0.97</td>
</tr>
<tr>
<td></td>
<td>Levene’s test</td>
<td>15.58</td>
</tr>
<tr>
<td></td>
<td>Test of equal slopes</td>
<td>2.29</td>
</tr>
<tr>
<td>Angina frequency</td>
<td>Group effect</td>
<td>11.17</td>
</tr>
<tr>
<td></td>
<td>Levene’s test</td>
<td>1.70</td>
</tr>
<tr>
<td></td>
<td>Test of equal slopes</td>
<td>7.63</td>
</tr>
<tr>
<td>Physical limitation</td>
<td>Group effect</td>
<td>0.00</td>
</tr>
<tr>
<td></td>
<td>Levene’s test</td>
<td>0.33</td>
</tr>
<tr>
<td></td>
<td>Test of equal slopes</td>
<td>0.71</td>
</tr>
<tr>
<td>Treatment satisfaction</td>
<td>Group effect</td>
<td>0.16</td>
</tr>
<tr>
<td></td>
<td>Levene’s test</td>
<td>0.95</td>
</tr>
<tr>
<td></td>
<td>Test of equal slopes</td>
<td>0.05</td>
</tr>
<tr>
<td>Quality of life</td>
<td>Group effect</td>
<td>0.13</td>
</tr>
<tr>
<td></td>
<td>Levene’s test</td>
<td>0.02</td>
</tr>
<tr>
<td></td>
<td>Test of equal slopes</td>
<td>1.09</td>
</tr>
</tbody>
</table>
7.8.4 Summary for aim 4

There was no significant difference between the groups at 6 months for the change in HADS anxiety, depression and psychological distress scores. Likewise there was no difference between the groups at 6 months for the change in SAQ angina stability, physical limitation, treatment satisfaction and quality of life scores. However, a change was seen between the groups for the SAQ angina frequency scores. Univariate analysis indicated a significant improvement of SAQ angina frequency for the non-representation group; in the re-presentation group there was a slight non-significant worsening of scores at 6 months.

7.9 Aim 5: Associations of psychological health and angina symptoms within the groups

Aim 5: To determine the association between the levels of anxiety, depression, psychological distress and angina symptoms for both groups at baseline and at 6 months.

7.9.1 Introduction

The distribution of HADS anxiety, depression, psychological distress and SAQ sub-scale mean scores at baseline and 6 months were all determined as a preliminary step for calculating the correlations among the variables. Details of HADS and SAQ sub-scale distributions can be found in Appendix L.

The majority of distributions for the mean HADS scores were non-normal at baseline and 6 months. Likewise, normality testing for SAQ sub-scales also indicated that the majority of SAQ mean scores had a non-normal distribution at baseline and 6 months. Correlations were therefore calculated using a non-parametric correlation Kendall’s tau_b (Field, 2009, page 181). The results of the Kendall’s tau_b test can be seen in Table 7-37 and Table 7-38 showing the baseline and 6 month (respectively) results for both re-presentation and non-representation groups.

As previously discussed in section 5.5.1, low HADS scores indicates good psychological health as recommended by Zigmond and Snaith (1983). The reverse is the case for the SAQ scores with low scores indicating poorer health status (Spertus et al., 1995). In the case of the correlations in this section, a negative correlation indicates higher scores for one variable and lower scores for the other variable, i.e. higher HADS scores and lower SAQ scores. The detailed scoring
mechanism for HADS and SAQ are described in full in section 5.10.1.3 and 5.10.2.3.

7.9.2 Baseline correlation

7.9.2.1 Re-presentation group

In the re-presentation group at baseline, HADS anxiety and depression scores were significantly positively correlated with each other (0.56, p < 0.001). Both were positively correlated with HADS psychological distress, as expected since this is the sum of the two scores. Furthermore, anxiety, depression and psychological distress scores were significantly, negatively correlated with a moderate effect, with angina frequency scores (-0.41, -0.35, -0.39), and treatment satisfaction scores (-0.42, -0.38, -0.29). Quality of life scores were negatively associated with anxiety and psychological distress scores with moderate effect (-0.41, -0.49), but had the strongest negative association with depression scores (-0.52), Table 7-37.

7.9.2.2 Non-representation group

In the non-representation group at baseline, HADS anxiety and depression scores were again significantly positively correlated (0.55, p < 0.001). Additionally, in the non-representation group anxiety and psychological distress scores were negatively association with treatment satisfaction (both -0.31) with moderate effect. Depression and psychological distress scores were also moderately negatively associated with physical limitation (-0.37, -0.31) and angina frequency (-0.34, -0.31). Anxiety, depression and psychological distress scores were negatively correlated with quality of life (-0.35, -0.34, -0.36) with a moderate effect, Table 7-37, Table 7-38.
Table 7-37 Correlation (Kendall’s tau_b) table at baseline for the presentation groups

<table>
<thead>
<tr>
<th></th>
<th>HADba</th>
<th>HADbd</th>
<th>HAD Psychological distress</th>
<th>SAQ Physical limitation</th>
<th>SAQ Angina stability</th>
<th>SAQ Angina frequency</th>
<th>SAQ Treatment satisfaction</th>
<th>SAQ QoL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-representation group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HADba</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HADbd</td>
<td>0.563*** P&lt;0.001 n=38</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HAD psychological distress</td>
<td>0.801*** P&lt;0.001 n=38</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAQ Physical limitation</td>
<td>-0.097 P=0.414 n=37</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAQ Angina stability</td>
<td>-0.171 P=0.180 n=38</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAQ Angina frequency</td>
<td>-0.406*** P&lt;0.001 n=38</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAQ Treatment satisfaction</td>
<td>-0.418*** P&lt;0.001 n=38</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAQ QoL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

** Correlation is significant at the level 0.01 (2-tailed).
*** Correlation is significant at the level 0.001 (2-tailed).
* Correlation is significant at the level 0.05 (2-tailed).
7.9.3 6 months correlation

7.9.3.1 Re-presentation group

In the re-presentation group at 6 months, HADS anxiety and depression scores were again significantly positively correlated (0.63, p < 0.001). Additionally for the re-presentation group anxiety scores were significantly negatively associated with angina frequency (-0.32) and treatment satisfaction (-0.35), both with moderate effect. Depression and psychological distress scores were significantly, negatively associated at moderate effect with physical limitation (-0.36, -0.29) and angina stability (-0.36, -0.30), angina frequency (-0.42, -0.39) and treatment satisfaction (-0.42, -0.40). Anxiety, depression and psychological distress scores demonstrated the strongest negative correlation with quality of life (-0.49, -0.48, -0.52) at 6 months, Table 7-38.

7.9.3.2 Non-representation group

In the non-representation group at 6 months, HADS anxiety and depression scores were again significantly positively correlated (0.63, p < 0.001). Moreover, in the non-representation group, anxiety, depression and psychological distress scores all demonstrated a moderate to strong negative correlation with physical limitation (-0.42, -0.54, -0.49) and angina frequency. Likewise anxiety, depression and psychological distress scores showed a moderate correlation with angina stability (-0.36, -0.33, -0.36) and treatment satisfaction (-0.40, -0.35, -0.40). Quality of life demonstrated the strongest negative association with anxiety, depression and psychological distress scores (-0.52, -0.52, -0.54), Table 7-38.
### Table 7-38 Correlation (Kendall’s τb) table at 6 months for the presentation groups

<table>
<thead>
<tr>
<th></th>
<th>Non-representation group</th>
<th>Re-presentation group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HAD6a</td>
<td>HAD6d</td>
</tr>
<tr>
<td>HAD6a</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>HAD Psychological distress</td>
<td>SAQ Physical limitation</td>
</tr>
<tr>
<td></td>
<td>0.625*** P&lt;0.001 n=37</td>
<td>0.491*** P&lt;0.001 n=144</td>
</tr>
<tr>
<td></td>
<td></td>
<td>-0.359*** P&lt;0.001 n=147</td>
</tr>
<tr>
<td></td>
<td></td>
<td>-0.454*** P&lt;0.001 n=147</td>
</tr>
<tr>
<td></td>
<td>HAD Psychological distress</td>
<td>SAQ Angular stability</td>
</tr>
<tr>
<td></td>
<td>0.903*** P&lt;0.001 n=37</td>
<td>0.515*** P&lt;0.001 n=147</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>SAQ Angina frequency</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.493*** P&lt;0.001 n=147</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>SAQ Treatment satisfaction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.606*** P&lt;0.001 n=147</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>SAQ QoL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.493*** P&lt;0.001 n=147</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>SAQ Physical limitation</td>
</tr>
<tr>
<td></td>
<td>-0.302* P=0.003 n=37</td>
<td>-0.393*** P&lt;0.001 n=147</td>
</tr>
<tr>
<td></td>
<td>-0.393*** P&lt;0.001 n=37</td>
<td></td>
</tr>
<tr>
<td></td>
<td>-0.347** P&lt;0.001 n=37</td>
<td></td>
</tr>
<tr>
<td></td>
<td>-0.477*** P&lt;0.001 n=37</td>
<td></td>
</tr>
<tr>
<td></td>
<td>-0.523*** P&lt;0.001 n=37</td>
<td></td>
</tr>
<tr>
<td></td>
<td>-0.472*** P&lt;0.001 n=34</td>
<td></td>
</tr>
<tr>
<td></td>
<td>-0.399*** P&lt;0.001 n=37</td>
<td></td>
</tr>
<tr>
<td></td>
<td>-0.504*** P&lt;0.001 n=37</td>
<td></td>
</tr>
<tr>
<td></td>
<td>-0.431*** P&lt;0.001 n=37</td>
<td></td>
</tr>
</tbody>
</table>

* Correlation is significant at the level 0.05 (2-tailed).
** Correlation is significant at the level 0.01 (2-tailed).
*** Correlation is significant at the level 0.001 (2-tailed).
7.9.4 Summary for aim 5

At baseline and at 6 months, HADS anxiety and depression scores were significantly and positively correlated with each other in both groups.

At baseline and 6 months the psychological variables demonstrated significant weak to moderate correlations with angina symptoms, and these associations occurred in both groups.

At baseline for both groups all the psychological variables had weak associations with angina stability, and moderate association with angina frequency. Overall the psychological variables were also associated with quality of life (QoL) with moderate effect for both groups, with the exception of depression in the re-presentation group which had a stronger association.

The results at 6 months identified that the psychological variables were moderately associated with angina frequency and treatment satisfaction for the re-presentation group. Quality of life was also moderately to strongly correlated with all of the psychological variables. Additionally, in the re-presentation group depression was associated with physical limitation and angina stability with moderate effect.

At 6 months, in the non-representation group, moderate to strong associations were demonstrated between the psychological variables across all the SAQ subscales. Anxiety, depression and psychological distress were all strongly correlated with QoL in the non-representation group.

Overall in both groups correlations were stronger at 6 months than at baseline for anxiety, depression and psychological across the SAQ subscales.
7.10 Aim 6: Association of psychological health, angina symptoms physiological health and re-presentation

Aim 6: To determine the association of psychological health (anxiety, depression and psychological distress), angina symptoms (stability, frequency and physical limitation) and physiological health (severity of STEMI and comorbidity) with re-presentation due to potential IHD symptoms, adjusted for other expected contributing or confounding factors previously identified in the conceptual model.

As noted in section 5.23.1, it was not possible to include all contributing and confounding variables in regression models to test the conceptual model. It was decided to include the following set of predictor variables as independent variables in statistical models, all measured at baseline: the SAQ angina frequency score and angina stability score to incorporate symptoms; the HADS anxiety score and HADS depression score (or their total, the HADS psychological distress score) as measures of psychological health; the GRACE score covering the severity of STEMI (based on a composite of key physical parameters) and the CCI score covering comorbidity as measures of physiological health. Justification for inclusion was given in Table 5-7.

7.10.1 Multicollinearity

An assessment of multicollinearity was conducted prior to running the logistic regression models (Table 7-40) and in terms of Norusis’s (2006) rules (i.e. Variance Inflation Factor >10) there was no evidence of collinearity between HADS anxiety and HADS depression at baseline, as their tolerances were not unusual enough to suggest a problem.

Table 7-39 Variance Inflation Factor (VIF) for independent variables.

<table>
<thead>
<tr>
<th>Variable name</th>
<th>Co-linearity statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Variance Inflation Factor</td>
</tr>
<tr>
<td>GRACE</td>
<td>1.205</td>
</tr>
<tr>
<td>CCI</td>
<td>1.136</td>
</tr>
<tr>
<td>HADS anxiety</td>
<td>2.576</td>
</tr>
<tr>
<td>HADS depression</td>
<td>2.520</td>
</tr>
<tr>
<td>SAQ angina stability</td>
<td>1.296</td>
</tr>
<tr>
<td>SAQ angina frequency</td>
<td>1.517</td>
</tr>
</tbody>
</table>

However, although there was no clear evidence of multicollinearity as all VIF results were <10, anxiety and depression were the highest of the VIF results and their tolerance scores were the lowest of all the scores (Table 7-39). It was possible that
these two variables could be closely associated and may affect the stability of logistic regression models. Kendall’s tau_b between them was 0.63 in both the presentation groups (Table 7-38).

Further investigation showed that according to Field (2009, page 242) there was a suggestion of collinearity between HADS anxiety and HADS depression (see section 5.23.1.4). Collinearity diagnostics in SPSS showed that the smallest eigenvalue had a condition index of 17.6, which was greater than the rule-of-thumb of 15.0, indicating potential collinearity (Table 7-40). However, only angina frequency was associated with this component. HADS anxiety and HADS depression shared variance on the fifth component, which had a satisfactory condition index of 8.5. There was no clear evidence of multicollinearity but there were suggestions of a relationship between HADS anxiety and HADS depression.

Table 7-40 Collinearity results

<table>
<thead>
<tr>
<th>Dim</th>
<th>Eigenvalue</th>
<th>Condition index</th>
<th>Variance Proportions (constant)</th>
<th>GRACE</th>
<th>CCI</th>
<th>HADS Anx</th>
<th>HADS Dep</th>
<th>Angina Stab</th>
<th>Angina Freq</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5.444</td>
<td>1.0</td>
<td>0.00</td>
<td>0.00</td>
<td>0.01</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>2</td>
<td>0.697</td>
<td>2.795</td>
<td>0.00</td>
<td>0.00</td>
<td>0.83</td>
<td>0.00</td>
<td>0.00</td>
<td>0.01</td>
<td>0.00</td>
</tr>
<tr>
<td>3</td>
<td>0.592</td>
<td>3.033</td>
<td>0.00</td>
<td>0.01</td>
<td>0.02</td>
<td>0.06</td>
<td>0.10</td>
<td>0.03</td>
<td>0.01</td>
</tr>
<tr>
<td>4</td>
<td>0.115</td>
<td>6.893</td>
<td>0.01</td>
<td>0.21</td>
<td>0.08</td>
<td>0.16</td>
<td>0.08</td>
<td>0.50</td>
<td>0.01</td>
</tr>
<tr>
<td>5</td>
<td>0.075</td>
<td>8.547</td>
<td>0.01</td>
<td>0.02</td>
<td>0.00</td>
<td>0.60</td>
<td>0.78</td>
<td>0.32</td>
<td>0.01</td>
</tr>
<tr>
<td>6</td>
<td>0.060</td>
<td>9.525</td>
<td>0.03</td>
<td>0.59</td>
<td>0.06</td>
<td>0.04</td>
<td>0.02</td>
<td>0.13</td>
<td>0.30</td>
</tr>
<tr>
<td>7</td>
<td>0.018</td>
<td>17.615</td>
<td>0.95</td>
<td>0.17</td>
<td>0.00</td>
<td>0.14</td>
<td>0.02</td>
<td>0.01</td>
<td>0.67</td>
</tr>
</tbody>
</table>

Dependent variable: potential IHD re-presentation

7.10.2 Univariable Logistic Regression Models

Table 7-41 shows that HADS anxiety, depression and psychological distress were all significantly associated with re-presentation (p=0.008, p=0.007 and p<0.001) when unadjusted for other variables. Of the predictors HADS psychological distress was the most significant, had the narrowest confidence interval for the odds ratio (OR) and the highest amount of generalised variation explained (Nagelkerke R² = 0.140). The predictors SAQ angina stability, SAQ angina frequency, GRACE and CCI were not significantly associated with re-presentation (p= 0.273, p=0.331, p=0.824 and p=0.533).
Table 7-41 Univariable logistic regression models estimating each predictor with representation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model $\chi^2$</th>
<th>df</th>
<th>P value</th>
<th>Nagelkerke R Square</th>
<th>OR</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HADS anxiety</td>
<td>7.13</td>
<td>1</td>
<td>0.008</td>
<td>0.056</td>
<td>1.09</td>
<td>0.7-1.175</td>
<td>0.008</td>
</tr>
<tr>
<td>HADS depression</td>
<td>7.18</td>
<td>1</td>
<td>0.007</td>
<td>0.056</td>
<td>1.10</td>
<td>0.8-1.194</td>
<td>0.007</td>
</tr>
<tr>
<td>HADS psychological distress</td>
<td>17.05</td>
<td>1</td>
<td>&lt;0.001</td>
<td>0.140</td>
<td>1.08</td>
<td>1.04-1.123</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SAQ angina stability</td>
<td>1.17</td>
<td>1</td>
<td>0.279</td>
<td>0.009</td>
<td>0.98</td>
<td>0.98-1.004</td>
<td>0.273</td>
</tr>
<tr>
<td>SAQ angina frequency</td>
<td>0.92</td>
<td>1</td>
<td>0.338</td>
<td>0.007</td>
<td>0.98</td>
<td>0.97-1.008</td>
<td>0.331</td>
</tr>
<tr>
<td>GRACE 6 month death or MI</td>
<td>0.05</td>
<td>1</td>
<td>0.825</td>
<td>&lt;0.001</td>
<td>1.00</td>
<td>0.96-1.042</td>
<td>0.824</td>
</tr>
<tr>
<td>CCI</td>
<td>0.37</td>
<td>1</td>
<td>0.542</td>
<td>0.003</td>
<td>1.09</td>
<td>0.81-1.471</td>
<td>0.533</td>
</tr>
</tbody>
</table>

From past experience, there were concerns about having HADS anxiety and HADS depression in the same model due to the correlation between the two scores, as expected and as found in sections 5.9.2 and 5.9.3. Table 7-42 shows the inclusion of predictors HADS anxiety and HADS depression together in the same model without the other predictors.

Table 7-42 Logistic regression model estimating association between HADS anxiety and HADS depression with re-presentation.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model $\chi^2$</th>
<th>df</th>
<th>P value</th>
<th>Nagelkerke R Square</th>
<th>OR</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HADS anxiety</td>
<td>12.080</td>
<td>8</td>
<td>0.148</td>
<td>0.064</td>
<td>1.053</td>
<td>0.948-1.170</td>
<td>0.333</td>
</tr>
<tr>
<td>HADS depression</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.061</td>
<td>0.945-1.191</td>
<td>0.319</td>
</tr>
</tbody>
</table>

The inclusion of HADS anxiety and HADS depression led to a non-significant model (p=0.148), demonstrating that they were no longer associated with re-presentation (anxiety: adjusted OR=1.053, p=0.333, depression: adjusted OR=1.061, p=0.319), even though they were significantly associated with re-representation in separate models (anxiety: OR=1.097, p=0.008; depression: OR=1.108, p=0.007), see Table 7-41. This was due to the result of fixing one of the two variables to examine the residual association of the other with the outcome. When the HADS depression score was considered to be fixed, the OR measuring the association between the HADS anxiety score and re-representation fell from 1.097 to 1.053 and became non-significant. When the HADS anxiety score was considered to be fixed, the OR measuring the association between the HADS depression score and re-representation fell from 1.108 to 1.061 and also became non-significant. The overall model was also non-significant. The level of association between the HADS...
anxiety score and the HADS depression score appeared to be strong enough to prevent them being included in the same model based on the current sample.

### 7.10.3 Multiple logistic regression models

#### 7.10.3.1 Model 1

Six predictors HADS anxiety, HADS depression, SAQ angina stability, SAQ angina frequency, GRACE and CCI were chosen for inclusion in model 1 (Table 7-43); HADS psychological distress was not included due its perfect linearity with HADS anxiety and HADS depression (being the sum of the anxiety and depression scores).

Table 7-43 Multiple Logistic Regression Model estimating association between HADS anxiety, HADS depression, SAQ angina stability, angina frequency, GRACE and CCI with re-presentation.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model ( \chi^2 )</th>
<th>df</th>
<th>p-value</th>
<th>Nagelkerke R Square</th>
<th>OR</th>
<th>95% CI</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HADS anxiety</td>
<td>10.023</td>
<td>6</td>
<td>0.124</td>
<td>0.079</td>
<td>1.084</td>
<td>0.968-1.214</td>
<td>0.161</td>
</tr>
<tr>
<td>HADS depression</td>
<td></td>
<td>1.054</td>
<td>0.934-1.188</td>
<td>0.393</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAQ angina stability</td>
<td>0.995</td>
<td></td>
<td></td>
<td></td>
<td>0.983-1.007</td>
<td>0.399</td>
<td></td>
</tr>
<tr>
<td>SAQ angina frequency</td>
<td>1.009</td>
<td></td>
<td></td>
<td></td>
<td>0.988-1.031</td>
<td>0.394</td>
<td></td>
</tr>
<tr>
<td>GRACE</td>
<td>1.022</td>
<td></td>
<td></td>
<td></td>
<td>0.981-1.064</td>
<td>0.304</td>
<td></td>
</tr>
<tr>
<td>CCI</td>
<td>1.022</td>
<td></td>
<td></td>
<td></td>
<td>0.746-1.400</td>
<td>0.890</td>
<td></td>
</tr>
</tbody>
</table>

The theoretical justification for inclusion of the variables in model 1 can be seen in section 5.23.1.3 and the theoretical underpinning of these variables is discussed in detail in the conceptual model (Chapter 3).

Table 7-43 demonstrates that when anxiety and depression were included in the model together, the model performance was poor. The Nagelkerke R squared (0.079) was small, indicating that only 8.0% of the generalised variation in the outcome variable (re-presentation) was being explained (Nagelkerke, 1991). Furthermore, results showed that the model was not significant (model \( \chi^2=10.02, \text{df}=6, p=0.124 \)) and the odds ratio for anxiety (adjusted OR=1.084, \( p=0.161 \)) and depression (adjusted OR=1.054, \( p=0.393 \)) were also not significant. Overall the model suggested that based on the variables included, none of the variables were a significant predictor associated with re-presentation. This agreed with the findings for models with HADS anxiety and depression scores in the same model, which contradicted findings for models with either HADS anxiety or HADS depression included separately (section 7.10.2).
7.10.3.2 Model 2

In a further model (model 2, Table 7-44), the predictors HADS anxiety and HADS depression were replaced by HADS psychological distress as the main predictor. In univariable logistic regression modelling HADS psychological distress showed the most significant fit; this model is empirically driven, unlike Models 1 and 3.

Table 7-44 Multiple Logistic Regression Model estimating association between HADS psychological distress, SAQ angina stability, SAQ angina frequency, GRACE and CCI with re-presentation.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model χ²</th>
<th>df</th>
<th>p-value</th>
<th>Nagelkerke R Square</th>
<th>OR</th>
<th>95% CI</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HADS psychological distress</td>
<td>9.954</td>
<td>5</td>
<td>0.077</td>
<td>0.078</td>
<td>1.069</td>
<td>1.020-1.121</td>
<td>0.005</td>
</tr>
<tr>
<td>SAQ angina frequency</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.995</td>
<td>0.983-1.007</td>
<td>0.420</td>
</tr>
<tr>
<td>GRACE</td>
<td>1.021</td>
<td></td>
<td></td>
<td></td>
<td>1.009</td>
<td>0.981-1.030</td>
<td>0.316</td>
</tr>
<tr>
<td>CCI</td>
<td>1.023</td>
<td></td>
<td></td>
<td></td>
<td>0.748</td>
<td>1.400</td>
<td>0.885</td>
</tr>
</tbody>
</table>

In model 2, Table 7-44, again the Nagelkerke R squared (0.078) indicated that only 7% of the generalised variation in re-presentation was being explained (Nagelkerke, 1991). This model just failed to be statistically significant (model χ²=9.95, df= 5, p=0.077), so there was insufficient evidence to reject the null hypothesis that all regression coefficients (adjusted log-odds) were 0 (all adjusted odds ratios were 1). However, the adjusted odds ratio for HADS psychological distress was significant (adjusted OR=1.069, p=0.005). This was close to the unadjusted odds ratio (OR=1.081, p<0.001) in Table 7-41, suggesting that the other predictors were having little impact on the association between HADS psychological distress and re-presentation.

7.10.3.3 Model 3

The final model (model 3) was based on the theoretical justification for the inclusion of predictor HADS anxiety. Theoretically, anxiety is the more important variable than depression in relation to participant re-presentation (see section 3.5.2). The evidence discussed in the conceptual model demonstrates that anxiety has been reported as an important factor during A&E attendances for chest pain and ACS events (Grace et al., 2004). The results for model 3 are presented in Table 7-45.
Table 7-45 Multiple Logistic Regression Model estimating association between HADS anxiety, SAQ angina stability, SAQ angina frequency, GRACE and CCI with re-presentation.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model $\chi^2$</th>
<th>df</th>
<th>p-value</th>
<th>Nagelkerke R Square</th>
<th>OR</th>
<th>95% CI</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HADS anxiety</td>
<td>9.300</td>
<td>5</td>
<td>0.098</td>
<td>0.073</td>
<td>1.120</td>
<td>1.030-1.219</td>
<td>0.008</td>
</tr>
<tr>
<td>SAQ angina stability</td>
<td>0.994</td>
<td></td>
<td></td>
<td>0.982-1.006</td>
<td>0.988</td>
<td>1.006-1.030</td>
<td>0.306</td>
</tr>
<tr>
<td>SAQ angina frequency</td>
<td>1.008</td>
<td></td>
<td></td>
<td>0.988-1.030</td>
<td>1.022</td>
<td>0.981-1.065</td>
<td>0.435</td>
</tr>
<tr>
<td>GRACE</td>
<td>1.022</td>
<td></td>
<td></td>
<td>0.981-1.065</td>
<td>1.022</td>
<td>0.744-1.405</td>
<td>0.290</td>
</tr>
<tr>
<td>CCI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The Nagelkerke R squared (0.073) indicated that the model explained only 7.0% of the generalised variation in re-presentation (Nagelkerke, 1991). Results also showed that the model as a whole just failed to be statistically significant (model $\chi^2=9.30$, df= 5, p=0.098), like model 2. Again, the adjusted odds ratio for HADS anxiety (adjusted OR=1.120) and its significance (p=0.008) were similar to the unadjusted odds ratio and p-value in Table 5-41 (OR=1.097, p=0.008). This again suggested that the other predictors were having little impact on the association between HADS anxiety and re-presentation.

### 7.10.4 Summary for aim 6

Of the predictor variables considered, HADS anxiety, HADS depression and their sum HADS psychological distress were significantly associated with re-presentation. The other predictors, SAQ angina stability and SAQ angina frequency, GRACE and CCI were not significantly associated with re-presentation.

When HADS anxiety and HADS depression were both included together in the same theoretically-driven logistic regression model (with or without the predictors SAQ angina stability and angina frequency, GRACE and CCI), neither variable was a significant predictor of re-presentation.

In logistic regression modelling, HADS psychological distress was the most statistically significant predictor of re-presentation. An empirically-driven multivariate logistic regression model including HADS psychological distress demonstrated a significant association with re-presentation with adjusted OR=1.069 (p=0.005). However, the overall model fit was marginally non-significant (p=0.077), which may have been due to the small size of the group of re-presenters (38) in the models. This suggested that taken as a whole, the other variables in the model were hiding the significance of HADS psychological distress but having little impact on the association between HADS psychological distress and re-presentation.
HADS anxiety was the second most significant predictor of re-presentation. A final multivariate logistic regression model included HADS anxiety as the main predictor on theoretical grounds (see section 5.23.1). The adjusted odds ratio of 1.12 for HADS anxiety was statistically significant (p=0.008) but the overall fit was again non-significant (p=0.098). As with modelling for HADS psychological distress, other variables in the model appeared to hide the significance of HADS anxiety and had little impact on its association with re-presentation.

### 7.11 Results summary

A total of 202 individuals returned baseline questionnaires and of these individuals 18.8% (95% CI 14.0% to 24.8%) re-presented to acute healthcare services with potential IHD events during the 6 month study follow-up. This group was known as the re-presentation group (n=38), the remaining individuals were referred to as the non-representation group (n=164).

Seventy four re-presentation events occurred during 6 month follow-up for the re-presentation group. The highest proportion (39.2%) of re-presentation events did not have a clearly defined discharge diagnosis in the patients’ healthcare records and were categorised as ‘no diagnosis’ for the purposes of this study. Additionally the discharge diagnosis for almost a quarter (24.3%) of events was cardiac ischaemia or another cardiac cause.

In the re-presentation group at baseline and 6 months there were mild levels of anxiety (mean=9.5 and 9.4) and depression (mean=7.5 and 8.4). Conversely, in the non-representation group at baseline and 6 months anxiety (mean=7.1 and 6.0) and depression (mean=5.3 and 4.4) scores were in the normal range. Comparison between the groups at baseline and 6 months for anxiety, depression and psychological distress, showed that all were significantly higher in the re-presentation group.

Comparison from baseline to 6 months for the re-presentation group demonstrated that there was no change in anxiety, depression or psychological distress scores. In contrast, the change in HADS anxiety, depression and psychological distress all significantly improved at 6 months for the non-representation group. However, at 6 months there was no significant difference between the groups for the change in HADS anxiety, depression and psychological distress scores.

There was overall low burden of angina symptoms for both groups at baseline and 6 months; angina stability was high and angina frequency was low at both time
points. However, at 6 months the re-presentation group did experience angina more often than the non-representation group (p<0.001). Quality of life at baseline was moderately reduced across the groups, whereas at 6 months it improved slightly for the re-presentation group. A larger improvement was seen in the non-representation group leading to only a slight reduction in quality of life for the non-representation group at 6 months (re-presentation 56.8 vs non-representation group 69.7, p=0.003). At 6 months there were no significant changes in the SAQ sub-scales for the re-presentation group. Conversely, in the non-representation group physical limitation, angina frequency and quality of life significantly improved at 6 months.

For the majority of the SAQ sub-scale scores there was no difference between the change (from baseline to 6 months) between the groups. However, for the SAQ angina frequency scores there was a difference between the groups at 6 months (p<0.001). In the re-presentation group, at 6 months there was a non-significant worsening of mean change for SAQ angina frequency, whereas in the non-representation group a significant improvement was indicated (mean change -4.41, SD=21.4 and mean change 4.01, SD=20.2).

At baseline and at 6 months, HADS anxiety and HADS depression were significantly positively correlated within the re-presenting and non-representing groups. The correlation results at baseline for anxiety and psychological distress showed significant, negative associations with angina frequency and treatment satisfaction of medium or strong effect for both the presentation groups. In the re-presentation group at baseline, depression had a strong negative correlation with quality of life, whereas in the non-representation group the correlation was of medium effect. At 6 months for both the re-presentation and non-representation groups anxiety, depression and psychological distress all demonstrated stronger negative correlations (either medium or strong) on all SAQ sub-scales compared to baseline. The strongest association for both groups was with SAQ quality of life.

Multivariate regression modelling was conducted with the inclusion of predictors HADS anxiety, HADS depression, SAQ angina stability, SAQ angina frequency, GRACE and CCI in a theoretically-driven model. Initial multicollinearity measurements did not indicate a problem between HADS anxiety and HADS depression. However, when the variables were included in the logistic regression model together their association with re-presentation disappeared as the variables appeared to cancel each other out.
Univariable logistic regression modelling indicated that HADS psychological distress was associated with re-presentation and that it was the most stable of the models. A multivariate regression model including HADS psychological distress (in place of HADS anxiety and HADS depression) demonstrated that psychological distress significantly predicted re-presentation, with an adjusted odds ratio of 1.069 (p=0.005), which was little changed from the unadjusted odds ratio of 1.081 (p<0.001). There was no evidence that the other predicting variables were affecting the association between HADS psychological distress and re-presentation. However, the overall model itself just failed to be significant (p=0.077), which may be due to the small number of re-presenterers available.

Theoretically anxiety is the more important of the variables in relation to re-presentation. A final multivariate logistic regression model included HADS anxiety as the main predictor. Anxiety was predictive of re-presentation with an adjusted odds ratio of 1.120 and significance of p=0.008, again little changed from the unadjusted odds ratio of 1.097 (p=0.008). There was no evidence that the other predicting variables were affecting the association between HADS anxiety and re-presentation. Again, the overall model itself failed to be significant (p=0.098).
CHAPTER 8 QUALITATIVE FINDINGS

8.1 Introduction

This chapter of the thesis contains the qualitative study findings. Details of the participant selection and the reasons for the inclusion of participants are described. The demographic and re-presentation characteristics of those interviewed are also illustrated. Furthermore, the final thematic framework and narrative are presented.

8.2 Participants

The participants in this study were purposefully selected using maximum variation sampling. The a priori characteristics for selecting participants included those who had re-presented to acute services due to potential IHD symptoms within 6 months following treatment PPCI for STEMI. The aim was to reflect the diversity of the study population, including a mixture of men and women. Participants were chosen for their ability to share ‘rich’ data at interview and as data collection continued participants were selected to achieve maximum variation of the group. Participants were identified during the telephone calls conducted to collect re-presentation data at 6 months. All participants were interviewed on one occasion in the context of their own home. Full details of the qualitative methods can be found in chapter 6.

8.2.1 Participant details

Twenty-eight participants were initially selected and of those 25 agreed to be interviewed. Of those who did not participate, one refused because it was not his ‘cup of tea’, one acted as carer to his wife and did not have sufficient availability to be interviewed and a further individual became acutely unwell with cancer and felt unable to participate.

The 25 participants who agreed to participate consisted of 9 women and 14 men, from 27 to 79 years (Table 8-1). The proportion of women to men interviewed reflected the gender split of the re-presentation group as a whole (36.0% women interviewed and 34.2% women in the re-presentation group). Details of all 25 interviewee demographics, re-presentation details and the researchers’ reasons for choosing to interview the individuals can be seen in Table 8-1.
Table 8-1 – Demographics, representation data and the reasons for inclusion in the interviews

<table>
<thead>
<tr>
<th>Interview number</th>
<th>ÆID</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Ethnicity</th>
<th>Employment</th>
<th>Re-presentation</th>
<th>Re-presentation discharge diagnosis</th>
<th>Reason for choice of interviewee</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5</td>
<td>64</td>
<td>f</td>
<td>²Cau</td>
<td>Retired</td>
<td>0</td>
<td>Nil</td>
<td>See section ***</td>
</tr>
<tr>
<td>2</td>
<td>21</td>
<td>52</td>
<td>m</td>
<td>²Cau</td>
<td>Lorry driver</td>
<td>1</td>
<td>Clinic acute admission</td>
<td>Cardiac- stent re-stenosis</td>
</tr>
<tr>
<td>3</td>
<td>13</td>
<td>64</td>
<td>f</td>
<td>²Cau</td>
<td>Retired</td>
<td>4</td>
<td>999</td>
<td>Depression</td>
</tr>
<tr>
<td>4</td>
<td>48</td>
<td>31</td>
<td>f</td>
<td>²Cau</td>
<td>Housewife /mother</td>
<td>1</td>
<td>999</td>
<td>Indeterminate</td>
</tr>
<tr>
<td>5</td>
<td>30</td>
<td>63</td>
<td>M</td>
<td>²Cau</td>
<td>Solicitor</td>
<td>3</td>
<td>999</td>
<td>Failed thrombolysis</td>
</tr>
</tbody>
</table>

²Indicates second language
<table>
<thead>
<tr>
<th>Interview number</th>
<th>ID (yrs)</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Ethnicity</th>
<th>Employment</th>
<th>Re-presentation</th>
<th>Re-presentation discharge diagnosis</th>
<th>Reason for choice of interviewee</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>18</td>
<td>53</td>
<td>M</td>
<td>2Cau</td>
<td>Lorry driver</td>
<td>1</td>
<td>GP &amp; 999</td>
<td>No diagnosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Retired man due to ill health- had his heavy goods vehicle (HGV) licence revoked. GP sent participant to A&amp;E when he experienced symptoms, but participant didn’t believe that symptoms were related to his heart. He thought that they were a chest infection.</td>
</tr>
<tr>
<td>7</td>
<td>34</td>
<td>79</td>
<td>M</td>
<td>2Cau</td>
<td>Retired</td>
<td>1</td>
<td>999</td>
<td>Cardiac- ACS</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Elderly retired man who re-presented whilst visiting his niece. Initially he thought symptoms were related to his heart, but subsequently believed they weren’t, but his niece ‘panicked’ and called 999.</td>
</tr>
<tr>
<td>8</td>
<td>120</td>
<td>47</td>
<td>F</td>
<td>2Cau</td>
<td>Works as carer in care of the elderly home</td>
<td>2</td>
<td>GP &amp; 999</td>
<td>? Pulmonary embolism anxiety/or depression</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>A woman on long term sick leave since her STEMI due to ongoing fatigue and chest pain symptoms. She also suffered from the comorbid condition asthma.</td>
</tr>
<tr>
<td>9</td>
<td>108</td>
<td>61</td>
<td>M</td>
<td>2Cau</td>
<td>Engineering/ manual labour + full-time carer for disabled wife.</td>
<td>2</td>
<td>Friend drove to A&amp;E Cardiac- angina Cardiac- ACS</td>
<td>Man employed in semi-manual labour role, who had the additional responsibilities of caring for disabled wife. Experienced ongoing symptoms that he found difficult to differentiate and used multiple medications to treat symptoms.</td>
</tr>
<tr>
<td>10</td>
<td>124</td>
<td>67</td>
<td>M</td>
<td>2Cau</td>
<td>Retired due to ill health (ex-lorry driver)</td>
<td>1</td>
<td>GP &amp; 999</td>
<td>Cardiac- heart failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Retired man due to ill health (had his HGV licence revoked) Suffered a previous heart attack (received PPCI) and recognised the symptoms when he had the latest STEMI.</td>
</tr>
<tr>
<td>11</td>
<td>149</td>
<td>43</td>
<td>M</td>
<td>2Cau</td>
<td>War photographe r</td>
<td>1</td>
<td>Wife drove to A&amp;E No diagnosis</td>
<td>A man with a young family. Participant had made major adjustments to lifestyle due to STEMI. Had also suffered from</td>
</tr>
<tr>
<td>Interview number</td>
<td>ID (yrs)</td>
<td>Age</td>
<td>Sex</td>
<td>Ethnicity</td>
<td>Employment</td>
<td>Re-presentation</td>
<td>Re-presentation discharge diagnosis</td>
<td>Reason for choice of interviewee</td>
</tr>
<tr>
<td>------------------</td>
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<td>----------------</td>
<td>----------------------------------</td>
<td>----------------------------------</td>
</tr>
<tr>
<td>12</td>
<td>192</td>
<td>48</td>
<td>F</td>
<td>2 Cau</td>
<td>Cook in care of the elderly home</td>
<td>1 Partner drove to A&amp;E</td>
<td>Cardiac- ACS</td>
<td>A woman on long term sick leave since her STEMI, who experienced financial and relationship problems that she directly related to her heart attack.</td>
</tr>
<tr>
<td>13</td>
<td>116</td>
<td>67</td>
<td>M</td>
<td>2 Cau</td>
<td>Builder (self-employed)</td>
<td>1 Friend drove to A&amp;E</td>
<td>Indeterminate</td>
<td>Man who ran his own building firm. He returned to a very physical job quickly after his STEMI, but wasn't sure how far he could safely physically push himself.</td>
</tr>
<tr>
<td>14</td>
<td>184</td>
<td>49</td>
<td>M</td>
<td>2 Cau</td>
<td>Floor fitter (self-employed)</td>
<td>1 999 No diagnosis</td>
<td>Indeterminate</td>
<td>Self employed man doing manual labour, on sick leave since his STEMI and was experiencing severe financial difficulties. He had also been diagnosed with depression since his STEMI.</td>
</tr>
<tr>
<td>15</td>
<td>173</td>
<td>57</td>
<td>M</td>
<td>2 Cau</td>
<td>Incapacity benefit</td>
<td>1 Taxi to A&amp;E</td>
<td>Cardiac- angina</td>
<td>Man receiving incapacity benefit, suffering from diabetes, had previously had a heart attack and had been treated with CABG. Participant was also very isolated as he lived alone and had few friends.</td>
</tr>
<tr>
<td>16</td>
<td>89</td>
<td>55</td>
<td>F</td>
<td>2 Cau</td>
<td>Clerical, worker</td>
<td>2 Friend drove to ward 999 Cardiac- angina</td>
<td>Indeterminate 999</td>
<td>Woman who returned to work after 4 months after STEMI. First re-presentation her friend panicked and took her to hospital. On second occasion participant had severe chest pain and called an ambulance- she thought it was another heart attack.</td>
</tr>
<tr>
<td>17</td>
<td>175</td>
<td>76</td>
<td>M</td>
<td>2 Cau</td>
<td>Retired lecturer</td>
<td>3 Wife drove to A&amp;E</td>
<td>Indeterminate 999 Cardiac- ACS Non-cardiac</td>
<td>Retired educated man. Multiple post STEMI admissions with IHD symptoms. Diagnosed with NSTEMI after index STEMI and was treated with CABG, also diagnosed with bowel cancer and</td>
</tr>
<tr>
<td>Interview number</td>
<td>ID</td>
<td>Age (yrs)</td>
<td>Sex</td>
<td>Ethnicity</td>
<td>Employment</td>
<td>Re-presentation</td>
<td>Re-presentation discharge diagnosis</td>
<td>Reason for choice of interviewee</td>
</tr>
<tr>
<td>------------------</td>
<td>----</td>
<td>-----------</td>
<td>-----</td>
<td>-----------</td>
<td>-------------</td>
<td>----------------</td>
<td>------------------------------------</td>
<td>---------------------------------</td>
</tr>
<tr>
<td>18</td>
<td>320</td>
<td>51</td>
<td>M</td>
<td>2Cau</td>
<td>Incapacity benefit</td>
<td>2 Daughter drove to A&amp;E</td>
<td>Non-cardiac</td>
<td>Man receiving incapacity benefit. Suffered from severe anxiety, particularly in relation to HCP and hospitals. Experienced a non cardiac admission and then a further STEMI and was again treated with PPCI.</td>
</tr>
<tr>
<td>19</td>
<td>273</td>
<td>76</td>
<td>F</td>
<td>2Cau</td>
<td>Retired machinist</td>
<td>2 999</td>
<td>No diagnosis</td>
<td>Elderly woman with a number of comorbid conditions (asthma, epilepsy, previous breast cancer), but still tried to remain active. Prior to her heart attack she was still working part-time. She experienced similar symptoms to her heart attack, but was unsure of the cause.</td>
</tr>
<tr>
<td>20</td>
<td>359</td>
<td>54</td>
<td>M</td>
<td>2Cau</td>
<td>Gardener (with City Council)</td>
<td>3 999</td>
<td>Cardiac- angina</td>
<td>Man on sick leave from a job involving manual labour. Lived with his wife and daughter but reported feeling very isolated, also expressed anxiety regarding symptoms and the need to return to work.</td>
</tr>
<tr>
<td>21</td>
<td>362</td>
<td>63</td>
<td>M</td>
<td>2Cau</td>
<td>Retired due to ill health</td>
<td>1 GP then walked to A&amp;E</td>
<td>Cardiac- angina</td>
<td>Retired man due to ill health. Had suffered 2 previous heart attacks (treated with PPCI and CABG). He still found it difficult to differentiate between symptoms.</td>
</tr>
<tr>
<td>22</td>
<td>419</td>
<td>27</td>
<td>M</td>
<td>Asian</td>
<td>Tailor</td>
<td>2 GP &amp; 999</td>
<td>No diagnosis</td>
<td>Young man working 6 days a week as a tailor was identified as being of Asian origin. In addition he reported having very little social support.</td>
</tr>
<tr>
<td>Interview number</td>
<td>ID (yrs)</td>
<td>Sex</td>
<td>Ethnicity</td>
<td>Employment</td>
<td>Re-presentation</td>
<td>Re-presentation discharge diagnosis</td>
<td>Reason for choice of interviewee</td>
<td></td>
</tr>
<tr>
<td>------------------</td>
<td>---------</td>
<td>-----</td>
<td>-----------</td>
<td>------------</td>
<td>----------------</td>
<td>--------------------------------------</td>
<td>----------------------------------</td>
<td></td>
</tr>
<tr>
<td>23</td>
<td>378</td>
<td>M</td>
<td>2Cau</td>
<td>Member of Clergy</td>
<td>0</td>
<td>Nil</td>
<td>See section 8.2.2</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>An educated working man. Previously experienced 2 heart attacks and also suffered panic attacks. The symptoms of the panic attacks resembled STEMI. Since the most recent STEMI he had suffered symptoms but decided that it was a panic attack and therefore did not re-present.</td>
<td></td>
</tr>
<tr>
<td>24</td>
<td>234</td>
<td>F</td>
<td>2Cau</td>
<td>Retired due to ill health</td>
<td>3</td>
<td>Partner drove to A&amp;E</td>
<td>Gastric, Indeterminate, Non-cardiac</td>
<td>A woman had previously had a STEMI and was heavily disabled due to comorbid conditions (heart failure and chronic obstructive airways disease). She also suffered gastric problems after the most recent STEMI.</td>
</tr>
<tr>
<td>25</td>
<td>258</td>
<td>F</td>
<td>2Cau</td>
<td>Retired due to ill health</td>
<td>2</td>
<td>GP &amp; 999</td>
<td>Cardiac-heart failure, 999, Indeterminate</td>
<td>Retired women who had experienced 2 STEMI's and received PPCI (on one occasion). Experienced symptoms related to her bowels following STEMI (similar to index STEMI) and thought she was having another heart attack. Had severe depression following most recent STEMI.</td>
</tr>
</tbody>
</table>

1 Study identification number  
2 Caucasian  
3 Number of re-presentations
8.2.2 Participants not meeting the inclusion criteria

Twenty-one participants met the qualitative study inclusion criteria i.e. they had re-presented to acute services with potential IHD symptoms. Four interviewees did not meet the *a priori* interview inclusion criteria. The inclusion of these cases was discussed with the supervisory team and the decision was made to include them in the study. The reasons for their inclusion are discussed below.

The first individual to be interviewed (study no. 5) had not re-presented acutely but had received planned PCI six weeks following PPCI. Initially this participant was chosen for interview due to the inexperience of the researcher in gaining the correct information relating to re-presentation during the telephone interview. Likewise interviewee 2 (study no. 21) also did not re-present to acute services. This individual experienced severe potential IHD symptoms and was admitted as an emergency from the consultants’ clinic for urgent assessment. Despite not attending A&E due to his symptoms this patient sought help for his symptoms via another route that resulted in emergency admission and it was therefore decided to include this individual.

Interviewee 5 (study no. 30) received emergency ‘rescue PCI’ following failed coronary reperfusion after thrombolytic therapy and not PPCI. This participant had been wrongly classified in the quantitative study dataset as having received PPCI as his STEMI treatment. The error in classification did not become apparent until the individual participated in the qualitative interview. This participant re-presented on two occasions due to potential IHD symptoms during the 6 month follow-up. During the interview, the participant shared extremely rich, relevant, data and it was therefore decided to include this participants’ interview data with the benefit of diversifying the study sample.

A fourth individual, (interview 23, study no. 378) was purposefully selected, because he had experienced symptoms that he self-diagnosed as panic attack “*initially I thought it was a heart attack*” and chose not to seek help. This individual had a long history of heart disease including experiencing two previous heart attacks. He had also previously experienced panic attacks that had led to seeking help via acute services due to his believe that the symptoms were cardiac related. During the telephone interview this individual spoke of finding it very difficult to differentiate between a panic attack and a heart attack. Although this individual had not re-presented since his most recent STEMI, it was decided to interview him to explore his experiences of interpreting and differentiating between anxiety and IHD related symptoms.
8.3 Thematic framework

The processes undertaken to develop the thematic framework are described in section 6.3.2. Examples of the thematic framework set-up in NVivo8 used to develop the analysis can be seen in 0. Furthermore, an excerpt of the journal that was used to record the steps taken to refine concepts including the collapsing and merging of codes can be seen in Appendix P. An excerpt of a chart used as part of the analytical process can be seen in Appendix S, Photographs of the mapping and interpretation phase can also be seen in Appendix T.

During the initial stages of developing the thematic framework, early concepts included disbelief and shock at having had a heart attack and uncertainty regarding symptoms. Financial problems, adjusting to life after a heart attack, anxiety, depression, anger and isolation were also identified.

"I thought well.....you know I never had a pain or anything....because when you hear of people having a heart attack you think of them oooh you know", (interviewee 1 (f), p4, 20-22).

"the more I think about it....you know because everybody’s gone to bed...or everybody’s gone out because there goin’ out playin’...the kids or because (wife)’s gone out to visit next door neighbour or some other friends and I’m sat here and I’m thinking right well what’s this.....(coughs).....and it doesn’t work.........you know it just makes you think harder about the way I am” (interviewee 2 (m), p12, 34-39).

As further data collection and analysis progressed additional concepts included change of role, employment, slowing down, embarrassment, confusion and doubt. Other factors noted were, thoughts of death, cardiac rehabilitation, using or not using an ambulance, chest pain, trying to understand symptoms and self diagnosis.

"It’s taken me a long time this time (to recover after the heart attack), I don’t know, I really don’t know why it’s taken longer this time” (interviewee 25 (f), p10, 8-9).

Interviewee 14 (m), p3, 4-7, “Sunday night started feeling a bit dodgy, erm, woke up on the Monday morning to go to work, I thought it’s, you know, it’d be something or nothing, woke up on the Monday morning ready to go to work and I just felt lousy, I felt like I was having another heart attack”.

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### 8.4 Themes and sub-themes

The final thematic framework included four main themes, along with related sub-themes and can be seen in the tables of themes (Table 8-2).

Table 8-2 - The final four themes and related sub-themes

<table>
<thead>
<tr>
<th>Theme</th>
<th>Sub-Theme</th>
<th>Details of Sub-Themes</th>
</tr>
</thead>
</table>
| 1.0 Fear of experiencing a further heart attack | 1.1 Fear of death and dying                    | Fearful that they may die from a heart attack  
Thoughts of death at time of heart attack                                      |
|                                            | 1.2 Anxious may experience a further heart attack | Anxious that symptoms are related to heart.  
Distressed that may have another heart attack causing them to feel depressed.  
Worried that doing normal activities may bring on a heart attack. |
|                                            | 1.3 Panic that another heart attack was occurring | Participant fearful that symptoms were related to the heart or like heart attack symptoms and sought help, “I thought I was having another heart attack”.  
Doubt regarding cause of symptoms at time of re-presentation felt by participants and their relatives.  
Help seeking, participants either called emergency services or for the help of a relative. Some relatives “panicked” and either took individual to A&E or called an ambulance.  
Some participants sought help from their GP, who thought symptoms were serious and called an ambulance. |
| 2.0 Uncertainty and inability to determine cause of symptoms | 2.1 Anxiety and uncertainty in differentiating symptoms | Anxious that cannot differentiate symptoms; is it angina, indigestion, pulled muscle, anxiety or psychological?  
Uncertain if multiple causes of symptoms occur at the same time i.e. angina and indigestion.  
Worried that symptoms relate to the heart.  
Anxious may interpret symptoms incorrectly i.e. when it is a heart attack or another condition such as indigestion. |
|                                            | 2.2 Stereotyping heart attack symptoms         | Do not recognise STEMI symptoms as a heart attack.  
Mental picture of someone having a heart attack “it wasn’t me” “It wasn’t like you see on the telly”.  
Knew he/she was having a heart attack, recognised the pain, “Once you’ve had a heart attack you never forget the pain” |
<table>
<thead>
<tr>
<th>Theme</th>
<th>Sub-Theme</th>
<th>Details of Sub-Themes</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.3</td>
<td>Inability to control body or symptoms</td>
<td>Embarrassed as cannot control body. Has to live with symptoms. Unable to control symptoms. Denial- wants to believe that symptoms are indigestion/wind. Tiredness debilitating, had to slow down and cannot do what used to do. Didn’t know how should be after a heart attack, whether they should experience some symptoms. Uncertain how much can expect to be able to do after a heart attack.</td>
</tr>
<tr>
<td>2.5</td>
<td>Need for a diagnosis.</td>
<td>Not sure what caused symptoms during re-presentation Lack of or an indeterminate diagnosis at the time of re-presentation led to feelings of doubt and confusion. The need for a diagnosis was strong in participants to enable them to comprehend their symptoms. Self-diagnosis was used to learn how to interpret future symptoms, causes such as heart, lungs, indigestion or anxiety were mentioned by participants.</td>
</tr>
<tr>
<td>2.6</td>
<td>Self-treatment</td>
<td>Taking medications to alleviate symptoms. Using multiple treatments (i.e. taking something for both indigestion and angina). Alternatives to medication used. May rest prior to taking GTN when gets pain.</td>
</tr>
<tr>
<td>3.0</td>
<td>Insufficient opportunity to validate self-construction of illness</td>
<td>3.1 Absence of illness ceremony Disbelief at speed of treatment. Fast, calm treatment, “alright, it was easy” Early discharge, with minimal delivery of information or debriefing during in-patient stay. Severity of illness and consequences negated due to speed of treatment and lack of follow-up. Felt well “fantastic” immediately afterwards; “like I’d been for a tooth out”. “could have gone home that afternoon” (post PPCI). Feeling grateful and fortunate to have received PPCI. Lack of explanation or poor memory of information given during acute event.</td>
</tr>
</tbody>
</table>
### Theme: Delivery of information

#### Sub-Theme: Details of Sub-Themes

- Availability of written information, participants generally accessed this themselves from ward dayrooms and through cardiac rehab.
- Insufficient verbal information, dissatisfaction in relation to lack of information.
- Lack of follow-up through clinics.
- Presence of conflicting information, leading to confusion.

### Theme: Attendance at cardiac rehabilitation

#### Sub-Theme: Details of Sub-Themes

- “Excellent” attended it all
- “Gave me confidence”
- Dissatisfied with what it offers. Not physical enough. All old people attending. Wasn’t offered it.

### Theme: Self-construction of illness

#### Sub-Theme: Details of Sub-Themes

- Constructed beliefs and own conclusion regarding cause of symptoms.
- Assimilated knowledge from different sources to understand their heart attack and treatment.
- Using past experiences (theirs and others) and comparing past and current symptoms to enable self diagnosis of symptoms.

### Theme: Difficulty adapting to life after a heart attack

#### Sub-Theme: Details of Sub-Themes

- Disbelief at having had a heart attack: Shocked that it could happen to them. Doesn’t think that they are the type of person to have a heart attack, thought they were fit.
- Grieving for loss of self and past life: Denial, Anger, Guilt, Isolation
- Feeling low or depressed relating to difficulty adapting: Feeling depressed due to the effects the heart attack has had on everyday life. Due to loss of work and anxiety associated with symptoms.
- Enforced change of identity: Loss of role, unable to do what used to do. Unable to work, financial troubles for workers. Men questioning their masculinity. Unable to be the breadwinner. Women unable to be carers, to do usual household chores. Change in self image, Inability to get back to normal
- Confidence: Lost confidence in own abilities and to do everyday activities because may lead to a heart attack. Returning to work helps build confidence. Sense of normality and previous self. Cardiac rehab helped to gain confidence. Over time became more confident at recognising symptoms. Reassured by medic, unlikely to have
<table>
<thead>
<tr>
<th>Theme</th>
<th>Sub-Theme</th>
<th>Details of Sub-Themes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>another heart attack.</td>
<td>Feels confident could recognise another heart attack.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Receiving information and reflecting on past experiences help build confidence.</td>
</tr>
<tr>
<td>4.7</td>
<td>Life consequences of a heart attack</td>
<td>Feeling unable to control life.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Having to slow down, difficult to adjust.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Making changes to life, diet, stopping smoking</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Side-effects of medications.</td>
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<tr>
<td></td>
<td></td>
<td>Life changes distressing</td>
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<td></td>
<td></td>
<td>Affected whole family.</td>
</tr>
</tbody>
</table>

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8.5 Narrative

The final study themes and sub-themes are described through the words of the interviewees in the form of a narrative in this section.

8.5.1 Theme 1: Fear of experiencing a further heart attack

The main study theme encompassed the fears of the participants regarding experiencing a further heart attack. At the time of re-presentation the participants believed that they were either having another heart attack or that their symptoms were related to their heart. This was the central reason for presenting to acute services.

However, despite participants expressing the belief that symptoms were another cardiac event, they also spoke of doubt regarding the cause of symptoms.

Many participants were frightened that they may die from a heart attack and thoughts of death at the time or shortly after the heart attack were relatively common. This developed into anxiety that they may have another heart attack.

There were numerous modes of help seeking used by participants. Some spoke of either themselves or their relatives “panicking”. The majority called emergency services, but others gained the help of a relative. Some individuals attended their GP who thought symptoms were serious and called an ambulance.

1.1 Fear of death and dying

Some of the participants indicated that they were afraid of dying from a heart attack or had thoughts of death at the time of their heart attack.

“*I’m only 53 in April you know……still too young yet (laughs)…..got a lot more to do….a lot more yet, and if I suddenly go (die) I’m comin’ back, I don’t give a damn, I don’t care, I ain’t goin’ anywhere else*, (interviewee 2 (m), p7, 32-39).

“(crying) I’m like this all the time and … I had (HCP) on the phone today, up at the hospital… and erm… she said I need congenital (cognitive) thinking therapy or something, because of the way I think about things, because… (crying), (whispers) I’m scared of dying…(crying)”, (interviewee 4 (f), p1, 26-32).

1.2 Anxious may experience a further heart attack
Feelings of anxiety were reported by the group regarding the possibility of experiencing a further heart attack. Only a minority of individuals felt certain that they had not had a heart attack when they re-presented. The majority of participants appeared to be worried that symptoms were related to their heart and these thoughts and feelings led to participants feeling anxious.

"felt like I was having another heart attack", (interviewee 14 (m), p3, 16).

"the pain was like in my heart, you know. And I had a bit of pain on the shoulder there (points to left shoulder)". "I thought it was another heart attack, I did", (interviewee 15 (m), p10, 36-37: p11, 9-13).

"I have thoughts, which is, what's brought on anxiety about it as well as part it's part of that vicious circle with pain and...and things is...erm, and you're right, when, you know...my...my erm...my anxiety has been about having another heart attack, and...dying from it, that's what my anxiety has been about", (Interviewee 11 (m), p32, 39-45).

An elderly retired man who re-presented whilst on holiday at his niece’s house did not believe that he had suffered another heart attack. He stated that his niece panicked and called the ambulance when he experienced symptoms.

"well they couldn’t find much wrong with me, and the symptoms wore off...fairly quickly” “I mean they said I hadn’t had another heart attack, I’m sure I hadn’t”, (interviewee 7 (m), p 2, 1-6 & 25-28)

1.3 Panic that another heart attack was occurring

There was a sense of uncertainty regarding cause of symptoms at time of re-presentation for some participants and their relatives; this led to panic and subsequent help seeking. In some cases the individual called emergency services or enlisted the help of a relative. Other individuals sought the help of their GP, who called an ambulance when it was required.

"I felt a bit breathless and... I do know I over reacted, because since having my heart attack I’ve never had to use my spray thing or anything and never really had any problems, apart from feeling... tired” “I just ... I over reacted, I felt all breathless and... then I think I got myself worked up more than anything” (interviewee 4 (f), p1, 12-17).
"I had... these pains came on me during the night.... and I persevered with it and kept rubbing and rubbing, 'til it got to the next morning and I couldn't stand the pain anymore. I phoned my partner, he said 'right, phone the ambulance” (interviewee 24 (f), p3, 17-29).

In some cases it was relatives or friends who initiated help seeking and took the participants to A&E.

"When I woke up I got these similar feelings in my arm (to the heart attack)....so panic set in for my niece and her husband and my wife and er...they sent for an ambulance” (interviewee 7 (m), p1, 41-46; p2, 25-28).

8.5.2 Theme 2: Uncertainty and inability to determine cause of symptoms

Anxiety was expressed with regard to differentiating between symptoms and participants found this particularly challenging. Participants were also uncertain whether it was normal to experience symptoms following a heart attack and if it was possible to have concurrent causes of symptoms. Stereotypical symptoms were referred to as represented by those in the media. Individuals experienced difficulty in recognising symptoms when they deviated from those matching their expectations. Individuals’ spoke of self medicating to alleviate symptoms and using multiple treatments (i.e. taking something for both indigestion and angina).

Embarrassment at being unable to control the body was implicit in some of the interviews. Fatigue and debilitation were problematic and some individuals denied the impact that the heart attack had forced on their body.

The need for a definitive diagnosis related to the re-presentation event was strong and many individuals were uncertain of what caused their symptoms. The lack of a diagnosis or an ‘indeterminate’ diagnosis led to enhanced feelings of uncertainty and confusion. Participants often used self-diagnosis to enable them to interpret symptoms.

2.1 Anxiety and uncertainty in differentiating symptoms

Amongst the interviewees there was a sense of anxiety regarding differentiating between the causes of symptoms i.e. whether symptoms were angina, indigestion, pulled muscle, anxiety or psychological. There was also a lack of understanding whether multiple causes of symptoms may have occurred at the same time i.e. angina
and indigestion. Some participants were anxious that they may interpret symptoms incorrectly, for instance when symptoms indicate a heart attack or an alternative cause.

"I get a dull pain in me chest and I suffer terribly with, erm, heartburn..., which I was told, erm, can either be reflux acid or angina and it’s difficult to distinguish which it is”, (interviewee 14 (m), p9, 1-39).

“for the last few months I don’t know if it’s me tablets ....but after I’ve taken them after about half an hour...I sort of start having these hot ...going into like hot sweats....and I think ooh....you know....cause that how I was that morning....apart from the dizziness...and at first...I thought ooooh...it’s not starting again is it (the heart attack)?”, (interviewee 1 (f), p7, 38-45).

In a number of cases participant attributed their symptoms to psychological causes.

"I think psych...part of its (chest pain) psychological definitely...but you wind, get yourself in a vicious circle like oh what is this and, what am I feeling? and erm...and...my sort of mind going round and round in circle” (interviewee 11 (m), p1, 21-27).

**2.2 Stereotyping heart attack symptoms**

The symptoms of a heart attack were not recognised by some of the participants at the time of their heart attack. This was despite some participants having previously experienced multiple heart attacks. However, one individual who had previously had 2 heart attacks was very confident in recognising his most recent heart attack.

"Just a bit of tightness really and...maybe a little bit of breathlessness, but nothing else....no, nothing like you see on the television, people clutching their chests or anything, I’ve not had it with any of them” (interviewee 25 (f), p8, 4-10).

“everybody says....oh you get clamps as though somebody’s put their arm round you and squeezed you...you know what I mean......I didn’t have any of that.......all I felt was as though somebody had opened a freezer door and shoved the freezer right against me chest....that’s basically what it was” (interviewee 2 (m), p2, 1-7).

This man confidently recognized the symptoms of his heart attack.
“I was just taking...fella (dog) walk and I got to the end...and once you’ve had a heart attack, you never forget that pain...you know, you do know what’s coming...and I got to end and came straight back” (interviewee 10 (m), p4, 12-14).

2.3 Inability to control body or symptoms

There was an acknowledgement from some individuals that they could not control their bodies. A number of participants expressed this with embarrassment. Other individuals appeared to deny what was occurring with their body. One participant (interviewee 10) was told by the cardiologist that only 25.0% of his heart was functioning and that he would have to live with the symptoms, yet the participant denied having any heart problems. Furthermore, tiredness and debilitation causing individuals to slow down was an issue reported by some individuals. Feelings of doubt and difficulty in establishing what normal levels of activity should be following a heart attack were also reported. Some individuals were unsure how they should feel after a heart attack. Furthermore, others were not sure whether experiencing symptoms after a heart attack was to be expected.

“um, my body threatens...... from time to time” (interviewee 23 (m), p8, 1-5).

"when you breathe in you feel it catching...and then, I try to burp a lot...because I keep telling myself it’s wind, I won’t tell you that it’s not (laugh) you know” (interviewee 2 (m), p19, 16-46)

This woman experienced chest pain, collapsed and was incontinent of urine during a severe episode of depression shortly after her index STEMI. She was extremely embarrassed at having been incontinent.

“they said that I passed out because I was so uptight...and erm ...and  wet myself (said quietly)” (interviewee 3 (f), p1, 13-21).

A number of individuals were uncertain regarding what was ‘normal’ and how they should feel following a heart attack and PPCI.

“so I don’t know whether...you’re meant to feel just the way you was before it...and if you are, I don’t” (interviewee 8 (f), p25, 15-20).

“the doctor said I would just be the same (after heart attack), so I was thinking ‘I shouldn’t be this tired’, you know, ‘cause I knew I couldn’t... I got up for work and I felt as tired as I went to bed and I’d just sit there and I’d be like an hour and I’d still
be sat there with a cup of tea thinking oh, ‘I’ve got to move!’” (interviewee 16 (f), p9, 2-5).

One man was unsure whether the discomfort in his chest was normal following PPCI and whether it was due to the stents.

“I’ve got this discomfort (in chest) and they, I wasn’t clear whether it was as a result of having the stents fitted or whether I was still... suffering from angina or whatever it may be” (interviewee 5 (m), p2, 17-28).

2.5 Need for a diagnosis

The need for a diagnosis following re-presentation was particularity strong in some participants to enable them to understand their symptoms. Some participants, who had not received or were given an indeterminate diagnosis at the time of re-presentation, were left feeling doubtful and confused. Some participants were told “fortunately your okay” (interviewee 20 (m), 21, 31-34) and others told “you haven’t got angina” (interviewee 19, (f), p3, 40-50). Other participants weren’t given a diagnosis, “they didn’t know what it was” (interviewee 24 (f), p2, 4-11). Yet they had all experienced symptoms and were left uncertain of the cause. Self-diagnosis was often used in a bid to learn how to interpret symptoms in the future. Causes such as heart, indigestion, lungs or “in my head” were mentioned by participants.

“Err I had some pain and the pains was underneath here (under left breast) and it was going to my chest and I thought of my heart straight away, but thank God, touch wood, it wasn’t my heart, they kept me in three days and they didn’t know what it was. But I think it was trapped wind” (interviewee 24 (f), p2, 4-8).

“I thought it was another heart attack, I did” “they put me onto the... is it the angiogram?” “they said they would... they’d treat it as a heart attack” (interviewee 15 (m), p11, 1-16).

This individual was left feeling hesitant when his belief that symptoms were due to indigestion, were brought into doubt due to the ECG reading.

“It could have been just indigestion or something I’d eaten or whatever, I don’t know...but I kind of like made it go anyway...within an hour or so, it had cleared up...but they said (A&E staff) there was something there (on the ECG) but they couldn’t say what it was and then it just...don’t know, faded away, whatever it was” (interviewee 6 (m), p 8, 2-7).
2.6 Self-treatment

Self-treatment of symptoms was common amongst interviewees. A number of individuals had developed strategies to address symptoms. Some spoke of taking GTN spray when they experienced shortage of breath or chest pain and others used multiple treatments. Alternatives to medication such as resting or rubbing the chest were also used to alleviate discomfort.

“I’ll have it (GTN) when I come down before I even have a brew or anything else to eat, so I have two or three sprays and then I’ll sit down and wait” “If pain doesn’t go, just get on with it, get on with the day” (participant 20 (m), p 10, 4-42).

“What I do the first thing I do is go and sit down...um....sort of try and relax myself....um...... and... and if I’ve not taken me medication, I then take me medication” (interviewee 21 (m), p 26, 26-49).

This man took medication for both angina and indigestion when he experienced chest pain.

“It (chest pain) will go when I take that (GTN spray), erm, and it’ll also go... because I take the Ranitadine as well” “Yeah, so I don’t know which it is” (participant 14 (m), page 9, 9 to 39).

8.5.3 Theme 3: Insufficient opportunity to validate self-construction of illness

All participants reported being impressed by the speed and efficiency of PPCI treatment and overall they expressed gratitude for the high standard of care they had received. However, the fast treatment and early discharge appeared to result in a lack of illness ceremony and appeared to lead to disbelief regarding what had happened.

A number of procedures, events and social rituals surround acute illness (Parsons, 1951, Charmaz, 1997). This may include visual clues (e.g. scar or the number of intravenous infusions) or the frequency of monitoring by healthcare professionals (e.g. blood pressure, cardiac monitoring) and the number of other medical procedures (e.g. echocardiography) or interventions (blood sampling). Other cues may be the length of time that the patient is confined to bed or the length of hospital admission. In this thesis these events and social rituals surrounding the illness (STEMI) and treatment (PPCI) are conceptualised as an illness ceremony.
Some of the participants’ experiences of the illness ceremony surrounding their heart attack appeared to be different to their expectations, which were aligned with the experiences of relatives or friends who had suffered a heart attack in the past. Generally, participants were surprised at how well they felt immediately after treatment, as they expected to feel extremely unwell. In some cases individuals reported believing that they were well enough to return immediately to their normal daily activities. Moreover there was a sense of disbelief at the speed of the treatment. Participants were also shocked at how soon they were discharged; although this did not appear to be related to the timing of discharge, as some patients remained hospitalised up to 5 days and others just 3 days.

Consequently some individuals were unable to believe that they had actually had a heart attack. This appeared to lessen the seriousness of the heart attack event and influenced some participants’ wishes to attend cardiac rehabilitation. Additionally, participants’ ability to process information appeared to be influenced by their disbelief that they had suffered a heart attack.

Participants were generally quite vague about the information that they had received. Overall participants were dissatisfied regarding the type and amount of information received and the amount of follow-up after the heart attack. In particular additional psychological care and practical help with dealing with stress was required. Participants spoke of a lack of opportunity to discuss what had occurred during their heart attack and the treatment. Recall was poor relating to whether the information received included symptoms attribution.

Cardiac rehabilitation was attended by some participants who found it helped to build their confidence and their return to normal. However, others expressed a need for sessions to be tailored to their needs rather than being fixed. Some of the younger men believed that the physical exercises were too easy as they were very fit prior to their heart attack.

3.1 Absence of illness ceremony

Many of the participants were ‘shocked’ at the speed of PPCI treatment and also at how well they felt immediately after treatment. Early discharge was also commented on by participants and some believed that it was too early; others felt that they could have gone home the same day as their heart attack. The speed of treatment and feeling well afterwards appeared to lessen the seriousness of the heart attack event.
Although individuals were very complimentary of the PPCI service and were grateful that they had received the treatment.

“alright, it was easy,...in and out” (interviewee 6 (m), p8, 19-21)

This individual felt well enough for immediate discharge following PPCI.

“I was a bit shaky but that would be all the drugs wouldn’t it? the morphine and the...you know...but other than that I felt fine... I felt as though I could have, got up, straight away come home...that’s how I felt...as though nothing had happened, like I’d been for a tooth out” (interviewee 8 (f), p24, 14-25).

A few participants believed that discharge was too early.

“the doctor came and said .... erm you’d probably be able to go home tomorrow ... so erm I thought it seems a bit quick that” (interviewee 1 (f), p5, 18-31).

The seriousness of the event (STEMI) appeared to be lost on some individuals.

“So it all happened that fast, you didn’t really have time to think”, “I felt fantastic when I got there (coronary care),... to tell you the truth I could have come home [laughter] and they wouldn’t let me out” (interviewee 16 (f), p8, 7-18).

Most of the participants reported feeling fortunate that they had received PPCI treatment.

"you can’t know er...can’t knock any service I’ve had with the National Health, the service I’ve had has been absolutely superb hasn’t it?” (interviewee 10 (m), p4, 18-20).

“I was really grateful that, you know, the fantastic efforts of everybody and the technology involved and the expertise involved saved my life” (interviewee 14 (m), p4, 1-4).

### 3.2 Delivery of information

Many of the participants in this study either received written information whilst on the ward or during CR. The majority of information received was in the form of the British Heart Foundation (BHF) booklets and many individuals spoke of obtaining these themselves from the ward dayroom. Verbal information was often received from healthcare professionals such as the participant’s GP or Consultant. Some participants
spoke of their frustration and confusion when conflicting information was delivered by different healthcare professionals. One participant received conflicting information from two different consultants whilst in hospital, regarding the type of treatment he was to receive. Some participants also spoke of gathering verbal information from friends, relatives and other lay people.

"oh I got some pamphlets that were hanging about the hospital, you know them little books (BHF booklets) and that, and I read it all myself, but...there was nobody actually came up and said anything, this is what you’ve got to do, you could this, you could do that, it was a bit...er, hit and miss sort of thing...so, I just read all the pamphlets and that was it" (interviewee 6 (m), p4, 26-30).

Receiving conflicting information from different sources in relation to the use of GTN was reported by this individual.

“they said (GP) I shouldn’t need to use it (GTN) that much I said well...yeah but I was also told by...doctors (at the hospital) that I needed to use it as I felt it (angina) coming on...that’s the idea of it, preventing like you know to, open the air I said”
(interviewee 9 (m), p11, 36-44).

This man gathered information from a lay person in an attempt to try and better understand his ongoing chest pain.

"but the (GP) receptionist’s mother had heart attack...and was left the same way...with the chest pain and that’s when, told her it was this muscle, exact, without me even saying what the, they said the physios had said, said same thing it was a detached muscle” (interviewee 9 (m), p17, 8-32)

This individual was dissatisfied that he had not received more information and that he had not been invited to attend the consultant’s clinic since discharge following his heart attack.

"it’s the aftercare that let it..., I thought yeah, yeah...I thought the aftercare was...”, “like I said I didn’t get much information at all did I?”, “never been back (for follow-up) at all since (heart attack)” (interviewee 10 (m), p21, 2-20).
3.2 Attendance at cardiac rehabilitation

A third of those interviewed did not attend CR. A number of participants reported that they had not received an invitation and others were not sure what it involved. One woman stated that it was too far for her to travel.

Overall there were mixed feelings regarding the benefits of CR from ‘excellent’ and ‘it gave me confidence’ to ‘it was too easy’, and of those who did attend many did not complete all the sessions. Some individuals expressed the view that CR did not entirely meet their needs. One participant completed the course and was happy that his physical fitness improved. However, he believed that the level of physical activity was ‘too easy’ for him. Another individual felt that the programme was mostly orientated around dealing with physical risk reduction and wanted more guidance on dealing with stress. One woman felt that it was for ‘old people’ and that there wasn’t enough time to talk through her problems. The majority of participants asked, had not received any information on symptoms at cardiac rehabilitation and expressed a wish to receive more.

“Oh I think it has yes (given him confidence) …because…you see, you have your monitor on and er…and you go on maybe the treadmill…or the bicycle machine, two bicycle, there’s two types of bicycle machine…and, you can see that you’re working quite hard, and your heart rate is staying very steady…and that is encouraging” (interviewee 7 (m), p7, 37-46).

Despite experiencing ongoing chest pain since his heart attack this man reported that the CR physical exercises were too basic.

“I found it…a bit slow and er…it’s stuff that I’ve done for years”, “without being big headed I found a lot of it too easy, because…erm…like I say it’s stuff…that I’d give kids to do…a lot of it”, “but like I said but then again there was a part where I didn’t find it easy…and it did catch up with me” (interviewee 9 (m), p34, 4-32)

This woman believed that CR did not offer sufficient psychological support to meet her needs.

“it was just all about…flaming charts, seeing how many things you could do the bike, well that wasn’t what I wanted to go for…it was more mentally for me…I mean I’m dreading going back to work…but yet I want to go…but I don’t know if I’ll be able to do it, because I’m so much slower” (interviewee 12 (f), p12, 24-29).
3.4 Self-construction of illness

In this study participants used past experiences and other people’s experiences to construct their beliefs and make sense of symptoms. In some instances interviewees had assimilated knowledge from different sources to comprehend their heart attack and treatment. When one individual (interviewee 14, m) experienced his heart attack he remembered the symptoms that his uncle had suffered when he died from a heart attack years earlier. Another individual (interviewee 5, m) had assimilated knowledge relating to dealing with his angina. When he had a further heart attack he thought, “here we go again!” and based his decision making on his previous experience.

The experiences of a family member were used by this individual when interpreting his own symptoms.

“I just got a real tightening of the chest and er...then I had this overwhelming urge to actually go to the toilet and then that just brought back memories of...”, “what my cousin...... had said because he was in the house when his dad died...”, “he, like, followed his dad into the toilet and he said, bloody stinks in here, you know, came out the toilet and his dad was dead on the bed” (interviewee 14 (m), p17, 1-17)

This man interpreted his re-presentation symptoms based on his previous heart attack (the index STEMI).

"I don’t know erm... I said to my partner *** I said you had better call an ambulance because I knew what to expect this time...the first time of course I didn’t, it came as a big surprise” (interviewee 5 (m), p1, 36-38).

This man used his past experiences from more than a decade earlier to try and self-diagnose his symptoms.

"the pain was, um, it was like in my heart, you know. And I had a bit of pain on the shoulder there (left shoulder), but I didn’t have any pain straight down the hand. when I had the heart attacks in 19, um, it was 89 or 99, whatever, um, I had severe pain all the way down the arm, the left arm and shoulder, and on my chest,.....um, but this was pain, this was pain like round the heart, you know” (interviewee 15 (m), p11, 1-16)
8.5.4 Theme 4: Difficulty adapting to life after a heart attack

The shock that the participants displayed in relation to having experienced a heart attack appeared to influence their adaptation. Aspects of the grieving process were displayed by some of the participants relating to the loss of their previous lives or their roles within society or their families.

4.1 Disbelief at having a heart attack

Many of the participants were shocked, and found it difficult to comprehend that they had suffered a heart attack. Some stated that they didn’t believe that a heart attack could happen to them or that they were the type of person to experience a heart attack.

“And then, this heart attack … I would never ever have said I was a person for heart attacks” (interviewee 19 (f), p5, 42-43).

“It’s summert (heart attack) beyond my control…hence, as I said, of all…the categories of people’s lifestyles, mine didn’t warrant a heart attack” (interviewee 13 (m), p12, 24-27).

“I mean I was dead surprised that I’d had a heart attack at my age…but I mean they, they said then, no…you’re not that young really there is, people have them, younger” (interviewee 12 (f), p11, 15-18).

4.2 Grieving for loss of self and past life

Some of the participants displayed components of the grieving process including denial, anger, guilt, isolation and feeling fortunate or grateful. The participants spoke of the loss of aspects of their working, family and social lives. Some of the participants spoke of developing depression since their heart attack and attributed this to the difficulties they endured adapting their lives.

Expressions of anger were apparent for some individuals in relation to having had a heart attack or due to the lifestyle changes and financial aspects of their lives. In particular two of the interviewees (2 and 14) were angry throughout the interview and expressed themselves with swearing and a raised tone of voice.

Guilt was expressed by some of the participants in relation to the feelings that they were experiencing. One participant (interviewee 20, m) felt guilty for using the ambulance service and so drove to A&E when he got further chest pain. Furthermore,
feelings of isolation from loved ones and from society were expressed by some participants. However, overall the majority of individuals felt fortunate to have received PPCI when they had their heart attack.

Denial

This woman lived near an ambulance station "I knew it was the ambulances going (when hearing the sirens) and I used to hate it, and yet they saved my life", “but it’s just that fear, I don’t know, it just brings back all the memories that you don’t really want to remember” (participant 25 (f), p4, 37-49)

"I mean to be honest with you...I’d never got over the fact that I’d had a heart attack anyway...I mean I keep turning round and saying that it wasn’t......I mean I keep telling me self that it’s not a heart attack it’s bronchitis” (interviewee 2 (m), p2, 1-7)

Anger

Anger was expressed by other individuals in relation to having had a heart attack.

"now and again, obviously I will be totally focussed on whatever I am doing and forget that I’ve had a bloody heart attack or any of the symptoms that follow it, but also ‘uughh’ and I just get reminded and then I’ll know then that I can’t go...I’ll have a sit down or walk away” (interviewee 20 (m), p9, 3-9).

"since I er... this has happened... obviously I’ve been out of work ..... governments all a bunch of ******* anyway because they don’t allow people to live .... every time they’d say oh yeah you can have the sick pay .... they take a load more money off you”, “you may as well go back to work ... you know.... I thought why should I, why should I go round and kill meself for some other ****head,.... sorry ... but er...” (interviewee 2 (m), p12, 34-44).

Guilt

Guilt was expressed by some of the participants in relation to the feelings that they were experiencing.

"I just don’t care, other times..... I think, well let it ‘the big one’ (another heart attack that will kill him), because as things are, there’s nothing (no point to life) I I I.... can feel guilty then because I think... how hard everybody works,... you know, to to
......make sure I got through this and how worried my family were” (interviewee 14 (m), p20, 12-20).

“You feel guilty about using the emergency services..... you always think there’s somebody worse than you, you know”, “I can be there in 10 minutes, probably quicker than what the ambulance takes to get here, if you understand what I mean?” (interviewee 21 (m), p20, 1-51).

Isolation

A number of participants demonstrated feeling isolated from their loved ones and from society.

“because everybody’s gone to bed...or everybody’s gone out, because there goin’ out playin, ...the kids, or (wife’s) gone out to visit next door neighbour, or some other friends, and I’m sat here and I’m thinking right well what’s this?” (interviewee 2 (m), p12, 34-44).

"I would be sat here on my own and if I wasn’t watching something...Oh I’m sick of watching that bloody thing anyway, because I’d rather be working, seriously I would” (interviewee 20 (m), p12, 47-49).

4.3 Depression due to difficulty adapting

Feelings of low mood were reported by many of those interviewed and on occasions participants used the term depression. The severity of symptoms varied across the group from mild feelings of low mood expressed as becoming upset more easily than usual, to severe depression requiring admission to hospital. In one extreme case a women who had a history of depression was admitted to a psychiatric hospital to receive treatment for severe depression; her admission lasted for 17 weeks.

Some individuals were receiving formal treatment including anti-depressants, counselling or psychotherapy and other individuals had refused treatment. Other individuals acknowledged feeling depressed but had not sought formal help. Some individuals related their low mood directly to their heart attack, whilst others believed it was related to loss of control. Several attributed their low mood to their financial difficulties due to their heart attack. One individual (interviewee 14) in particular, believed that the re-presentation event was the start of his problems.
"I had the heart attack... I was in hospital for only 3 days...then I come home...and 3
weeks after that, I just woke up one morning... and that was it... I was depressed... in
fact I was going mad wasn’t I (asks husband)? ... so he took me up to erm... A&E ...
and erm... they said that I passed out because I was so uptight” (interviewee 3 (f), p1,
13-26).

"then it works out that... the depression kicks in.... with the hassle that you get., and
you confidence in your own ability”, “and then when you stick alongside that all the
grief that you get and all the hassle that you get, you know, from finance companies
and Government departments that it just adds to it” (interviewee 14 (m), p41, 32-
50).

This man believed that his depression was due to the loss of control he had on his life
since his heart attack.

“you’re vulnerable yes, yeah” “er.... (sighs)....little bit of depression I think....but
because...mainly because I...I’m not in control...you know...it’s summert beyond my
control” (interviewee 13 (m), p12, 17-27).

4.4 Enforced change of identity

In this study some participants appeared to experience a ‘change of self image’ due to
the consequences of having had a heart attack. Many individuals adapted their roles
within the family and within society. A combination of factors seemed to affect some
of the interviewees’ ability to return to what they considered to be normal life.

Physical functioning and masculinity was affected for some of the younger men. In a
number of cases there was a need for them to know how far they could physically
push themselves before risking further symptoms or exacerbating their illness. When
their families offered to help them with chores that they normally undertook, help was
angrily rejected. Some of the women also reported finding it difficult to undertake
household chores and their role as carer in the home, although they were more willing
to accept assistance.

A number of participants were still on sick leave more than 6 months following their
heart attack. These individuals spoke of the financial difficulties and the stress that
this had caused them. In a number of cases individuals were extremely distressed and
angry about the treatment that they had received by the Department for Work and
Pensions (DWP).
"I think 'you must be more...you’re capable of doing something more strenuous than that’", “the biggest thing that I’ve done since, in the last few weeks is reconstructed the back fence at the back there. Some of it is quite heavy erm...but that didn’t seem to affect me it was...it was err...like the bending or coming up and I...I just got quite dizzy with it. So I just had to sit down erm...” (interviewee 20 (m), p6, 1-7)

"I can’t do what I used to do...you know.......I have to take my time more, but erm ...and like me husband he does a lot now.....erm, I don’t know, I can’t well I suppose because I get a bit breathless or I soon get tired so obviously erm instead of...doing everything at in a day.....its like over 2 or 3 days” (interviewee 1 (f), p10, 24-37).

This man had not been able to return to work more than 6 months after his heart attack due to his symptoms. This man expressed extreme anger during the interview regarding his loss of earnings and financial problems.

“But it’s very debilitating (symptoms) you know, I’ve not been able to go back to work and to top it all, two weeks ago, they just stopped the benefits. So I’ve got no money at all. So I’ve gone from, like, in February from somebody who used to go to the gym three times a week, to not being able to work, to taking tons of bloody pills a day to losing my business, erm... having credit card companies chasing me, to having my car repossessed, to having my benefits stopped completely” (interviewee 14 (m), p3, 27-51).

4.5 Confidence

A mixture of gaining confidence and lack of confidence was expressed across the participants in relation to returning to normal activities.

Gaining confidence

A number of factors helped participants to regain their confidence following their heart attack. One man found that his social activities helped him to regain his confidence and return to some sort of normality. In other cases attending cardiac rehabilitation helped participants to regain their confidence in undertaking everyday activities. There was also a level of acceptance with some participants that having another heart attack is out of their control. One woman felt confident that she did not need to worry too much because she had only had one episode of angina (interviewee 16 (f), p21, 5-9).

Learning from experiences and previous symptoms helped some of the participants to gain confidence in dealing with their symptoms. One woman had experienced chest
pains and re-presented twice when she had taken co-codomol for back pain. She had concluded that the pain was a reaction to the medication. The next time she experienced the pain she decided to wait until the next morning and contact the doctor rather than calling an ambulance (interviewee 19 (f), p7, 2-42).

"I don’t worry about it as much now (having another heart attack), if it happens it happens, you know” (interviewee 25 (f), p8, 35-51).

This man found that returning to social activities helped him to regain his confidence and return to normal.

“three or four weeks I was sort of going back to that (social activities)”, “because I can’t sit at home doing nothing all day... I like to feel I’m doing something useful... and I think if you’re doing something that you enjoy ...then it’s not so bad really” (interviewee 5, p 3, 16-30).

This woman partly regained her confidence through attending cardiac rehabilitation.

"Till I did that (cardiac rehab) you’re frightened of doing different things. ‘Don’t do this’, and when you go and do that it gives you confidence that you can do ... you know, like lifting something. I don't mean heavy things, but stretching up and my husband would be saying... don't do this, shouldn't be doing all that” (interviewee 19, p1, 1-10).

Lack of confidence

A lack of confidence was expressed in doing everyday activities and in their own abilities due to the heart attack was an issue for some of the participants. One woman was worried about going out alone with her dogs in the woods in case she had another heart attack (interviewee 19, p 5, 51-54). Other individuals did not feel confident enough to go on holiday or to travel abroad.

"and it (shakes) your confidence in your own ability” (interviewee 14 (m), p41, 39-40).

"it’s a big stumbling block.......it’s.... stupid little ideas that keep poppin’ in me head so often.....and you think I can’t do this, you can’t do that”, (interviewee 2 (f), p8, 36 to 45)
Several of the participants no longer felt confident to travel or go abroad, as they wished to be near healthcare facilities that they trusted.

"we booked one (holiday abroad) for this May, and I said, oh I don't want to go” “suppose I end up in bed for two or three days, or am in hospital? So he (husband) cancelled that. I said, don't book any more now” “I have to go to the doctor's to tell them. So he (doctor) said, well I think you're being wise really, you know, till you feel more confident. I'm just frightened, I'm frightened of anything going wrong” (participant 19 (f), p18, 1-10)

“I would like to… to travel to all sorts of places, but I... I think 'What kind of hospitals do they have there? How far am I going to be away from a hospital?’” (interviewee 23 (m), p15, 13-25).

4.6 Life consequences of a heart attack

The affects of the heart attack were far reaching for some individuals, including taking regular medication to deciding to no longer travel long distances. In some cases the side-effects of medication impinged on the participants' lives and individuals reported feeling unable to control bodily symptoms. Other individuals found it difficult to adjust to the changes that their bodies were enforcing on them and were physically unable to continue with their usual activities.

Many of the interviewees had made changes to their lifestyles including altering their diet and stopping smoking. Several of the participants had made an active decision to no longer travel or go abroad, as they wished to be near healthcare facilities that they trusted. Younger individuals with family commitments found that the effects of the heart attack and in some cases symptoms, had implications for the whole family.

This individual had experienced problems with medication side-effects.

"I was very, very tired at first for ages and ages, months, and I kept thinking this can’t be right, I shouldn’t be this tired and I went back to the doctors and he said ‘oh well you’re on the lowest beta blockers so, can’t do that’. Anyway, he changed my cholesterol tablets. I was on the highest cholesterol tablets and he changed them to Stativin I think it is, and I’ve been great ever since” (interviewee 16 (f), p 2, 2-8).

This woman was no longer able to walk the same distance she could prior to her heart attack.
“I used to love walking and then I find out I can’t do it anymore, the walking. It’s, um, I can, I can walk, don’t get me wrong, I’m not saying I can... I can’t walk, I can walk. But it’s, um, it... I have to take my time” (interviewee 15 (m), p 19, 6-10)

Some participants spoke of stopping smoking and others reported stopping and starting again.

"I’ve stopped that (smoking), straight away...yeah I used to smoke, that was my biggest risk factor, yeah...haven’t touched a cigarette, haven’t had one puff since ...the heart attack, so yeah, I’m pleased with that“ (interviewee 11, p 10, 18-27).

This woman no longer went abroad; as she felt safer being near healthcare facilities that she knew and trusted.

"I mean it takes a while to get your confidence back, you know, to sort of...things like go on holiday and things like that because you want to be where you’re safe“  “I mean we don’t...we’re not people for going abroad anyway, we don’t like the sun but I would never, ever, go out of the country now” (interviewee 25 (f), p8, 35-51).

8.6 Summary

Twenty-five participants, 9 women and 14 men of various ages (27 to 79 years) were interviewed in their homes. The group varied in their final re-presentation discharge diagnosis and in other respects, such as whether they experienced comorbidities, were suffering from anxiety or depression or were of employment age.

Early familiarisation and the interview schedule were used to determine the thematic framework, which was used during the indexing of the interviews. The thematic framework comprised of ten themes including, construction of illness event, re-presentation, STEMI acute event, symptoms, anger and hostility, anxiety and depression, interaction with healthcare professional, making a recovery, other support and information needs. Sub-themes were also developed and explored during indexing and the charting phase of the study.

In the mapping and interpretation stage of the analysis further exploration and expansion of the analysis identified four final themes, ‘Fear of experiencing a further heart attack’, ‘Uncertainty and inability to determine cause of symptoms’, ‘Insufficient opportunity to validate self-construction of illness’ and ‘Difficulty adaptation to life after a heart attack’. Each theme also had a number of sub-themes.
A ‘narrative’ using the words of the participants, was developed to draw out the important concepts involved with the themes and sub-themes. The main theme included the participants’ fear of experiencing another heart attack and for some dying from a heart attack. Many of the participants believed at the time of re-presentation that their symptoms were related to the occurrence of another heart attack. Throughout the interviews there was an undercurrent of anxiety relating to symptom interpretation and adapting to lifestyle changes. On the whole participants found it difficult to interpret symptoms and a variety of means were used to try and comprehend them. This included reflecting on their own and other peoples past experiences.

The implications of suffering a heart attack were referred to by participants and for many individuals involved them ‘having to slow down’ due to symptoms such as chest pains, breathlessness, fatigue or general debilitation. Some participants believed that the effects of the heart attack on their lifestyle as well as loss of control and financial worries had led to low mood or feelings of depression.

Differing degrees of depression were experienced by the participants, including severe, requiring in-patient admission, to feeling a ‘bit more weepy than usual’ when watching television. Worries relating to symptoms and the occurrence of a further heart attack were also attributed to feeling depressed. Components of the grieving process were also evident and some individuals expressed powerful emotions including anger, denial and guilt.

Many participants had made adjustments to their lifestyles, although for many this had been a difficult process. Gender differences appeared to come to light with the younger men who were in employment appearing to struggle to adapt to their changing roles. There was some suggestion that they found their masculinity to be in question. Women reported finding undertaking their usual household chores difficult and required the assistance of their partners or relatives.

The need to receive a diagnosis at the time of re-presentation appeared to be strong, although many participants reported not knowing the final diagnosis. On certain occasions, particularly when symptoms re-occurred, individuals adopted strategies such as self-diagnosis and self-treatment.

Many of the participants felt grateful for the PPCI treatment that they had received and were surprised and impressed at the speed of treatment. However, the lack of
‘illness ceremony’ due to the speed of treatment and early discharge appeared to add to the participants’ anxiety and disbelief at having experienced a heart attack.

There was a mixture of feelings regarding how useful cardiac rehabilitation was or the appropriateness of content. A number of interviewees found it helpful in boosting their confidence and in helping them to return to normal. Other participants reported a need for heightened physical assessment and others required more help with dealing with stress and psychological problems. Additionally there was general dissatisfaction relating to the amount of information and follow-up received. In particular participants reported receiving very little information relating to how to interpret and deal with symptoms.

Overall, participants were shocked at having suffered a heart attack. Additionally they experienced a great deal of anxiety and uncertainty in relation to symptoms. The seriousness of the heart attack event appeared ameliorated due to the speed of treatment and early discharge and in some cases this influenced a participants’ decision not to attend cardiac rehabilitation.
CHAPTER 9 DISCUSSION

9.1 Introduction

The main purpose of this study was to investigate the proportion of PPCI patients who re-presented acutely due to potential Ischaemic Heart Disease (IHD) symptoms, and to examine the factors associated with re-presentation within 6 months of STEMI and PPCI treatment. The definition of potential IHD symptoms in this study refers to the occurrence of symptoms that participants believed to be associated with their heart whether or not ultimately they were cardiac related (see section 3.2.1 for the full definition).

A conceptual framework was developed which enabled the researcher to highlight linkages between the most pertinent concepts potentially associated with re-presentation (see section 9.4). The potential relationships between the concepts were investigated in an explanatory mixed methods study. This included a quantitative phase leading to identification of the purposeful sample for the qualitative phase of the study.

Self-report measures the Seattle Angina Questionnaire (SAQ) and the Hospital and Anxiety and Depression Scale (HADS) were used to collect cardiac ischaemic symptom and psychological health data. Semi structured interviews were conducted to explore the experiences of PPCI patients who re-presented due to potential IHD symptoms.

In this chapter the findings from the quantitative and qualitative phases of the study are synthesised and interpreted in the context of other research findings. The unique findings from this study will be highlighted. Additionally the conceptual model and study methods will be evaluated, and the strengths and weaknesses of the study design will be discussed. Suggestions for further research, recommendations for policy and the implications for practice will also be discussed.

9.2 The main study findings

This is the first study to show that 18.8% of PPCI patients (95% CI 14.0% to 24.8%) re-presented to acute healthcare services with potential IHD symptoms during 6 month follow-up. Furthermore, the study determined the factors related to psychological health (anxiety and depression) as moderately associated with re-presentation. In particular, the study identified anxiety as the main predictor with
adjusted odds ratio 1.12 for re-presentation due to potential IHD symptoms (see section 7.10).

It was also determined that the re-presentation group experienced significantly higher HADS anxiety, depression and psychological distress scores at both baseline and 6 months than the non-representation group. Most of the SAQ symptom scores were stable and did not differ between the groups at baseline, although quality of life was moderately reduced for both groups at both time points and at 6 months angina frequency was statistically significantly increased in the re-presentation group.

The qualitative study showed that participants found recognising and differentiating between symptoms difficult and anxiety-provoking. Additionally, adaptation to life following STEMI was also challenging (see section 9.6.4). The results of the quantitative and qualitative phases of the study can be seen in chapters 7 and 8 respectively. These findings are discussed further later in this chapter.

9.3 The study cohort

In terms of being representative, this study cohort was similar in relation to mean age, gender split and comorbidity of previous ‘representative’ PPCI study cohorts (see Table 9-1). In this section of the thesis ‘representative’ refers to cohorts that mirror the general PPCI population (i.e. including low, medium and high risk patients). Cohorts included as representative consist of unselected patients from clinical populations recruited as part of large patient registries, observational and some cohort studies (with few exclusion criteria). This is opposed to highly selective patients selected for randomised controlled trials (RCTs). The study cohort in this chapter of the thesis refers to participants who responded to baseline questionnaires (202).

The cohort included 75.5% men and the mean age was 59.7 years (26 to 87 years). Overall, at baseline the group were relatively healthy with very few comorbidities. This was demonstrated by a low predictive weighted Charleson Co-morbidity Index (CCI), with a mean of only 0.7 (CCI score 0= no comorbidities and CCI score 1= low levels of comorbidities, in contrast a score of 33= almost certain death). The CCI was not measured in the other studies presented in Table 9-1, but rates of diabetes were presented and are used as a comparison of comorbidity with the current study cohort. Rates of diabetes were relatively low and similar between the current study (19.8%) and the majority of representative studies presented in Table 9-1. Additionally, in this study prior cardiac events were somewhat low with less than a quarter (23.9%) of
participants previously experiencing events such as angina, cardiac arrhythmia or heart failure.

The groups in this study were generally similar overall and demonstrated only three baseline clinical differences. These included more of the re-presentation group compared to the non-representation group receiving PCI prior to STEMI (21.1% vs 5.5%, p=0.002), more had reduced LV function of <30 (16.7% vs 4.4%, p=0.056) and more patients reported having received treatment for depression prior to STEMI (40.0% vs 14.6%, p<0.001). Furthermore at 6 months more of the re-presentation group chose not to attend all the cardiac rehabilitation sessions offered (35.1% vs 56.8%, p=0.032); the potential reasons for this are discussed in section 9.6.2.

A higher proportion of patients in the re-presentation group with reduced LV function and receiving PCI prior to STEMI may indicate that the group had more prior cardiac morbidity than the non-representation group. Other cardiac morbidity baseline measures were all similar for the re-presentation and non-representation groups including no prior myocardial infarction (MI) (81.6% vs 86.0%, p=0.657), level of heart failure (measured using the New York Heart Association (NYHA) score) and angina (measured using the Canadian Cardiovascular Society Angina Grading Scale (CCSC)); see section 7.3.4 for NYHA and CCSC categorical results.

Nevertheless, 21.1% of the re-presentation group compared to 5.5% of the non-representation group (p=0.002) had previously received PCI, indicating that IHD symptoms were severe enough to require intervention whether or not these individuals had experienced a previous MI. However, PCI is likely to have improved symptoms for those affected in the re-presentation group and this is the likely reason for equivalent CCSC scores at baseline between the groups. It therefore cannot be confirmed or discounted that the re-presentation group had a greater prior cardiac morbidity than the non-representation group (discussed further in section 9.6.3.1).
Table 9-1 Comparison of current study cohort with those of previous relevant studies.

<table>
<thead>
<tr>
<th>Author</th>
<th>Cohort</th>
<th>Age (mean yrs)</th>
<th>Gender Men (%)</th>
<th>Previous MI (%)</th>
<th>Previous PCI (%)</th>
<th>Diabetes (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current study</td>
<td>PPCI (cohort study)</td>
<td>59.7 (26-87)</td>
<td>75.7</td>
<td>14.9</td>
<td>8.4</td>
<td>19.8</td>
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<tr>
<td>Lambert et al (2010)</td>
<td>PPCI (representative)</td>
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<td>74.0</td>
<td>13.5</td>
<td>7.4</td>
<td>17.3</td>
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<tr>
<td>Ortolani et al (2009)</td>
<td>PPCI* (representative)</td>
<td>67.0 (±13)</td>
<td>71.0</td>
<td>16.0</td>
<td>9.0</td>
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<tr>
<td>Jakobsen et al (2010)</td>
<td>PPCI (representative)</td>
<td>65.2 (62-66)</td>
<td>74.1</td>
<td>12.4</td>
<td>Unk</td>
<td>9.0</td>
</tr>
<tr>
<td>Mortensen et al (2005)</td>
<td>PPCI (DANAMI-2, RCT-sub study)</td>
<td>62.2 (25-95)</td>
<td>75.0</td>
<td>10.4</td>
<td>4.4</td>
<td>7.2</td>
</tr>
<tr>
<td>Anderson et al (2003)</td>
<td>PPCI (DANAMI-2, RCT)</td>
<td>63.1 (62-64)</td>
<td>74.0</td>
<td>9.2</td>
<td>Unk</td>
<td>7.1</td>
</tr>
<tr>
<td>Stone et al (2002)</td>
<td>PCI (CADILLAC- RCT)</td>
<td>60.0</td>
<td>72.5</td>
<td>11.9</td>
<td>8.8</td>
<td>16.5</td>
</tr>
<tr>
<td>Hubbard et al (2007)</td>
<td>PCI- mixed cohort (representative)</td>
<td>64.3</td>
<td>68.0</td>
<td>Unk</td>
<td>Unk</td>
<td>27.6</td>
</tr>
<tr>
<td>McGowan et al (2004)</td>
<td>STEMI (cohort study)</td>
<td>57.6 (±11.2)</td>
<td>Unk</td>
<td>Nil</td>
<td>1st MI</td>
<td>Unk</td>
</tr>
<tr>
<td>Dickens et al (2006)</td>
<td>STEMI (cohort study)</td>
<td>57.6 (±11.2)</td>
<td>63.0</td>
<td>Nil</td>
<td>1st MI</td>
<td>Unk</td>
</tr>
<tr>
<td>Roest et al (2010)</td>
<td>STEMI Meta-analysis (12 studies)</td>
<td>54.0 to 63.0</td>
<td>82.5</td>
<td>Unk</td>
<td>Unk</td>
<td>Unk</td>
</tr>
<tr>
<td>Van Melle et al (2004)</td>
<td>STEMI Meta-analysis (22 studies)</td>
<td>61.0</td>
<td>75.0</td>
<td>Unk</td>
<td>Unk</td>
<td>Unk</td>
</tr>
<tr>
<td>Dickens et al (2007)</td>
<td>STEMI (cohort study)</td>
<td>60.0 (±11.1)</td>
<td>70.4</td>
<td>19.0</td>
<td>Unk</td>
<td>9.7</td>
</tr>
</tbody>
</table>

Unk - data unknown
Representative- representative of the general PPCI population
‡PPCI cohort only presented for the DANAMI-2 sub study
± standard deviation
^K characteristics of the group receiving stents during PPCI
¥ characteristics of the placebo group who received PPCI and no glycoprotein IIb/IIIa agent (IIb/IIIa agent)
*early treatment (prior to cath lab) with agent at time of MI, **usual MI care with glycoprotein IIb/IIIa delivered in the cathlab.
9.4 Evaluation of the conceptual framework

The conceptual framework identified potential IHD symptoms, psychological health and physiological health as the most likely factors directly related to re-presentation due to potential IHD symptoms. Sociodemographic aspects were identified as indirectly related (see Figure 9-1).

Figure 9-1 Concepts included in the conceptual model

An in-depth review of the literature and appraisal of clinical practice led to justification of the factors included in the model (see conceptual framework Chapter 3).

9.4.1 Potential IHD symptoms

The patients’ experience of angina symptoms at baseline, assessed using the self-report measure the Seattle Angina Questionnaire (SAQ), was not predictive of re-presentation due to potential IHD symptoms in this study. The quantitative study analysis for aim 2 showed that symptoms of angina were stable, infrequent and equivalent for both groups. The low levels of angina are likely to have been the main reason that SAQ angina stability and SAQ angina frequency scores were not predictive of re-presentation in the regression model. The good stability and infrequency of angina symptoms may have been due to the quick and effective treatment delivered through PPCI.

Nevertheless, potential IHD symptoms featured strongly in the qualitative study findings due to the participants’ uncertainty regarding symptom attribution. Additionally the analysis for aim 5 showed a significant negative correlation with moderate effect for SAQ angina frequency and HADS scores at baseline for both groups. The correlation supports the conceptual framework, as the framework
postulated that a reciprocal relationship existed between psychological health and symptoms. Within the structure of this study it was not possible to show the causal relationship between the baseline SAQ angina frequency and HADS scores.

**9.4.2 Psychological health**

Through logistic regression modelling (aim 6) psychological distress (total HADS anxiety and HADS depression scores) was shown to be the most statistically significant predictor of re-presentation. However, on theoretical grounds HADS anxiety was included in the final logistic regression model and was found to be the main predictor of re-presentation (see section 3.5 of conceptual framework regarding justification of regression model variables). Additionally the descriptive statistics in aim 2 identified that significantly more of the re-presentation group reported symptoms of anxiety at baseline (HADS score ≥8) than the non-representation group (65.8% vs 39.0%, p=0.003). Furthermore throughout the qualitative study interviews there was evidence of reduced psychological health.

Numerous previous studies have reported negative effects on psychological health immediately following STEMI as a phenomenon (Strik et al., 2003, Frazier et al., 2002, Grace et al., 2004, Ladwig et al., 1994). The common occurrence of reduced psychological health following STEMI and the similarity of some of the symptoms to those associated with anxiety and depression may explain the findings of the current study.

The quantitative and qualitative findings of the study suggest that there was an association between reduced psychological health and re-presentation to acute healthcare services due to potential IHD symptoms. The inclusion of psychological health in the conceptual model is therefore upheld.

**9.4.3 Physiological health**

Physiological health, measured in terms of comorbidity and severity of the original myocardial infarction, was not identified as a predictor of re-presentation in this study. However, the discharge diagnoses for those who re-presented included both cardiac (ischaemia or cardiac cause) and non-cardiac or comorbid (musculoskeletal, gastric, and pulmonary) causes. In the qualitative study findings participants spoke of believing that they had suffered a further heart attack at the time of re-presentation. Additionally some individuals believed that ongoing symptoms were related to a physiological cause such as indigestion or a musculoskeletal problem.
This demonstrates that physiological health was an appropriate aspect of the conceptual framework.

The Charleson Co-morbidity Index (CCI) was used to measure comorbidity (Charlson et al., 1987). The most likely reason that comorbidity was not found to be predictive in the logistic regression model in this study was the low level of comorbidity reported for the current study cohort. The mean CCI score was low (0.7) for the study cohort, demonstrating that overall the cohort were relatively well and had few comorbidities.

The Global Registry of Acute Coronary Events (GRACE) was used to assess severity of the myocardial infarction (Granger et al., 2003). The GRACE probability of death or MI at 6 months was low to moderate (mean 28.5%) for the cohort as a whole. Interestingly, the mean GRACE probability of death or MI at 6 months in the current study was more than twice as high as that of the actual 10.4% death and cardiac re-presentation rate (included STEMI, N-STEMI, Acute Coronary Syndrome (ACS) event, angina or cardiac arrhythmia) at 6 months. This was most likely influenced by the very low mortality rate of 1.5% for the current cohort compared to the 7.0% predicted GRACE probability of death at 6 months.

Some of the potential reasons to explain this disparity include the heterogeneous GRACE cohort and the point in time that the GRACE study was conducted. The GRACE study involved 11,389 ACS patients recruited between 1999 and 2001 (Granger et al., 2003, Fox et al., 2006). The GRACE study included mostly (65%) non-STEMI or unstable angina patients; in contrast the entire current study cohort had a diagnosis of STEMI. Additionally, the majority of STEMI patients in the GRACE study are likely to have received thrombolysis (lysis) as this was first line treatment in most countries at the time of the study. In the current study the cohort received PPCI. It is now well recognised that PPCI is superior to lysis in terms of mortality and morbidity (Keeley et al., 2003) and this may have influenced the improved outcome in the current study.

In the present study the GRACE appears to overestimate death and MI at 6 months and it is therefore possible that GRACE is not an appropriate or accurate measure for use in PPCI cohorts. Little evidence is available demonstrating validation of the GRACE score in present STEMI cohorts receiving PPCI. This may partly be due to the only recent recommendation by the National Institute for Health and Clinical Excellence (NICE), that GRACE should be adopted by clinicians to formally assess individual risk of future adverse cardiovascular events (National Institute for Health
and Clinical Excellence, 2010b). Additional publications are awaited to enable further evaluation of the GRACE score specifically for PPCI patients. Until more evidence is available to support the use of GRACE in PPCI patients it may be prudent to incorporate an additional measure of severity of MI such as a biomarker (e.g. Troponin) or left ventricular function (see conceptual framework) in future similar studies.

9.4.4 Sociodemographic factors

Sociodemographic factors were identified in the conceptual framework as potentially indirectly associated with re-presentation due to potential IHD symptoms. The specific factors identified included age, gender and social support (measured using The Enhancing Recovery in Coronary Heart Disease (ENRICHD) social support instrument (ESSI)) (ENRICHD Investigators, 2000, Vaglio et al., 2004).

However, ultimately none of these factors were included individually in the final regression models (although age was part of the GRACE and the CCI score). This was due to potential collinearity and the limitation in the number of variables that could be incorporated, according to the rules of Peduzzi et al (1996) and Vittinghoff and McCulloch (2007) (see section 7.10). It was therefore not possible to determine whether sociodemographic factors such as gender or social support held any association with re-presentation in this study within a multivariable statistical model, nor was it possible to evaluate the conceptual model in terms of these factors. According to the literature reviewed in section 3.5.3 it would seem theoretically appropriate for sociodemographic factors to remain in the conceptual framework when applied to a future and larger study investigating similar phenomena to the current study.

The bivariate analysis for sociodemographic variables between groups did not reach statistical significance, although there were small differences between the groups with regards to gender and the ESSI scores. There were fewer men in the in re-presentation group (65.8%) compared to the non-representation group (78.0%, p=0.112) and the ESSI scores were also lower for the re-presentation group (27.7% vs 28.9, p=0.277). It is possible that with a larger cohort differences may have reached significance.
9.4.5 Reflection on the framework

Overall the study findings supported the conceptual framework. Psychological health and symptoms were found to share a relationship and psychological health was determined to be the main predictor of re-presentation.

Physiological health was not found to be a statistically significant predictor of re-presentation, although a number of re-presentation events received a cardiac or non-cardiac comorbid diagnosis. This may have been related to the measures that were used in the study to assess physiological health. Nevertheless, the inclusion of physiological health in the model was considered appropriate and it appears justifiable to include this concept in a future conceptual model underpinning studies investigating similar phenomena. Additionally in future studies it would also be desirable to include additional variables to investigate physiological health; sociodemographic variables may also be included in a similar model as part of a larger study.

On reflection the conceptual framework was a helpful means of framing the study as a whole. However, overall the framework could not be fully tested due to the limited number of variables that could be included in the regression analysis due to the small number of re-presenters. It is therefore possible that aspects of the framework may not fully reflect the core issues related to re-presentation due to potential IHD symptoms. The framework requires further development and evaluation based on the findings of this study to fully identify the main concepts related to the phenomenon under investigation. The concept of illness perceptions may be worthy of additional inclusion in a similar future model as reflected by the qualitative findings of this study.

9.5 Re-presentation rates and causes

In the current study 18.8% of participants (n=38) re-presented at some point during 6 month follow-up with symptoms that they believed to be related to their heart. Many of those who re-presented did so once (16) or twice (12). A total of 74 potential IHD events occurred across the re-presentation group.

Aim 1 not only set out to identify the re-presentation rate but also the causes of re-presentation. More than a third (39.2%) of the 74 events (the largest proportion), were categorised as 'no diagnosis'. The second largest category showed that 24.3% of events were categorised as cardiac ischaemia or cardiac cause. Other causes were relatively uncommon such as musculoskeletal (5.4%), gastric (4.1%),
psychological (4.1%) and pulmonary (2.6%). The mortality rate for the 202 responders was only 1.5% (3) in this study and all deaths occurred in the non-representation group.

The overall potential IHD symptom re-presentation rates are discussed in this section and compared to those reported in previous studies. Additionally, the two main discharge diagnosis categories, no diagnosis and cardiac ischaemia or cardiac cause, are also discussed.

9.5.1 Reported potential IHD symptom re-presentation rates

The bulk of existing PPCI re-presentation or incident rates reported in the literature are often through the conduct of highly selective randomised clinical trials (RCTs). They also generally only include death, cardiac related episodes, composite event rates or hospitalisation rates. These differ from the current study definition of re-presentation, which includes patients who attended acute healthcare services for assessment due to cardiac and non-cardiac IHD symptoms.

Comparison of the current study re-presentation rates with those of previous studies is difficult due to the lack of literature reporting re-presentation rates due to potential IHD symptoms for PPCI patients. However, some comparison may be drawn with a retrospective study conducted by Hubbard et al (2007), see section 2.12.3.1 of the literature review for details of this study. In summary, Hubbard et al (2007) reported that 9.3% (255) of patients were investigated for potential cardiac ischaemia when blood was tested for a serum cardiac biomarker during accident and emergency (A&E) attendance 30 days following percutaneous coronary intervention (PCI). The following diagnoses were reported: 33.0% (84) non-specific chest pain, 20.0% (51) coronary atherosclerosis, 6.0% (15) MI, 6.0% (15) heart failure and 5.0% (13) cardiac dysrhythmias while 30.0% (77) did not receive a diagnosis.

The 9.3% re-presentation rate (due to potential cardiac ischaemia) within 30 days appears to be much higher than the 6.9% (14) rate at 30 days (18.8% at 6 months) for the current study cohort (202). The possible reasons for this difference include the different cohorts and respective treatments. Hubbard et al (2007) study included a heterogeneous cohort receiving PCI treatment under a variety of circumstances including 18.9% (518) elective PCI, 17.7% (483) emergency PCI and 63.4% (1,730) urgent PCI; 46.0% of PCI’s occurred in the setting of acute MI. In contrast the entire current study cohort experienced acute MI and received PPCI.
Hubbard et al (2007) did not clearly state what initial treatment (i.e. PPCI or thrombolysis (lysis)) MI patients received. The 46.0% of patients receiving PCI in a setting of MI seemed incongruous with the 17.7% of emergency PCIs (including PPCI). It is therefore most likely that the majority of MI patients received lysis along with urgent PCI (during an unscheduled hospital admission). The superior index treatment of PPCI conducted in the current study may therefore partly explain the potential differences between the re-presentation rates with those of the Hubbard et al (2007) study.

However, there are several other possible reasons for the lower 30 day re-presentation rate for the current study. In the Hubbard et al (2007) study the participants were categorised as being investigated for cardiac ischaemia when they had blood analysed for a serum cardiac biomarker. This does not necessarily indicate that a patient has presented with potential IHD symptoms. Generally when patients return to acute services shortly after being treated with PCI, as a matter of caution physicians routinely assess the patients’ cardiac biomarkers (National Institute for Health and Clinical Excellence, 2010b). Methodological aspects of the two studies were also different. Hubbard et al (2007) used retrospective methods to identify the study cohort and collect data. In comparison, the current study used prospective study methods, including consecutive sampling and prospective data collection, which enhanced the accuracy of the data collected. However, taking into consideration these differences, overall the re-presentation rates for the current study are broadly in-line with those reported by Hubbard et al (2007).

Interestingly, 30.0% of participants did not receive a diagnosis in the Hubbard et al (2007) study. It is possible that some of those who did not receive a diagnosis had re-presented due to reduced psychological health (see section 9.5.2.1 for further discussion). Unlike the present study Hubbard et al (2007) did not consider or investigate both physiological and psychological factors related to re-presentation. This sets the current study apart from the existing literature and the findings offer a unique insight into the mixed causes of re-presentation for PPCI patients.

### 9.5.2 Causes of representation

#### 9.5.2.1 No diagnosis

More than a third (39.2%) and the largest proportion of the 74 re-presentation events in this study did not have a definitive discharge diagnosis recorded in the participants’ health records. These events were categorised as receiving ‘no
diagnosis’ in this study. The ‘no diagnosis’ category comprised of absence of any diagnosis, or when a physician had stated in the patient records ‘not cardiac’, ‘atypical chest pain’ or ‘troponin negative chest pain’, all of which suggest that the symptoms were not serious in cardiac terms.

The qualitative findings for the current study showed that participants found the lack of a diagnosis at the time of re-presentation anxiety provoking. When a diagnosis was not available some individuals tried to determine the diagnosis themselves by reflecting on previous symptoms and illnesses experiences. The need for a diagnosis fed into the second qualitative theme ‘uncertainty and inability to determine cause of symptoms’. Absence of a diagnosis appeared to heighten the individuals’ anxiety regarding the cause of symptoms and amplify their inability to differentiate between symptoms, as they were unable to make sense of the re-presentation event.

The large proportion of events that were categorised as ‘no diagnosis’ may be due to a number of factors. A possible explanation is that in the acute situation, physicians may be focussed of identifying cardiac ischaemia and may not look further when ischaemia is excluded. Additionally they may find determining a final diagnosis difficult when the results of investigations are negative, particularly if symptoms are atypical or vague. These challenges may be more difficult for junior or less experienced physicians, or those who do not have cardiology expertise. Furthermore, limited investigations may be available during an acute admission (e.g. thalium scan, exercise ECG, stress echo) and further investigations may be required as an outpatient. Data relating to subsequent post discharge investigations were not collected as part of this study. The limitations of this study are discussed later in section 9.8.1).

Determining the cause of potential cardiac symptoms can be complex and taxing for not only general physicians but also for cardiology specialists. Sekhri et al (2007) categorised more than 8000 attendees at rapid access chest pain clinics over 28 months as suffering either ‘angina’ (n=2366) or as ‘non-cardiac’ (n=6396). Rapid access chest pain clinics are generally run by cardiologists or clinicians with specialist knowledge of coronary ischaemia. Despite this heightened level of expertise misdiagnosis occurred for 194 (3.0%) patients in the ‘non-cardiac’ group of this study. Although this is a small percentage of misdiagnosis the consequences for patients could lead to serious detrimental outcome in terms of mortality and morbidity. Sekhri et al (2007) study highlights the challenges faced by physicians in determining a diagnosis when potential IHD symptoms occur.
Additionally, it is possible that medical staff working in a specialist area such as cardiology may only be interested in or feel equipped to deal with cardiology problems. Although this has not previously been reported in the literature, it is possible that medical staff choose not to pursue addressing non-cardiac aspects of the patient’s health, such as psychological health, even when they suspect that such a problem exists. This may partly explain the small proportion (4.1%) of re-presentation events categorised in this study as related to psychological health. This finding appears to contradict the results of the logistic regression analysis that psychological health was the main predictor of re-presentation in this study. At least half of the re-presenters had HADS psychological distress cut off scores ≥17 both at baseline (19/38) and at 6 months (20/38), so it is likely that half of the patients would be showing psychological distress at the re-presentation events.

A further possible reason for the high number of participants with ‘no diagnosis’ may relate to the findings of the physical assessment in accident and emergency (A&E). Once serious or life threatening conditions have been excluded, the need to determine a diagnosis may become less important for the physician, particularly when there is a need to assess other more potentially seriously ill patients. Goodacre (2006) proposed that A&E doctors often consider whether to admit or discharge an individual based on level of risk of adverse outcome rather than based on the potential benefits of admission.

A further consideration is that physicians could have been concerned about litigation and may not have been willing to indicate that the condition was not ‘serious’. Therefore failure to define a diagnosis in this study may have been an act of ‘defensive practice’ (Studdert et al., 2005). A culture of defensive practice has been reported in previous studies in the United Kingdom (Summerton, 1995, Passmore and Leung, 2002). Researchers have found that physicians altered their practice and in some instances the language that they used during record keeping to limit the possibility of litigation (Summerton, 1995, Goodacre, 2006).

Poor reporting or inappropriate labelling of a diagnosis may also account for some of the missing diagnoses. Inadequate record keeping has long been reported in the NHS, particularly pertaining to patient hospital records (Pullen and Loudon, 2006, Gray, 2008, Mann and Williams, 2003, Chauhan et al., 2010). It is therefore feasible that this is one of the reasons for the missing or inadequate detail relating to the re-presentation diagnosis for some of the participants in this study.
9.5.2.2 Cardiac ischaemia or cardiac cause

Sixteen (42.1%) of the re-presentation group received a diagnosis of cardiac ischaemia or cardiac cause related to at least one re-presentation event during the 6 month follow-up. This translates as 7.9% of the cohort (202). All the patients in this study who were categorised with a cardiac diagnosis were admitted to hospital.

Numerous previous studies have reported cardiac ischaemia event rates for MI patients, although the majority involve composite events including death. Section 2.3.1 reviews the literature on post STEMI cardiac events. The proportion of patients either dying or experiencing a cardiac ischaemia or cardiac cause re-presentation event (at least once) for the current study at 6 months was relatively low at 9.4% for the cohort. Comparison of cardiac events rates between this and other studies is challenging. This is due to the different composite event rates and the variety of follow-up time points. Moreover many of the studies available for comparison were conducted during the last decade during which time the advancements in PPCI techniques and therapies have positively influenced mortality and morbidity outcomes. Despite these factors, the range of reported death or cardiac related events in the literature is reasonably consistent with the reported rates in this study.

The current study rate of 9.4% (death and cardiac ischaemia or cardiac related cause) at 6 months compares with 13.6% (combined death and readmission at 12 months) from Lambert et al (2010), 8.9% (cardiac events at two years) from Ortolani et al (2009) and 11.5% (cardiac event rate at 6 months) from Stone et al (2002). Table 8-1 presents details of the study cohorts for the previously mentioned studies.

The methodological differences between this study and previous studies can be summarised in the following way:

- **Assessment time point:** This study measured death and cardiac re-presentation rates at 6 months whereas Lambert et al (2010) measured them at 12 months and Ortolani et al (2009) measured them two years.

- **Methodologies:** For example, Lambert et al (2010) used retrospective data collection from patient notes as opposed to a combination of prospective and retrospective methods used in this study. Stone et al (2002) and Ortolani et al (2009) conducted RCT’s.
• **Diagnoses:** In this study, the cardiac event rates incorporated a wider range of cardiac diagnoses including STEMI, N-STEMI, ACS event, angina or cardiac arrhythmia, as well as including both A&E attendances and hospital admissions. By contrast, Lambert et al (2010) for example only included readmission for heart failure or MI.

• **Timing of studies:** Some of the studies available for comparison were conducted in that last decade, and advances in PPCI techniques and therapies have positively impacted mortality and morbidity outcomes. For example the CADILLAC study (Stone et al., 2002) was conducted during the early part of the last decade when PPCI and adjunctive therapies were still evolving.

The re-presentation rates due to cardiac ischaemia or cardiac cause reported for the current study appear to be feasible and representative for PPCI patients in the present era.

**9.6 The influence of psychological health on re-presentation**

The conceptual framework identified psychological health as one of the potential factors leading to re-presentation (see chapter 3). Through the quantitative study findings, reduced psychological health was determined to be the most important predictor of re-presentation. Additionally, aspects of psychological health featured strongly in the qualitative study findings.

A multitude of complex reasons relating to reduced psychological health may play a role in the re-presentation (due to potential IHD symptoms) of PPCI patients. In this study these included participants experiencing shock at having had a heart attack as well as fear of death and dying. A pre-disposition to anxiety and depression and the treatment itself (PPCI) may also have been implicated with reduced psychological health at baseline. Furthermore hypervigilance of symptoms and difficulty with symptom attribution may also have played a part for some participants.

In this section the results from the quantitative analysis and the likely reasons that psychological health (anxiety and depression) was predictive of re-presentation will be discussed.
9.6.1 Symptoms of anxiety

Symptoms of anxiety were a central factor in relation to re-presentation in the current study. Importantly, the analysis for aim 6 identified that symptoms of raised anxiety were predictive of re-presentation at 6 months. This association remained present even when controlling for other factors that may have the potential to affect re-presentation rates, for example physiological health (severity of STEMI and comorbidity) and symptoms.

The logistic regression model 3 (see section 7.10.3) had an adjusted odds ratio (OR) of 1.12 for the HADS anxiety score. This is the factor by which the odds of representing are multiplied by when the HADS anxiety score increases by 1 and the other variables remain fixed. Therefore, if the HADS anxiety score increases by (x), then the odds of representing are increased by a factor of 1.12(x) (see Figure 9.2).

Figure 9-2 Increase in baseline HADS anxiety scores and relationship with re-presentation based on OR 1.12*

*The odds of re-presenting are calculated as the probability of re-presenting divided by the probability of not re-presenting.

This is the first study to show that raised anxiety levels, measured using the HADS, predict re-presentation due to potential ischaemic cardiac symptoms in STEMI patients, even after adjustment for other important variables.
9.6.1.1 Previous studies investigating MI and anxiety

Other researchers have investigated the roles that anxiety and depression play in terms of mortality and morbidity of post MI patients, but re-presentation due to potential cardiac symptoms has not previously been investigated. It is therefore not possible to compare these studies directly with the current study results. However, some insight may be gained through review of literature related to anxiety, mortality and/or cardiac events as such patients may have re-presented with IHD symptoms.

A recent meta-analysis of 12 papers describing STEMI patients, showed anxiety to be associated with cardiac events [Odds Ratio (OR) 1.71], all cause mortality (OR 1.47) and cardiac mortality (OR 1.23) (Roest et al., 2010). Only papers using validated instruments to measure anxiety within 3 months of STEMI were included in the analysis; mean follow-up was 2.6 years.

Similarly, in another meta-analysis of 22 studies investigating hospitalised MI patients, depression was shown to be associated with both all-cause mortality (OR 2.38), cardiac mortality (OR 2.59) and cardiac events (OR 1.95) at mean follow-up 13.7 months (van Melle et al., 2004). It is unlikely that the majority of patients in this meta-analysis and the one conducted by Roest et al (2010) would have received PPCI. This is because most of the included studies were conducted in the 1990s prior to the introduction of PPCI as first line treatment for STEMI. See Table 8-1 for details of both study cohorts, which were similar to that of the current study.

Neither van Melle et al (2004) or Roest et al (2010) investigated patients who attended with cardiac or non-cardiac IHD symptoms. The current study therefore plays an important part in highlighting the role that anxiety plays in the re-presentation of patients who experience potential cardiac ischaemic symptoms.

9.6.1.2 Comparison of HADS anxiety scores with previous studies

Analysis for aim 2 showed that the re-presentation group reported significantly increased mean HADS anxiety scores at both baseline and 6 months compared to the non-representation group. Furthermore, the re-presentation group experienced mean anxiety scores in the mild range (according to Zigmond and Snaith’s guidance) at baseline (9.5) and remained the same at 6 months (9.4). Conversely mean anxiety scores for the non-representation group were in the normal range at both time points.
A substantial amount of research has previously focused on depression and Coronary Heart Disease (CHD), but fewer studies have focused on anxiety and STEMI. Additionally to date no studies have presented HADS scores for STEMI patients who have re-presented with potential IHD symptoms.

One study that presented mean HADS anxiety scores for hospitalised MI patients was conducted by McGowan et al (2004). The authors investigated the relationship between depression and vital exhaustion; the majority of patients had received thrombolysis (lysis). McGowan et al (2004) reported ‘normal’ baseline mean HADS anxiety scores of 7.4 which were measured at a mean of 3.6 days after MI. This reflects the current study baseline findings for the non-representation group (HADS mean anxiety 7.1) and the study cohort as a whole (HADS mean anxiety 7.5), and identifies the re-presentation group as different in terms of their psychological health early on following STEMI. The similarity of the HADS scores for the two studies appears to indicate that the underlying STEMI, rather than STEMI treatment differences had an effect on psychological health. This theory is supported by the more recent study conducted by McGowan et al (2012) (the main study related to the current study), which confirmed that mean HADS anxiety scores for the PPCI and lysis groups (7.2 v 6.4, p=0.140) were similar at baseline.

Further comparison can be drawn between the HADS cut offs (≥11) in the current study and those presented for the PPCI cohort in the DANAMI-2 sub-study (Mortensen et al., 2005). The DANAMI-2 sub-study looked at the health related quality of life and psychological health of STEMI patients who received either PPCI or thrombolytic treatment (Mortensen et al., 2005). Follow-up data were only presented at one month post STEMI limiting information on long term psychological health outcomes information. See the literature review, section 2.10.2 for further details of this study.

In the current study at baseline (mean 19.4 days post MI) 40.0% of the re-presentation group reported moderate to severe levels of anxiety (i.e. HADS scores ≥11) compared to 23.0% of the non-representation group. Moderate to severe anxiety was seen in a quarter (26.0%) of the cohort as a whole. In comparison, 32.5% of patients in the DANAMI-2 sub-study had moderate to severe levels of anxiety at 30 days post PPCI, indicating similar levels of anxiety early after PPCI in the two studies (current study at 3 weeks and DANAMI-2 at 4 weeks).

The three studies discussed and the current study all demonstrate that some patients do experience moderate to severe symptoms of anxiety following STEMI.
and PPCI (McGowan et al., 2004, Mortensen et al., 2005, McGowan et al., 2012). Neither McGowan et al (2004) nor Mortensen et al (2005) reported re-presentation rates (due to potential IHD symptoms) and did not set out to explore the potential association with anxiety. The current study is therefore unique in indicating that anxiety is associated with re-presentation, an important finding in the context of previous studies that report increased mortality and morbidity associated with raised anxiety following STEMI (Roest et al., 2010, van Melle et al., 2004).

9.6.1.3 Symptoms of depression

The descriptive analysis in aim 3 suggested that some of the participants developed symptoms of depression during the 6 month follow-up. Levels of depression symptoms at baseline (mean 7.4) were in the normal range (HADS scores <8), whereas at 6 months they were ‘mildly’ raised (mean 8.4) for the re-presentation group albeit a non-significant increase (p=0.073). Conversely, for the non-representation group levels of both anxiety and depression symptoms improved at 6 months and were in the ‘normal’ range at both time points, although analysis for aim 4 did not show a significant difference for change in HADS anxiety and depression scores at 6 months.

Anxiety and depression are known to coexist, as each acts as a comorbidity for the other (Bech, 2006). It is possible that the increased depression levels at 6 months for the re-presentation group were due to the continuation of raised anxiety and the reciprocal relationship between anxiety and depression. This may partly explain the increase in the mean depression scores from the ‘normal’ range to ‘mildly’ raised at 6 months for the re-presentation group.

It is also possible that for those who re-presented in this study, symptoms of depression developed as a result of a reaction to the heart attack event and the burden of required adaptations. During the qualitative interviews in theme 4 (adaptation to life after a heart attack) the concepts ‘grieving for loss of self and past life’ and ‘forced change of identity’ experienced by participants may have contributed to the development of symptoms of depression (see section 9.6.4 for further discussion of adaptation). However, to establish the course of anxiety and depression during this study, repeated measures of HADS during the course of the 6 months would have been required (study limitations are discussed in section 9.8.1).
9.6.2 Pre-existing reduced psychological health

The reason for raised anxiety levels in the re-presentation group is unclear. One possibility is that participants may have already been experiencing anxiety at the time of STEMI. To establish previous episodes of anxiety and depression participants were asked to self-report whether they had received treatment for anxiety or depression prior to their heart attack.

Previous episodes of anxiety were relatively low in both groups. A higher percentage (17.1%) of the re-presentation group self-reported receiving treatment for anxiety prior to STEMI than the non-representation group (10.2%), although the difference was not significant. Interestingly, as many as 40.0% of the re-presentation group had been treated for depression prior to their heart attack compared to significantly fewer (only 14.6%, p<0.001) of the non-representation group.

The usefulness of these data may be questionable as it is not clear at what time point individuals received treatment for anxiety or depression prior to STEMI, as this was not explored in this study. Additionally ‘not being in receipt of treatment’ does not necessarily mean that an individual has not suffered from anxiety. Patients do not always seek help for anxiety or in fact accept help when it is on offer. It therefore cannot be confirmed or discounted whether participants were already experiencing anxiety or depression at the time of STEMI in this study. There is also no way of determining whether ‘new onset anxiety’ or ‘pre-existing anxiety’ or a combination of both, played the greatest part in predicting re-presentation due to potential IHD symptoms in this study. Nevertheless it is important to acknowledge that significantly more of the re-presentation group reported experiencing reduced psychological health (in the form of depression) compared to the non-representation group. This may indicate a pre-disposition to psychological health problems.

9.6.3 Interpretation of potential causes of reduced psychological health

The qualitative work undertaken as part of this study highlighted some of the reasons that anxiety was implicated in re-presentation due to potential IHD symptoms. Primarily, participants were anxious at having experienced a heart attack and fearful that they may experience a further heart attack or cardiac event, demonstrated by the main qualitative theme, ‘fear of experiencing a further heart attack’.
attack’. Participants also appeared to have difficulty with the attribution of symptoms and conflicts about the severity and longevity of their illness.

9.6.3.1 Shock at experiencing a heart attack

It is not surprising that patients experience distress during or following a life threatening event, as anxiety at such a time is a natural emotional reaction (Whitehead et al., 2005).

The participants in this study were interviewed more than 6 months after their heart attack. Despite this lapse in time, they were still able to recount the strong emotions, including shock and anxiety that they experienced soon after their heart attack. The strong emotions described by the interviewees appear to correspond with the high proportion (65.8%) of the re-presentation group reporting symptoms of anxiety (HADS score ≥8) at baseline (mean 19 days); in comparison significantly fewer (39.0%, $p=0.003$) of the non-representation group reported symptoms of anxiety as identified in the analysis for aim 2. At 6 months symptoms of anxiety (HADS scores ≥8) remained different for the re-presentation and non-representation groups respectively (67.6% vs 34.7%, $p<0.001$).

It is possible that an early strong emotional response following STEMI led to raised anxiety levels at baseline in this study. Furthermore, heightened anxiety with the occurrence of symptoms (leading to re-presentation) may have led to the continuation of anxiety symptoms at 6 months (see section 9.6.3.3 for further discussion). It is not possible to verify whether similar strong emotions were also experienced by the non-representation group, as this group was not interviewed in this study. However, other researchers who are likely to have included participants who did and did not re-present have also reported that MI patients were shocked at experiencing a heart attack, although they did not measure anxiety (Astin et al., 2009, Wiles, 1998). The findings of both Wiles (1998) and Astin et al (2009) qualitative studies appeared to suggest that the strong emotions experienced shortly after STEMI are commonplace whether or not a patient goes on to experience symptoms and re-present (see sections 2.2.1 and 2.6.2.3 of the literature review relating these studies).

The current qualitative study also showed that almost all the participants feared the prospect of experiencing a further heart attack or cardiac event. These feelings of anxiety may have been related to the occurrence of IHD symptoms (leading to representation). The additional symptoms may have led to some of the cohort
believing that they had continuing disease, although symptoms may equally have been related to raised anxiety levels (see section 9.6.3.3 for further discussion).

Concerns related to continuing disease may have been exacerbated for individuals with a prior history of IHD. The baseline descriptive analysis showed that a higher proportion of the re-presentation group (21.1%) compared to the non-representation group (5.5%) underwent PCI prior to their heart attack ($p=0.002$). Following their previous PCI, patients may have believed that their condition was stable, so the occurrence of IHD symptoms at baseline due to STEMI may have lead to raised anxiety levels. However, prior PCI applied to less than a quarter of the re-presentation group therefore it is difficult to establish whether concerns regarding experiencing a further heart attack influenced the anxiety levels of the group.

Nevertheless, in Astin et al (2009) study, also involving PPCI patients, participants feared experiencing a further cardiac event. Whereas conversely in Wiles (1998) study only a minority of the participants were fearful of experiencing a further heart attack. This brings into question whether the difference in treatment and the patient journey, between PPCI and lysis, played a part in the views of participants in Wiles (1998) study compared to Astin et al (2009) and the current study. See section 9.6.3.5 for further discussion related to the role that PPCI may play in adaptation following STEMI.

9.6.3.2 Fear of death and dying

In the qualitative study, under theme 1 (fear of experiencing a further heart attack) some participants spoke of experiencing thoughts of death and fear of dying either at the time of their heart attack or shortly afterwards.

It has previously been reported that between 75.0% and 88.0% of Acute Coronary Syndrome (ACS) patients experienced either intense or moderate distress or fear of dying at the time of their acute event (Whitehead et al., 2005, Steptoe et al., 2011). This state of intense distress has been shown to share biological changes (such as an intense inflammatory response) that are present during an ACS event (Koukkunen et al., 2001, Steptoe et al., 2007). Intense inflammatory responses are measured using inflammatory bio-markers. See literature review section 2.4 for further details.

It has been suggested that such changes and potential associations may go some way to explain increased cardiac events and mortality in post STEMI patients with raised anxiety (Steptoe et al., 2011, Martens et al., 2010). Steptoe et al (2011)
investigated the potential association between heightened inflammatory bio-markers and intense psychological distress in 208 patients during an ACS event. The researchers found that those who reported intense distress also experienced significantly higher levels of inflammatory biological response (Steptoe et al., 2011).

However, a further study, 'the Heart and Soul study', extended the area of investigation to 1015 stable CHD outpatients followed up for over 5 years (Martens et al., 2010). The researchers investigated whether there was a potential association between general anxiety disorder (GAD), inflammatory bio-markers and hormones that influence the cardiovascular system and the occurrence of further ACS events. Furthermore, behavioural factors were also included, such as physical activity levels, adherence to medication, alcohol intake and smoking. The authors found that the risk of cardiovascular events was 1.62 times higher when GAD was present. However, no association was found between raised anxiety and inflammatory bio-markers (Martens et al., 2010). The studies conducted by Steptoe et al (2011) and Martens et al (2010) are both described in detail in section 2.4 of the literature review.

Thoughts of death and dying reported by some participants in the qualitative study certainly played a part in determining their psychological state early on after their STEMI. However, currently an association between ACS, psychological distress, biological response and the long term clinical consequences appears to be unclear.

9.6.3.3 Hypervigilance of symptoms

The qualitative study findings indicated that the interviewees experiencing heightened awareness and sensitivity to potential cardiac symptoms, as described in theme 1 ‘fear of experiencing a further heart attack’.

Heightened awareness and sensitivity of symptoms may have been related to hypervigilance of symptoms due to raised anxiety at baseline. Additionally, during the 6 month follow-up some of the 74 re-presentation events (due to potential IHD symptoms) may also have been due to hypervigilance of symptoms associated with underlying health anxiety.

Health anxiety is described as an intense anxiety about health and death (Wells and Hackmann, 1993). It constitutes a central feature of some anxiety disorders that are based on physical symptoms (somatoform) such as hypochondrias and panic disorder (Barsky et al., 2001, Furer et al., 2007). Hypervigilance is a known
manifestation of anxiety and it may commence following a stressful life event such as an episode of acute illness (Taylor and Asmundson, 2004). It involves the body remaining on alert to potential threat and is associated with increased arousal and high responsiveness to stimuli (Furer et al., 2007). Individuals experiencing hypervigilance also often suffer with panic attacks or panic disorder (Barsky et al., 2001). Panic disorder has previously been identified in 21.9% of CHD patients (Dammen et al., 2004). However, few studies have specifically reported hypervigilance of symptoms in post STEMI patients.

The current study findings suggest that heightened awareness of symptoms, and therefore possible hypervigilance, was an issue for the re-presentation group. It seems reasonable to assume that had participants in the re-presentation group experienced hypervigilance, they would be more likely to report increased angina frequency on the Seattle Angina Questionnaire (SAQ). The SAQ is a self-report measure and thus cannot differentiate between organic and other symptoms; it is reliant on the patient’s interpretation of their symptoms as organic cardiac ischaemia (angina). Hypervigilance related to symptoms involves an acute sensitivity and awareness of bodily symptoms. This may lead the individual to identify minor twinges as more serious symptoms such as angina, which in turn may be determined as increased frequency of angina (due to the inclusion of significant and minor symptoms). One may therefore expect participants suffering from heightened awareness to report symptoms as angina frequency on the SAQ.

The quantitative results suggested that symptoms and psychological health shared an association. Analysis for aim 5 showed that there was a moderate negative correlation between HADS anxiety and HADS depression scores, and SAQ angina frequency at baseline and 6 months for both groups. This indicated that as symptoms of anxiety and depression increased or decreased, angina frequency similarly increased or decreased.

Additionally in the re-presentation group anxiety and depression scores were mildly raised at baseline and 6 months (aim 2). Analysis for aim 3 showed that from baseline to 6 months there was also a non-significant increase in angina frequency for the re-presentation group. Conversely, for the non-representation group, anxiety and depression scores were within the normal range at baseline and both significantly improved at 6 months. Furthermore, angina frequency significant decreased at 6 months for the non-representation group. Analysis for aim 4 demonstrated that there was a significant difference in the change scores for angina frequency between the groups at 6 months.
Overall the descriptive study results for aims 2 to 4 support the findings of aim 5 that anxiety and depression were correlated with angina frequency. Likewise the findings for aims 2 to 5 support the results of the logistic regression model, that psychological health predicted re-presentation due to IHD symptoms at 6 months. It is not possible to determine through this study whether reduced psychological health was preceded by symptoms or vice-versa. However, less than a quarter (24.3%) of patients who re-presented in this study received a diagnosis related to cardiac ischaemia, further suggesting that anxiety and depression were intrinsically linked with symptoms. This implies that health anxiety and hypervigilance of symptoms may have played a part in some of the re-presentation events in this study.

9.6.3.4 Symptom attribution

In this study the qualitative findings indicated that the participants’ heightened awareness of symptoms (or hypervigilance) appeared to be translated into concerns regarding whether symptoms were cardiac related. The participants’ anxieties were often related to difficulty interpreting and determining the cause of symptoms (e.g. angina, indigestion, pulmonary). This was represented by theme 2 ‘uncertainty and inability to determine cause of symptoms’.

At the time of re-presentation the participants reported believing that they were experiencing a further heart attack. This was the central reason for re-presenting to acute services. However, the majority (56) of the 74 re-presentation events were not due to a further heart attack or even a cardiac ischaemic event and therefore could be classed as a ‘false alarm’ (see section 7.5). The combination of a ‘false alarm’ and confusion related to interpreting STEMI and re-presentation symptoms appeared to heighten participants’ feelings of uncertainty and anxiety.

Difficulty in interpreting symptoms for individuals who have had a heart attack is not an entirely new concept. In the qualitative study of Astin et al (2009), the participants’ main information needs concerned how they might recognise subsequent symptoms relating to a further cardiac event. Further reports show that experiencing a previous MI does not necessarily lead to patients acting on future symptoms in an appropriate manner (Ruston et al., 1998, Moser et al., 2006, Rucker et al., 2001). Moser et al (2006) found that previously experiencing a STEMI actually led to delaying seeking medical help during a subsequent STEMI. Similarly, in a qualitative study exploring STEMI patients’ responses to symptoms, Ruston et al (1998) found that almost half of the patients with a history of MI delayed seeking
help. The authors found that those who did delay had either no or little knowledge of the symptoms related to cardiac ischaemia. See section 2.11 of the literature review for more in-depth review of the literature related to symptom attribution.

Immediate onset and dramatic crushing chest symptoms are what is expected by patients as related to a heart attack (Alonzo, 1986). Symptoms that are of a more gradual onset are more challenging for patients to interpret (Moser et al., 2006). In the current qualitative study some individuals described feeling confused that their original heart attack symptoms were not stereotypical, for instance ‘not like you see on the telly’. In the Ruston et al (1998) study, participants also used stereotypical symptoms projected by the media to determine the seriousness of their own symptoms.

The mismatch between stereotypical symptoms and those experienced in ‘real life’ in some instances leads to patients implementing self-treatment or waiting to see how symptoms evolve. In the Ruston et al. (1998) study, some participants interpreted STEMI symptoms as indigestion and self-managed this accordingly before seeking further help when symptoms did not resolve. Similarly, in the current study at the time of re-presentation, some of the participants interpreted symptoms as indigestion and implemented self-treatment prior to seeking help. Following re-presentation, participants went on to adopt strategies to determining the cause of the symptoms when they occurred. This often included taking a combination of medications for both indigestion (ranitidine or omeprazole) and angina (GTN spray) concurrently.

In light of the challenges that individuals face regarding symptom attribution, it is not surprising that the participants of this study experienced anxiety and uncertainty relating to symptoms. On the whole participants found differentiating between symptoms challenging. There was a general need to understand and to learn to interpret symptoms when they occurred.

9.6.3.5 Severity and longevity of illness

According to the qualitative findings, the interviewees experienced some confusion and uncertainty in relation to the severity and longevity of their illness. Initially following PPCI participants reported a sense of elation and feeling well, which appeared to be associated with an absence of illness ceremony related to the PPCI patient journey. The participants’ subsequent experiences of debilitation during recovery seemed to be at odds with their initial feelings of wellbeing. Raised anxiety levels at baseline and the occurrence of symptoms during recovery also appeared...
to be implicated in their conflicting beliefs. These concepts were presented in theme 3 ‘insufficient opportunity to validate self-construction of illness’.

‘Illness ceremony’ in this thesis refers to the procedures, events and social rituals surrounding STEMI and treatment. Historically, a heart attack was viewed as having severe immediate and longer term health consequences (Reynell, 1975). The care of patients prior to the introduction of thrombolysis often included a hospital admission of several weeks (McNeer et al., 1975). Initially patients were nursed in a quiet calm environment (Pentacost et al., 1967) and were kept on strict bedrest for the first few days when they were often critically ill (Levine and Lown, 1952, Crampton, 1973). Visual clues of critical illness were often evident, such as an intravenous (IV) infusion, continuous blood pressure monitoring and the patient being attached to a cardiac monitor. All these events and procedures are likely to have lead to a sense of ceremony surrounding the patients’ illness and may have aided the patients’ ability to take on the sick role as described by Parsons (1951).

The sick role involves an individual being exempt from their normal role and responsibilities in society, together with an acceptance (by society) that the individual should seek medical help and receive care from others (Parsons, 1951). The level of concern and advice given by a physician to the patient and relatives provides some evidence of the severity of the illness (Segall, 1997). Furthermore, it is possible that a longer hospital admission is required for the sicker patient indicating the severity of the medical event.

It may be argued that the current PPCI patient journey challenges the historical view of the illness ceremony surrounding a heart attack. Treatment with PPCI brings about dramatic, almost immediate, improvement in the patient’s condition, with prompt resolution of pain and feelings of wellness (Astin et al., 2009). Patients also generally return to the coronary care unit with few visual clues (these may only include an IV infusion and a dressing on the wrist or groin). Furthermore, patients frequently only receive one or two visits during admission by the treating cardiologist and are usually discharged home within less than five days of STEMI.

During the current study some interviewees expressed surprise at how well they felt immediately after the PPCI procedure. In some cases individuals reported believing that they were well enough to return immediately to their normal daily activities. Moreover participants expressed shock at the speed of the treatment and at how soon they were discharged; this did not appear to be related to the timing of discharge (3 to 5 days post STEMI). Some participants reported that PPCI
treatment was ‘easy’ and one man went as far as saying that it was no worse than having a tooth out. The combination of feeling well, speed of treatment and early discharge seemed to contradict the patients’ prior beliefs regarding what to expect when experiencing a heart attack. The patients appeared to believe that they had been ‘fixed’ and that there were no long-term consequences to their heart attack. It is possible that the lack of illness ceremony played a part in establishing the participants’ views that their illness was not serious and of a short duration.

Similar findings have been reported by other researchers. In Astin et al (2009) mixed methods study of PPCI patients, the revised illness perception questionnaire (IPQ-R) (Moss-Morris et al., 2002) was used to measure the patients’ beliefs related to their illness. The authors reported that the participants viewed their illness as acute rather than chronic and PPCI as curative. Additionally the participants reported an abundance of energy early after STEMI and believed that the lack of physical evidence of a heart attack (e.g. absence of a scar) indicated less serious illness. Additionally, in the Sampson et al (2009) study some individuals disbelieved that they had experienced a heart attack and others likened it to a minor event such as ‘cutting a finger’. The authors also reported that patients spoke of feeling fixed following their PPCI treatment and believed that their heart trouble was a one-off (Sampson et al., 2009).

In contrast to the early feelings of wellbeing, participants in the current study spoke of experiencing fatigue, debilitation and physical limitation during recovery; these were often combined with potential IHD symptoms. In some instances this combination had a major impact on the participants’ usual activities of daily living, leading to consequences such as having to slow down or being unable to return to work despite the passage of more than 6 months since their heart attack. The feelings of fatigue and weakness may well have been related to reduced psychological health, a concept that has previously been reported in MI patients (McGowan et al., 2004, Wojciechowski et al., 2000).

Fatigue and debilitation were not specifically measured in the current study. However the qualitative study findings suggested that fatigue, symptoms and physical limitation were all inter-related. Interestingly a relationship was seen between psychological health and physical limitation due to angina symptoms (measured using the SAQ). Analysis for aim 5 showed that at 6 months there was a weak negative correlation between HADS depression and SAQ physical limitation scores for the re-presentation group and a strong negative correlation for the non-representation group. Analysis for aim 2 determined that both the re-presentation...
and non-representation group experienced moderately reduced SAQ physical limitation scores at baseline for both groups. At 6 months physical limitation remained the same for the re-presentation group compared to a significant improvement for the non-representation group.

These descriptive findings appear to suggest that at 6 months for the re-presentation group as psychological health remained the same so did levels of physical limitation. Whereas for the non-representation group as psychological health improved so did physical limitation. Similar correlations were seen for psychological health and angina frequency (see section 7.9.3). It is possible that when such a combination of symptoms, fatigue and reduced psychological health occurred, the participants’ view that they had been fixed was challenged. This in turn may have led to conflicting beliefs, possible confusion and reinforcement of anxiety in relation to health status.

Conflicting beliefs relating to physical health were particularly relevant for some of the younger men who were unable to accept that their physical strength or fitness may have changed due to having had a heart attack. This was often associated with experiencing symptoms and individuals reported feeling confused when symptoms happened. In some instances offers of help from family members to do tasks that they saw as ‘their role’ were viewed as a slight to their masculinity and were angrily rejected. Similarly, Astin et al (2008) found in their study that some of the men wanted to know how far they could push themselves. The need for a change in role was also difficult for women to accept in the current study, although women were more willing to agree to assistance from friends or relatives.

The occurrence of symptoms appeared to conflict with the participants’ beliefs that their illness was not serious. It is possible that additional to the lack of illness ceremony, experiencing fatigue and symptoms challenged the re-presentation group’s belief that they were fixed which may have led to a continuation of raised anxiety levels at 6 months.

**9.6.4 Difficulty with adjustment**

In this study the interviewees found adapting to life after a heart attack challenging. These findings were presented in theme 4 ‘adaptation to life after a heart attack’. Adaptations to lifestyle, beliefs and expectation relating to bodily symptoms are to be expected following a life threatening event (Radley, 1995, Heller et al., 1993).
Many of the issues faced by participants in this study related to the life consequences of having experienced a heart attack. These included practical and emotional factors such as lifestyle changes, financial issues and changes to work, family and social lives, as well as anxiety and depression. Additionally, grieving for loss of self and past life, symptom attribution and physical debilitation also appeared to play a part. The quantitative findings also demonstrated that the factors related to recovery were complex and included reduced psychological health, increased frequency of symptoms and diminished physical limitation, all of which can be related to quality of life (QoL).

9.6.4.1 Quality of life
Measuring adaptation in a quantitative way is somewhat difficult due to its multifaceted nature. However, assessing QoL offers some insight of the impact that a disease and its treatments has on someone’s life (Cella, 1994). For example an individual may perceive that lack of symptom occurrence and changes to their lifestyle (e.g. through stopping smoking, losing weight or increasing fitness levels) following an illness event lead to an improvement to their QoL. Conversely, individuals may no longer be able to undertake sports that they once enjoyed due to physical symptoms or fatigue and this may lead to reduced QoL (Hofer et al., 2005, Hirani et al., 2006, Pocock et al., 1996). Experiencing illness and making lifestyle changes may also bring about emotional constructs such as fear, embarrassment, anger, feeling down and loss of confidence (Charmaz, 1997, Bowman, 2001). Some of these emotional aspects of adaptation have also been shown to influence Health Related QoL (HR-QoL) in heart attack patients and other chronic illness groups (McGowan et al., 2011, Parashar et al., 2006, de Nooijer et al., 2001, Dickens et al., 2006). The definitions of QoL and HR-QoL can be found in section 2.10 of the literature review, where the literature related to these factors are discussed.

Dickens et al (2006) reported patients who reported symptoms of depression (measured using HADS) at baseline experienced decreased physical HRQoL (on the SF-36) at 6 months compared to those who were not depressed. Parashar et al (2006) reported that persistent depression (continuing from admission to one month) was associated with worse HRQoL (measured on the Seattle Angina Questionnaire (SAQ) QoL sub-scale). The authors also found that those with depression experienced more frequent angina and increased physical limitation (measured on the corresponding SAQ sub-scales) compared to those who were not depressed (Parashar et al., 2006).
Quality of life was also assessed in the current study using the SAQ QoL sub-scale, which measures how symptoms of angina influence QoL. Analysis for aim 2 showed that at baseline QoL was moderately reduced across both study groups, and possibly this was due to the initial difficulties associated with having experienced a heart attack. Interestingly, at 6 months QoL remained the same for the re-presentation group yet improved for the non-representation group. Analysis for aim 5 showed a strong negative correlation between anxiety, depression and QoL scores for both the re-presentation and non-representation groups at 6 months. This indicates that for the re-presentation group at 6 months their QoL remained moderately reduced as did their psychological health, whereas for the non-representation group there was an improvement in both their psychological health and QoL.

Furthermore as discussed in sections 9.6.3.3 and 9.6.3.5, psychological health, angina frequency and physical limitation were all associated at 6 months for both groups. Moreover, angina frequency, physical limitation and QoL were all positively correlated (i.e. as QoL improved or deteriorated the same was true of angina frequency and physical limitation). This demonstrated that psychological health, angina frequency and physical limitation were all associated with QoL for both groups at 6 months.

According to the quantitative results, reduced QoL at 6 months for the re-presentation group appeared to be related to reduced psychological health, increased angina frequency and reduced physical limitation. The qualitative study findings also suggested that psychological health, symptom attribution, fatigue and limited physical functioning were implicated with poor adjustment and recovery for the group. However there was also suggestion that lifestyle changes and elements of the PPCI patient journey including absence of illness ceremony also played a part (see section 9.6.3.5).

9.6.4.2 Cardiac rehabilitation

A further factor that may have been implicated in reduced QoL and poor adjustment for the re-presentation group was related to cardiac rehabilitation (CR) attendance. Attendance at CR is known to help patients to improve behaviours associated with increased risk of further coronary events, reduce levels of angina and improve QoL (Oldridge et al., 1988, O'Connor et al., 1989, Goel et al., 2011, Balady et al., 2007). It is also an opportunity to receive physical and psychological
assessment by a healthcare professional and to receive information and education (Hughes, 2011, Hedback et al., 2001, Heller et al., 1993).

In this study significantly fewer of the re-presentation group (35.1%) attended all the CR sessions compared to the non-representation group (56.8%). In addition, more of the re-presentation group attended either one (21.6%) or just a few (16.2%) sessions and decided not to attend further compared to the non-representation group (one session 7.4% and a few sessions 7.4%).

There are many possible reasons that the re-presentation group felt reluctant to attend all the CR sessions, the group may have believed that CR was not useful and did not fulfil their requirements, or the timing of CR may not have fitted with the patient journey or matched the patients’ needs due to their illness beliefs. It is also possible that the re-presentation group’s lower attendance rate at all CR sessions was due to being more symptomatic. In the qualitative interviews there was a general belief prior to receiving an invitation to participate that CR involved mainly performing physical exercises. This was given as a reason for non-attendance by some of the participants because they felt too debilitated to participate or they believed that CR was not useful and did not fulfil their requirements. Participants spoke of not receiving sufficient information or follow-up after discharge and it may be that they did not view CR as a source of information or follow-up. Others stated that it just wasn’t for them and some expressed the opinion that it was for ‘old people’.

A further possibility is that the way CR was delivered and much of its content (related to long term cardiovascular risk reduction), conflicted with the participants’ beliefs that they were cured or had purely experienced an acute event (as discussed in section 9.6.3.5). Certainly some of the younger men reported that the physical exercises were ‘too easy’ as they were physically fit before their heart attack and still believed that they were able to physically push themselves. Previous studies have shown that patients are more likely to attend CR when they believe that their illness is serious or controllable. A systematic review of CR attendance for coronary heart disease (CHD) patients reported that when patients do not identify with or deny the seriousness of their condition they are more likely to fail to attend CR (Cooper et al., 2002). Additionally a meta-analysis found that MI patients were more likely to attend CR when they had greater understanding of their condition, believed that their illness was controllable and had severe consequences, as well as those who were more symptomatic (French et al., 2006).
A number of researchers have investigated the illness beliefs or perceptions of MI and CHD patients based on the self-regulatory model (SRM) developed by Leventhal et al (1980) (Whitmarsh et al., 2003, Petrie et al., 2002, Hirani et al., 2006, Astin et al., 2009). According to the SRM, adherence to treatments and attendance at CR may be viewed as coping responses (Whitmarsh et al., 2003, Petrie et al., 2002). Folkman and Lazarus (1980) define coping as “the cognitive and behavioural efforts made to master, tolerate, or reduce external and internal demands and conflicts among them” when an individual experiences psychological stress.

Whitmarsh et al (2003) applied the SRM as a framework for their study investigating attendance at CR for a mixed cohort of CHD patients. A self-report instrument, the illness perception questionnaire (IPQ) (Weinman et al., 1996), which is based on concepts of the SRM, was used to measure the participants’ illness beliefs. Whitmarsh et al (2003) reported attendance at CR was less likely when participants perceived fewer symptoms, that their illness could be cured or controlled and when fewer problem-focused coping strategies were adopted.

In the current study the re-presentation group appeared to believe that their illness was of a short duration, and additionally symptoms (measured on the SAQ) at baseline (mean 19.4 days) seemed to be stable and infrequent. The mean time point for the first re-presentation occurrence was 76.5 days (11 weeks), which was later than the usual time point that CR starts (6 to 8 weeks). It is possible that the re-presentation group did not experience symptoms (that concerned them) until later in their recovery. They therefore may not have deemed it necessary to attend all CR sessions due to feeling well and lack of symptoms early after their heart attack. However, it is not possible to confirm or exclude this theory through the findings of the current study; this may therefore be an area worthy of further investigation.

A further explanation for non-attendance (47.2%) at all sessions for the total cohort is that the timing of CR may not have matched the patients’ needs or fitted with the patient journey. Currently when patients receive PPCI treatment early (within the first 4 hours of onset of pain) minimal temporary or long term damage of the myocardium occurs (Keeley et al., 2003). Prior to the revascularisation era, STEMI patients were more likely to suffer from temporary as well as permanent damage to their myocardium and it was recommended that patients start CR once clinically stable (Dennis, 1991). In the Manchester region, the timing of CR has followed these practices, which were put forward in the 1990s and generally CR has
been offered to patients between 6 and 8 weeks following STEMI. Despite the changes to STEMI treatment, including the clinical benefits of PPCI and alterations to the patient journey, this time point has remained unchanged.

The qualitative study findings alluded to the possibility that patients required earlier follow-up or the opportunity to attend CR sooner than is currently offered. The underlying raised anxiety levels for the group may require earlier follow-up to address concerns and initiate any necessary psychological interventions. It is also an opportunity to promote the benefits of attending CR and address any misconceptions regarding the longevity of illness. However the interviews only included participants who re-presented it may be that the non-representation group shared similar experiences and earlier follow-up may be of benefit to all PPCI patients.

9.6.4.3 Information

The information needs of participants were discussed in the qualitative interviews, and a mixture of views were reported. Some individuals were satisfied whereas others were dissatisfied with the level and content of information and follow-up. There were two main aspects of information that participants appeared to be dissatisfied with, including how to deal with stress and symptoms.

Some of those who attended cardiac rehabilitation (CR) spoke of a lack of information and detail on how to address stress or psychological problems. The amount and type of information delivered on this subject is mixed across the UK and is dependent on each local CR service (British Heart Foundation, 2011). However, anxiety and depression are routinely screened across the UK for all patients attending CR, although referral rates to psychological services from CR are poor (Hughes, 2011, National Service Framework, 2000). It is possible that the lack of illness ceremony related to PPCI combined with raised anxiety levels at baseline for the re-presentation group led to the requirement for additional information on coping with feelings of anxiety and stress.

There was also a sense of dissatisfaction regarding the limited amount of information that appeared to be available relating to symptoms, both during admission and follow-up. Participants expressed a need for information related to symptom attribution, potential treatments for symptoms and what further actions to take when symptoms occur. It is likely that those patients who did not attend any or all of CR received less information or education relating to symptoms. Early discharge associated with PPCI may also have limited the opportunity for patients.
to receive information. The majority of participants (including CR attendees and non-attendees) did not recall having received any information on identifying and dealing with symptoms. It is difficult to determine whether this was due to a lack of information being delivered or due to poor recall on the behalf of the participants.

In a previous qualitative study conducted by Astin et al. (2008) to establish the information requirement of PPCI patients, participants received information relating to what to do in case of developing further chest pain. However, interestingly, recall of information was varied. When symptoms occurred some individuals were able to repeat the actions advised and others were unclear about what actions to take. The authors identified that the emotional shock at having had a heart attack and the speed of treatment appeared to be a barrier to absorbing information for some individuals particularly during admission (Astin et al., 2008). Other researchers have also attributed poor recall of information to reduced psychological health (Kizilbash et al., 2002, Mobini and Grant, 2007). It is possible that some of the interviewees in the current study exhibited poor recall due to psychological sequelae.

9.7 Evaluation of study methods

Overall the mixed methods study design worked well and enabled the research questions to be answered. The qualitative interviews added depth to the study findings and allowed exploration of the concepts associated with re-presentation. This section of the thesis contains a reflective discussion of the methods used and the adaptations that were made to address the issues that developed during that study.

9.7.1 Patient recruitment

Overall patient selection and consenting techniques were successful. This was partly achieved due to the author’s in-depth knowledge (as a current practitioner) of the STEMI and PPCI population. Additionally, as a practitioner she had a comprehensive understanding of the clinical background of the disease process and PPCI treatment. This also helped in identifying the most appropriate and effective strategies to identify and recruit patients. Additionally her knowledge of running large research studies (due to her role as a research nurse) was invaluable in adopting efficient processes leading to good response rates and effective participant retention.
However, there were some issues surrounding the length of time taken to reach the recruitment target and adaptations were made to address the matter. The acceptance rate for participation in the study was good; of the 274 patients who were screened during the recruitment phase 231 (84.3%) gave consent. The recruitment of participants was much slower than originally predicted. It was necessary to extend the planned 12 month recruitment phase to 20 months. This was partly due to the slower development of the PPCI service than was initially predicted. The expansion of the service to 24 hour, 7 days a week was delayed for 6 months resulting in the reduced availability of PPCI patients across the Manchester conurbation. There were also difficulties in accessing the names and details of PPCI patients at one of the PPCI centres (Trust 2), resulting in only 46 patients being recruited from this centre compared to 156 from the other PPCI centre (Trust 1).

Prior to the start of the study approval was gained through the Local Research Ethics Committee (LREC) and the Research and Development (R&D) Departments at both PPCI sites to access PPCI databases to enable identification of potential participants. Relationships were also developed with the practitioners working with the PPCI patients at Trust 2 and initially they were supportive in allowing access and took an active part in identifying potential study patients. However, due to a change of staff during the study at Trust 2, the matter was raised as an issue and access was denied for a period of time. The issue returned to the R&D Department for further consideration and following reassurances from the researcher access to patient identifiable data was renewed.

The recruitment strategy for the qualitative study participants was highly successful with all but three patients who were approached agreeing to participate (90.3% response rate). Success was mainly due to the use of the explanatory mixed methods study design, which enabled the purposeful selection of the interviewees. Patients were very willing to participate and the 6 month telephone calls designed to collect the re-presentation data, was the main method used to identify participants who were likely to be ‘rich’ data sources for the qualitative interviews. Sufficient in depth qualitative data was collected and data saturation was achieved.

**9.7.2 Data collection**

Data collection methods were generally successful and response rates to questionnaires were good. However, the main challenge of the study design related to the collection of re-presentation data. Despite the use of multiple data collection
techniques and several sources of data, verifying whether a re-presentation had occurred and establishing the discharge diagnosis was difficult.

9.7.2.1 Questionnaires

The response rate was good at baseline with 87.5% of participants returning their questionnaires. Additionally at 6 months the response rate was also good with 91.1% of questionnaires returned. A strategy that helped to improve response rates was the use of a follow-up phone call to participants within one week of mailing the questionnaires. This doubly acted as a reminder for patients to complete the questionnaires and to help them to do so if they should require assistance. Additionally when patients missed answering questions they were telephoned to collect the missing answers.

The initial face to face recruitment (in most cases) and follow-up telephone calls helped to develop and maintain a good relationship with the participants, which helped with participant retention. During the phone calls participants verbalised their appreciation for not only the initial treatment that they received but also being contacted by the researcher (a trained cardiac nurse). They saw it as an opportunity to check out their concerns and saw it as an additional follow-up despite the fact that this was not the purpose of the call.

9.7.2.2 Verifying re-presentation events

Re-presentation data were collected by telephoning participants at 6 months, and the completion of a diary card (by participants). Additionally medical records were checked for re-presentation events for all the participants. This included records at both the participants’ local hospital and at the PPCI centre.

The phone calls and diaries were varied in their success at capturing the data. During the 6 month phone calls some individuals had poor recall of re-presentation events and in some cases the dates and details of the events did not match those recorded in the medical records. The diary card completion also had a mixed response from participants. Some individuals diligently recorded more than was required, including GP visits, outpatient attendances as well all acute re-presentations. Other individuals did not complete the diary at all and discarded it soon after receiving it.

During the planning of the study, contacting the participant’s GP was a further means of collecting data that was considered. This was disregarded due to the limited time and resources that were available. However, following discussion with
the supervisory team, it was decided to adapt the data capture methods by incorporating contacting GPs in certain circumstances. This included when insufficient data were present in the medical records to verify a re-presentation event or if a participant suggested during the phone call that they had attended a hospital other than their local one or the PPCI centre. To implement this change to the study procedures it was necessary to seek further approval from the Local Research Ethics Committee (LREC).

Three participants (study numbers 34, 149 and 419) all reported during the 6 month telephone interview that they had re-presented to hospitals other than their local one or PPCI centre. Despite contacting the participants’ GPs it was not possible to verify the re-presentation events; this was due to lack of communication with the GPs by the treating hospital. As it was not possible to obtain details of the event from the hospital or GP records the participants were classified as a non-representer in the quantitative study. During the 6 month telephone interviews all three participants had good recall of the re-presentation event and were able to give rich accounts of the re-presentation events. It was therefore decided to include these individuals in the interview sample.

9.7.2.3 Establishing a diagnosis

Establishing a discharge diagnosis for each of the re-presentation events in this study was difficult. Review of participant medical records often did not supply a definitive diagnosis. If a diagnosis was not recorded in the medical records, data relating to the admission diagnosis as well as the results of investigations performed were collected and reviewed by the researcher in consultation with the supervisory team. When the diagnosis remained uncertain a consultant cardiologist was asked to give their expert opinion on the most likely cause of re-presentation. However, a diagnosis remained indeterminate for a large proportion of re-presentation events.

The difficulty in obtaining the discharge diagnosis was due to the use of retrospective data recorded in the medical records. It is possible that if the data had been collected prospectively the research process may have prompted the treating doctor at the time of the re-presentation event to establish a diagnosis. However, although the retrospective design was a disadvantage in respect of the reduced number of events being categorised, it was also an advantage as it highlighted the large proportion of patients who were discharged without receiving a diagnosis. This offered a valuable insight into the ‘real world’ experience of
patients and was supported by the findings of the qualitative interviews. During the interviews some of the participants spoke of feeling confused about the cause of their symptoms at the time of re-presentation.

9.8 Study strengths and limitations

In this section of the thesis the limitations and strengths of the current study are discussed.

9.8.1 Limitations

The main limitations of this study related to the small number of re-presentations, the potential under estimation of re-presentation events and the large number of indeterminate discharge diagnoses. Additionally, solely interviewing participants who re-presented was also a potential limitation.

9.8.1.1 Re-presentation events

The small number of individuals who were identified as re-presenting to acute services with potential IHD symptoms limited the number of variables that could be included in the logistic regression model.

Through the development of the conceptual model, 11 variables were identified as theoretically justified for inclusion in the logistic regression model. However, according to Peduzzi et al (1996), the ratio of individuals to predictor variables should be at least 10:1; having almost 40 re-presenters suggested including 4 variables in the model in this study. In contrast Vittinghoff and McCulloch (2007) suggest a compromise between obeying the rule of Peduzzi et al (1996) strictly and the inclusion of the most important variables from the conceptual model (see section 5.23.1). As this study was an exploratory study, and in line with the recommendations of Vittinghoff and McCulloch (2007), only the theoretically most important variables (seven variables) were ultimately included in the model.

The additional variables not included in the model were history of a previous IHD event, further revascularisation, gender and social support (see section 3.5). Further relationships with re-presentation may have been determined had it been possible to include the remaining variables in the model.

The limited size of the re-presentation group may also have influenced the stability of the final logistic regression model. Anxiety was chosen on theoretical grounds as the main predictor in the final model. However, the overall fit of the model was
non-significant, requiring the adjusted odds ratio and the significance that anxiety plays in predicting re-presentation to be viewed with caution.

Additionally the small size of the re-presentation group may have affected the external validity of the study. This is because the small sample may not be representative of those who re-present and some bias may have been introduced. However, all possible attempts were made to identify those who re-presented with potential IHD symptoms. Additionally a cross section of patients was recruited consecutively from six different Hospital Trusts in an attempt to be as inclusive as possible and limit bias.

9.8.1.2 Under-estimation of events

One reason for the small re-presentation group may have been related to an underestimation of events due to the difficulties involved in collecting the re-presentation data (see section 9.7.2.2). It is possible that patients who re-presented were missed partly because of the use of retrospective data collection to capture events. An attempt was made to collect data prospectively using diary cards with mixed success (see section 9.7.2.2). Alternative methods of prospective data collection would have necessitated the involvement, training and agreement of medical staff in six A&E departments. This would have made the study unmanageable. Medical records were the most reliable and effective means of checking the events reported by participants during the 6 month telephone calls. Retrospective data collection was therefore an inevitable weakness of the study.

Additionally the only means of becoming aware of individuals who re-presented to other hospitals was through participants notifying the researcher (see section 9.7.2.2). It is therefore possible that there were additional individuals who re-presented to other hospitals.

A further reason for the small number of re-presentations in this study may have been due to the six month follow-up time point. A longer follow-up (i.e. 12 months) most certainly would have increased the size of the re-presentation group. However, based on previous studies, 6 months was chosen because it was believed that sufficient re-presentation events would be identified to allow inclusion of all the potential covariates in the logistic regression model (see section 5.7.3). Furthermore, the length of time available for follow-up was confined due to the timeline of the academic qualification.
9.8.1.3 **Indeterminate discharge diagnosis**

A further limitation of this study relates to the large number of discharge diagnoses categorised as indeterminate. The design of this study was limited to collecting the investigations conducted purely during the acute event and did not include a diagnosis made post discharge. It is possible that some of those who did not receive a diagnosis or were classified with an ‘indeterminate diagnosis’ may have gone on to receive further investigations post discharge. They may have been followed up at a cardiology outpatient clinic and received further cardiac investigations leading to a diagnosis. Furthermore, those individuals who were told ‘it’s not cardiac’ may have been referred on by their GP to another specialist such as a gastroenterologist or chest physician for further consultation and investigations. It is therefore possible that there was an underestimation of cardiac events in this study as the final diagnosis related to the re-presentation event may have taken some weeks or months to have finally been determined.

9.8.1.4 **Interviewing only re-presenters**

An additional limitation was the sole inclusion in the interviews of patients who re-presented. Although this fulfilled the study purpose, it is possible that those who did not re-present had similar experiences to those in the re-presentation group. This could not be highlighted due to the exclusion of PPCI patients in the non-re-presentation group. It is therefore difficult to say how the re-presentation group differ in terms of their experiences of symptoms and adaptation to the non-re-presentation group. The additional viewpoints of this group would have elicited helpful comparisons between the two groups and aided interpretation of the quantitative results.

### 9.8.2 Study strengths

The main study strengths included the use of a conceptual framework and the use of mixed methods. Additionally, the study cohort was representative of a general PPCI population. Furthermore, the response rates and participant retention was good in this study and the percentage of missing data was low.

9.8.2.1 **Conceptual framework**

A major strength of this study was the use of a conceptual framework leading to a sound theoretical base. The framework worked well in terms of guiding the development of the study and led to identification of the study aims, the study methods and the most appropriate variables to be included in the logistic regression model. Additionally the main study finding, that anxiety was the main predictor of
re-presentation, was supported within the conceptual framework. See section 9.4 for further discussion regarding the evaluation of the conceptual framework.

9.8.2.2 Study design
A further strength of the study was the mixed methods design which combined both quantitative and qualitative investigation of re-presentation due to re-presentation. The quantitative phase of the study enabled the study aims to be successfully addressed, including rates of re-presentation to be determined. Additionally, it was confirmed that psychological factors were associated with re-presentation. The qualitative phase of the study assured depth and richness of understanding in relation to re-presentation which would not have been possible through quantitative methods alone.

Synthesis of the quantitative and qualitative findings offered additional insight in relation to some of the underlying reasons that the re-presentation group experienced psychological distress. Moreover, the qualitative study highlighted the complexity of issues faced by the participants who re-presented with potential IHD symptoms.

9.8.2.3 The study cohort
In the current study the intention was to include a cohort that was representative of a general PPCI population. Comparison with other studies (see Table 9-1) demonstrates that this aim was fulfilled and the study cohort was similar to other representative study samples. This strengthens the ability to generalise to further PPCI patients. Furthermore, the internal and external validity of this study were strengthened due to low levels of missing data, good response rates and retention of participants.

9.8.2.4 New ideas and areas for research
A further advantage of this exploratory study was the emergence of a large number of ideas for future investigation. The findings of this study are likely to lead to further exploration of the psychological health of PPCI patients and the relationship with symptoms.

9.9 Further research
Through the conduct of this study a number of further research areas have been highlighted and these are discussed in the following section.
9.9.1 Associations with re-presentation at 12 months

The small proportion of re-presentations in this study limited the number of potential covariates that were included in the logistic regression model. Although a different research question, the use of 12 month follow-up data may have led to a larger re-presentation group and the inclusion of more variables in the model (see section 9.8).

Following on from the current study 12 month re-presentation and questionnaire data has subsequently been collected. This offers the opportunity to conduct further logistic regression analysis, potentially including all the planned covariates. It is expected that the additional 12 month analysis will build on the initial 6 month study findings.

9.9.2 Psychological health

The 12 month data also offer an opportunity to review the progression of anxiety and depression for both PPCI patients who do and do not re-present, as currently the time course of anxiety and depression for PPCI patients is unclear. To establish the temporal relationship of anxiety and depression following STEMI, measurement of psychological health at only 6 and 12 months may not offer sufficient in-depth understanding of the phenomena.

Theoretically the collection of anxiety and depression data on a monthly basis would be ideal in highlighting the temporal relationship. Inevitably, it would not be feasible to collect self-report psychological health data so frequently, as managing such a study and maintaining response rates would be extremely difficult and costly. However, a future study including levels of anxiety and depression at 3 months may be beneficial. Currently, an optimum time point for collection of psychological health data is not defined in the literature. Further information related to the timelines involved with anxiety and depression and those who re-present may help practitioners to target appropriate services to address patient need.

9.9.3 Establishing a final diagnosis

It would be of value to further investigate the re-presentation group who were categorised with an ‘indeterminate’ discharge diagnosis, to determine whether they received further outpatient investigations, or were reviewed by their GP, or did not receive further review or follow-up. Additional data collection related to further investigations, referral and subsequent treatments would be required for the
current study cohort with the use of hospital and GP records. This would help to identify the final re-presentation diagnosis (i.e. whether cardiac, gastric, musculoskeletal or psychologically related).

9.9.4 Symptom attribution and primary care

The current study only included individuals who had sought help for their symptoms via acute services and did not incorporate those who attended their GP regarding symptom occurrence. An investigation of the proportion of post PPCI patients who visit their GP when experiencing potential IHD symptoms may be of benefit. This would necessitate the checking of GP records for a cohort of PPCI patients and would potentially be a costly study.

However, a comparison of demographic, physiological and psychological factors between those who present to acute services and those who attend their GP may highlight important differences. Furthermore, the use of qualitative interviews to explore the experiences of post PPCI patients who seek help from their GP may elicit important information relating to their understanding of symptoms and how they address them. The interviews may also be enlightening in terms of how the patients adjust to life following STEMI and PPCI and whether their recovery differs from patients who re-present to A&E.

9.9.5 Psychological interventions

Previous studies investigating pharmacological and non-pharmacological treatments for depression in post STEMI patients have shown mixed results. Pharmacological treatment with selective serotonin reuptake inhibitors (SSRI) used in the Sertraline Anti Depressant Heart Attack Randomised Trial (SADHART) led to a significant reduction in levels of depression out to 16 weeks post MI (Shapiro et al., 1999). However, there was not a significant improvement in morbidity and mortality for depressed patients (Shapiro et al., 1999). Similarly in the ENRICHD study at 6 month follow-up both depression and levels of social support improved with the use of cognitive behavioural therapy (CBT) compared to usual care. This study only included post MI patients who were identified as either depressed or with low perceived social support. At 29 months there was no significant difference in mortality rates between the groups receiving CBT or usual care (ENRICHD Investigators, 2003). Additionally in an interventional study comparing usual care with a home based nursing education and psychological support programme, post MI patients did not benefit in terms of improvement in psychological health or mortality (Frasure-Smith et al., 1997).
Presently there is little evidence available relating to the use of psychological interventions in STEMI patients who have received PPCI. However, through the Collaboration for Leadership in Applied Health Research and Care (CLAHRC) for Greater Manchester a study is being conducted to detect and manage depression in patients with CHD and diabetes (Gask et al., 2011). Part of the study involves training Psychological Well-being Practitioners (PWPs) to deliver counselling to CHD and diabetic patients with mild to moderate depression; PWPs are already employed by the NHS to deliver counselling to individuals with depression who do not have serious physical illnesses. The future benefits of such a programme may well be of value to PPCI patients experiencing psychological distress, although it may require further evaluation for PPCI patients.

Further psychological interventions that may be appropriate and worthy of investigation for PPCI patients involve addressing stress during recovery. The participants in this study spoke of not receiving sufficient support or information on how to deal with stress following their heart attack. They voiced the wish to receive guidance on methods to aide relaxation and promote stress relief. Mindfulness meditation is a concept that has been investigated in other illness groups (Speca et al., 2000, Bohlmeijer et al., 2010) and is currently being investigated in other cardiac groups (Dickens, personal communication October 2011) to aide psychological adaptation. This may be a technique that is of value to PPCI patients and is worthy of investigation.

9.9.6 Adapting follow-up and cardiac rehabilitation

Individuals in the qualitative study reported that there was insufficient opportunity to talk to healthcare professionals about their heart attack and treatment. The majority of participants found the interview process cathartic and were grateful for having the opportunity to speak about their experiences. It is possible that an early opportunity to access follow-up care and cardiac rehabilitation (CR) may be of benefit to patients.

Patient illness perceptions, based on Leventhal et al (1980) self-regulatory model (SRM), have been shown to influence adaptation in terms of return to work, symptom attribution and attendance at CR, following MI (Whitmarsh et al., 2003, Petrie et al., 2002, Hirani et al., 2006, Petrie et al., 1996). Petrie et al (1996) found that illness beliefs assessed early following an MI influenced the patients’ recovery. Individuals who perceived their MI to have serious lasting consequences experienced more illness related disability and were slower to return to work.
Patients who believed in less control of their illness, or that a cure was unlikely, were less likely to attend CR (Petrie et al., 1996). It may therefore be beneficial to conduct investigations of early follow-up and adapted CR using the SRM as a framework.

9.9.6.1 Follow-up

In Astin et al’s (2008) qualitative study, the majority of PPCI participants received a home visit from the CR nurse early following discharge, and uncertainty was experienced by participants who did not receive a visit. In the Manchester region home visits soon after discharge are not common practice for STEMI patients. Implementing a home visit as part of routine care may lead to unacceptable additional financial costs. However, potentially a more financial viable option would be to implement a nurse led PPCI follow-up clinic within one or two weeks of discharge.

The purpose of the clinic would be to improve the patients’ access to a healthcare professional soon after discharge. This would offer the patients an opportunity to talk through their experiences, to receive education, information and advice. An early psychological assessment may also be performed to identify patients with raised psychological scores, with the benefit of then targeting vulnerable individuals with psychological interventions. Additionally, the patients’ illness perceptions may be evaluated at baseline and again at 3 and 6 months, to ascertain whether such a clinic leads to positive illness perceptions. Conducting a cost analysis would also demonstrate whether the upfront costs of setting up the service are offset due to cost-savings associated with lower re-presentation rates. An intervention such as a follow-up clinic may be worthy of exploration initially through a pilot study and subsequently through a randomised controlled trial.

9.9.6.2 Cardiac rehabilitation

The qualitative findings of this study suggest that cardiac rehabilitation (CR) did not meet the needs of the entire re-presentation group (see section 9.6.4.2). Some of the patients who chose not to attend outpatient CR believed that the programme mainly comprised of exercises. Early delivery of information relating to what CR comprises of (i.e. information and education) may be of benefit.

It may also be necessary to evaluate whether the level of exercises delivered at CR do reflect the current clinical picture for PPCI patients. Some of the patients reported that the exercises were too easy and it is possible that this is a true
reflection, as clinically many PPCI patients experience very few short or long term physical consequences of their STEMI. However, it is also possible that the patients’ illness perception ‘that they are fixed’ leads them to believe that they are physically able of doing more than clinically advisable. A study evaluating the patients’ physical capabilities and their illness perceptions may be of value.

It may be that offering CR earlier in the patients’ recovery may lead to more positive illness perceptions. The current time point of 6 to 8 weeks may be inappropriate as negative illness perceptions may already have been formed. The delivery of information and education during CR appears to be an excellent opportunity to assist patients in formulating positive illness perception. Additionally, conducting CR sooner during recovery also would lead to earlier assessment of psychological health which is undertaken routinely during CR across the Manchester region. However, availability of psychological interventions at CR is generally poor and a study to investigate delivery of psychological interventions as part of CR may also be of value.

9.9.7 Education, information delivery and the affect on symptom attribution

Many of the individuals who participated in the interviews spoke of not having received education or advice on how to deal with symptoms. It is not clear whether they had received this information and could not recall it or whether it had not been delivered.

A study to investigate the delivery of an education programme relating to symptom attribution and symptom management for patients could be of benefit. This may include differing methods of information delivery (including verbal and visual) at different time points. Moreover, the evaluation of additional telephone support (run by a trained cardiac nurse) to help patients with addressing symptoms may be beneficial. However, a Cochrane review included 33 studies related to assessing the effectiveness of telephone follow-up (TFU) to address post discharge problems after hospital discharge for a variety of diseases (Mistiaen, 2008). The review showed that although TFU was not detrimental it was also not shown to be an effective intervention. The authors reported that the majority of studies conducted relating to TFU were of poor methodological quality and consequently the results of the review should be viewed with caution. It is therefore possible that a study undertaken with a strong methodology may elicit valuable findings regarding TFU in post MI patients.
Further research examining information delivery relating to symptom attribution may also lead to practitioners delivering information in a way that builds the patients’ confidence in addressing symptoms. This may help patients in their decision making regarding when to seek help. Dracup et al (1997) found in their study investigating delayed help seeking following the onset of MI symptoms, that participants were more likely to delay if they waited to see if symptoms resolved or if they did not recognise symptoms as important. In a subsequent randomised controlled trial the authors found that an education and counselling intervention to help CHD patients in appropriately addressing ACS symptoms, did not improve delay in seeking help (Dracup et al., 2009). However, the authors did find that at three and 12 months following the intervention, the experimental group experienced lower levels of anxiety compared to the control group (Moser et al., 2010). Clearly symptom attribution is an important area worthy of further investigation.

9.10 Implications for clinical practice and policy

9.10.1 Clinical Practice

The findings of this study demonstrate that raised anxiety levels may be predictive of re-presentation due to potential IHD symptoms following STEMI and PPCI. This demonstrates the importance of assessing anxiety levels early after STEMI and the potential need to initiate psychological interventions for those identified with raised anxiety. Further research is required to establish the most effective psychological intervention and whether a reduction in re-presentation events can be achieved through such implementation.

Furthermore, the participants in this study reported uncertainty and confusion relating to symptom attribution. There was a lack of participant recall regarding information delivery relating to symptoms in the study. This revealed a clear need for unambiguous and repeated information delivery regarding symptom identification during recovery following a heart attack.

The implementation of psychological care coupled with the delivery of education and support regarding symptom attribution may go some way to reduce inappropriate non-cardiac re-presentations. It may also aid patients to evaluate their symptoms and seek help through the most suitable services, within an appropriate timeframe when symptoms do occur. However, it is important to acknowledge that the Dracup et al (2009) nursing intervention study, which
involved ACS patients receiving education and support to identify cardiac ischaemic symptoms, failed to reduce the time it took for patients to seek help when further symptoms occurred. It is clear that this area of clinical practice requires additional research and evaluation prior to changes in patient care (see section 9.9.7).

The study findings indicated that a large proportion of participants either did not receive a diagnosis or had an indeterminate discharge diagnosis following their re-presentation event. The qualitative study findings indicated that participants found the lack of a definitive diagnosis anxiety provoking, leading to confusion and difficulty in making sense of the re-presentation event. This indicates to practitioners the value of communicating a clear diagnosis (whenever possible) to patients following a potential IHD event. It may be necessary to evaluate the training and education needs of healthcare practitioners in delivering such information in a clear concise manner to patients.

**9.10.2 Policy**

The current study did not set out to identify areas for policy change, as it was an exploratory study. However, the findings of the study did identify that those who re-presented experienced symptoms of anxiety. It may therefore be of benefit to identify patients with heightened symptoms of anxiety early during their admission with the potential of implementing psychological interventions, although this change would require further investigation and evaluation (see section 9.9.5).

**9.11 Conclusion**

The analysis related to the main aim of this exploratory mixed methods study established that 18.8% of patients (95% CI 14.0% to 24.8%) re-presented to acute healthcare services with potential IHD symptoms within 6 months of STEMI. Furthermore, the study showed that psychological health was predictive of representation at 6 months. In particular anxiety was most closely associated with representation and appeared to be implicated in the patients’ experiences of potential IHD symptoms.

The conceptual model included psychological health, physiological health and symptoms, as directly related to re-presentation, whilst sociodemographic factors were deemed to be indirectly related. Theoretically the model appeared to be justifiable with respect to re-presentation (due to potential IHD symptoms) at 6 months following STEMI, although further empirical evaluation is required.
A number of potential influencing factors related to reduced psychological health and re-presentation were identified in this study. The re-presentation group appeared to experience shock at having had a heart attack as well as fear of death and dying. Moreover, hypervigilance of symptoms and difficulty with symptom attribution appeared to play a role in the raised anxiety levels at baseline for the re-presentation group. Confusion in relation to severity and longevity of illness and perhaps a potential pre-disposition to poor psychological health may also have been implicated in the reduced psychological health of the group.

Adaptation during follow-up was challenging for the re-presentation group. There was some suggestion that the PPCI patient journey played a part in the difficulties that participants experienced. Implicated in this was the reduced attendance rates at cardiac rehabilitation (CR) for the re-presentation group, which appeared to be related to some of the patients’ beliefs that their illness was short lived. Changes to CR and post STEMI follow-up have not generally kept pace with those related to the patients’ treatment (i.e. PPCI). Therefore further exploration of potential adaptations to the CR service and post PPCI follow-up are worthy of further investigation. Additionally this study has identified a number of further areas requiring investigation, with potential implications for changes to policy and clinical practice.
Appendix A Literature Review Methodology

A.1 Literature review methods

The methods undertaken to conduct the literature review are described in this section of the thesis. The purpose of the review, the review question, the inclusion and exclusion criteria as well as the review strategy are defined.

A.1.1 Purpose of literature review

The literature review has been undertaken to:

1. Understand the clinical and psychological factors (specifically anxiety and depression) associated with re-presentation (due to potential Ischaemic Heart Disease (IHD) symptoms) during recovery following ST-elevation myocardial infarction (STEMI) and primary percutaneous coronary intervention (PPCI).
2. Understand the patient journey in relation to PPCI and STEMI, including patients experiences of PPCI, symptoms and follow-up (including cardiac rehabilitation), as well as receiving information and education.
3. Identify past and current knowledge of anxiety, depression and re-presentation due to potential IHD symptoms in STEMI and PPCI patients.
4. Identify gaps in the literature relating to anxiety, depression and re-presentation (due to potential IHD symptoms) in STEMI and PPCI patients.

A.1.2 Literature review question

The key question related to this literature review is:

- What are the clinical and psychological factors related to re-presentation due to potential Ischaemic Heart Disease (IHD) symptoms following STEMI and PPCI?

A.1.3 Search strategy

The relevant literature underpinning this study was fragmented in terms of subject area and study methodologies (quantitative and qualitative studies) and it was therefore not possible to conduct a formal systematic review (SR). However, the main principles of a SR were followed (i.e. clear purpose of the review, thorough and complete searches, and critically appraisal of studies) (Centre for Reviews and
Dissemination (CRD), 2009). This was to ensure a comprehensive review was completed and quality studies were included.

A.1.3.1 Measures used to identify relevant literature

Electronic databases, scanning reference lists of relevant studies, hand searching, citation links and searching using the names of authors with a number of publications in a subject area, were all incorporated in identifying relevant literature. The electronic databases used were:

- Medline
- CINAHL
- EMBASE
- PsychINFO
- British Nursing Index
- ASSIA

Abstracts were reviewed for relevance and then the full paper was obtained. Searches were conducted between 1982 and 2011. Early work (1982 to 1990) was included because during this time a relationship between CHD and psychological sequelae was identified. Literature pertaining to the physiological and clinical aspects of PPCI, have been produced much more recently (1999 to 2011).

A.1.3.2 Inclusion Criteria

- Adult studies.
- Coronary heart disease (CHD) studies including STEMI, non ST-elevation myocardial infarction (N-STEMI) and acute coronary syndrome (ACS).
- Treatments for CHD; Primary Percutaneous Coronary Intervention (PPCI), Percutaneous Coronary Intervention (PCI), thrombolytic therapy (lysis), Coronary Arterial Bypass Graft (CABG).
- Hospitalisation, re-presentation and readmission for CHD groups.
- Papers describing psychological studies involving anxiety, depression and general distress in CHD patients.
- Patient experiences of PPCI, chest pain and potential ischaemic heart disease (IHD) symptoms and additionally experiences of education and follow-up (including cardiac rehabilitation).
- Only literature written in English was included due to difficulty obtaining, and the cost associated with translation.
- Randomised controlled trials (RCTs), controlled trials, cohort studies and qualitative studies conducted in any healthcare setting were all included.

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- Papers relating to human studies.

A.1.3.3 Exclusion Criteria
- Non-English papers.
- Laboratory studies.
- Cardiac rehabilitation studies which incorporated only physical (exercise, dietary and smoking cessation studies) rather than psychological variables.
- Papers relating to heart failure studies.

A.2 The findings of the literature search

The literature review identified that there is a dearth of literature relating to the psychological health of PPCI patients. Generally, apart from clinical studies, PPCI treatment is under-represented in the literature. Re-presentation data for this group are limited to death and cardiac or cardiovascular complications, and these data are presented in the clinical studies. No publications were identified showing all cause re-presentation, hospitalisation or re-admission for PPCI patients. Only one abstract was found relating to anxiety and depression in PPCI patients. However a great number of studies covering anxiety and depression were found for STEMI patients, most of which were conducted in the 1990s when patients received either medical or thrombolysis therapy as first line treatment.

A number of systematic reviews were found relating to both treatment and psychological health. The majority of studies relating to the physical aspect of STEMI and PPCI were randomised controlled trials (RCTs) conducted in a clinical setting, whereas most of the psychological publications were cohort studies. Overall there was also a lack of qualitative studies in the STEMI population, although several qualitative studies directly related to PPCI patients were found.
## Appendix B LITERATURE REVIEW TABLES

### B.1 Clinical studies demonstrating event/re-presentation rates for PPCI and PCI cohorts

<table>
<thead>
<tr>
<th>Authors</th>
<th>Cohort</th>
<th>Study type</th>
<th>Follow-up time point</th>
<th>Event rate results at follow-up (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mehran et al (2009) (HORIZONS study)</td>
<td>ØPPCI 1802</td>
<td>RCT</td>
<td>12 months</td>
<td>11.9 MACE (death, stroke, MI and re-vascularisation) 3.8 cardiac death</td>
</tr>
<tr>
<td>Lambert et al (2010)</td>
<td>≈PPCI 1440</td>
<td>Retrospective</td>
<td>12 months</td>
<td>13.6 death, MI or heart failure</td>
</tr>
<tr>
<td>Ortolani et al (2009)</td>
<td>*PPCI 1124</td>
<td>Retrospective cohort study</td>
<td>24 months</td>
<td>21.0 death and MI 27.6 MACE (death, re-infarction, target vessel revascularisation)</td>
</tr>
<tr>
<td>Jakobsen et al (2010)</td>
<td>ÞPPCI 1320</td>
<td>Retrospective</td>
<td>12 months 24 months</td>
<td>17.8 MACE (death, stroke and MI) (12 months) 22.0 MACE (24 months)</td>
</tr>
<tr>
<td>Anderson et al (2003) (DANAMI-2 study)</td>
<td>PPCI 686</td>
<td>RCT</td>
<td>12 months 24 months</td>
<td>13.6 MACE (death, stroke and MI) (12 months) 17.3 MACE (24 months)</td>
</tr>
<tr>
<td>Montalescot et al (2001) (ADIMARAL study)</td>
<td>¥PPCI 151</td>
<td>RCT</td>
<td>30 days 6 months</td>
<td>10.3 MACE (death, MI, revascularisation) (30 days) 11.7 MACE (6 months) 2.1 MI (6 months)</td>
</tr>
<tr>
<td>Hubbard et al (2007)</td>
<td>βPCI (mixed cohort) 2731</td>
<td>Retrospective</td>
<td>30 days</td>
<td>9.3 (255) re-presented &amp; tested for cardiac ischaemia Diagnoses for re-presenter (255) 7.0 MI 20.0 coronary atherosclerosis 6.0 heart failure 5.0 cardiac dysrhythmia 33.0 non-specific chest pain Emergency/urgent vs elective PCI group likely to re-present Odds ratio 1.98, 95% CI 1.3, 3.0</td>
</tr>
<tr>
<td>Stone et al (2002) (CADILLAC study)</td>
<td>χPPCI 2082</td>
<td>RCT</td>
<td>6 months</td>
<td>14.6 MACE (death, stroke, MI, revascularisation) 6 months</td>
</tr>
<tr>
<td>Sutton (2005) (MERLIN study)</td>
<td>φPCI (rescue) 153</td>
<td>RCT</td>
<td>12 months</td>
<td>54.3 composite unplanned revascularisation, stroke, MI, heart failure</td>
</tr>
<tr>
<td>Authors</td>
<td>Cohort</td>
<td>Study type</td>
<td>Follow-up time point</td>
<td>Event rate results at follow-up (%)</td>
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<tr>
<td>Gershlick et al (2005)</td>
<td>◊PCI (rescue)</td>
<td>RCT</td>
<td>6 months</td>
<td>15.3 MACE (death, MI, stroke, severe HF)</td>
</tr>
</tbody>
</table>

◊PCI cohort only presented for the DANAMI-2 sub study
± standard deviation
★ characteristics of the group receiving stents during PPCI
☆ characteristics of the placebo group who received PPCI and no glycoprotein IIb/IIIa agent (IIb/IIIa agent)
* PPCI patients receiving usual MI care glycoprotein IIb/IIIa agent delivered in the cathlab or early IIb/IIIa agent prior to cathlab.
Ø PPCI control group results presented who received usual care with heparin in the cathlab.
≈ results for PPCI group only
â results presented for PPCI patients screened but not reaching entry criteria for DANAMI-2 study group only
β mixed cohort of PCI (emergency, urgent or elective) patients re-presenting to accident and emergency department
◊ results presented for rescue PCI (i.e. PCI delivered following failed thrombolysis MI treatment) sample only
MACE- major adverse cardiac event
## B.2 Psychological Studies

<table>
<thead>
<tr>
<th>Author</th>
<th>Cohort</th>
<th>Study type and country conducted</th>
<th>Assessment</th>
<th>Baseline</th>
<th>Follow-up</th>
<th>Results</th>
<th>Conclusion</th>
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</thead>
<tbody>
<tr>
<td>Roest et al</td>
<td>MI</td>
<td>5750</td>
<td>Meta-analysis (12 studies)</td>
<td>Within 3 months of MI</td>
<td>Mean 2.6 years post MI</td>
<td><strong>Anxiety</strong> 13.4% to 59.5% patients at baseline <strong>Anxiety associated with:</strong> cardiac and all cause mortality and cardiac events combined OR 1.36 (p&lt;0.001)</td>
<td>Bivariate analysis identified an association between anxiety and cardiac events and mortality following MI.</td>
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<tr>
<td></td>
<td>1649 anxious</td>
<td>4101 non-anxious</td>
<td>Anxiety: reliable valid instrument</td>
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<tr>
<td></td>
<td>(2010)</td>
<td>54.0 to 63.1 82.5</td>
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<tr>
<td>Frazier et al</td>
<td>MI</td>
<td>101</td>
<td>Anxiety: SAI</td>
<td>48 hours post MI</td>
<td>Nil</td>
<td><strong>Anxiety</strong> 22.8% mild (SAI scores 30-37) 24.8% moderate (SAI scores 38-44) 21.8% extreme (SAI scores 45-77) <strong>Clinical assessment of anxiety</strong> No association between the SAI scores and clinician assessment of anxiety (λ=0.03, p=0.05)</td>
<td>Anxiety found not to be routinely, logically or accurately assessed or treated appropriately by clinicians.</td>
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<tr>
<td></td>
<td>(2002)</td>
<td>60.7 (±12.8) 53.0</td>
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<tr>
<td>Martens et al</td>
<td>CHD</td>
<td>1015</td>
<td>Anxiety: computerised Diagnostic Interview Schedule for DSM-IV.</td>
<td>Attendance at outpatient clinic</td>
<td>5 years</td>
<td><strong>Anxiety</strong> (GAD) 10.4% of participants during previous 12 months <strong>Cardiovascular (CV) events</strong> 9.6% CV annual event rate for GAD group vs 6.6% for non-GAD (p=0.03) <strong>Anxiety associated with:</strong> increased risk of CV events (HR 1.62, p&lt;0.01) controlled for demographics, comorbidity, depression, severity of CHD and adherence to medication</td>
<td>In stable CHD patients anxiety was associated with increased CV events; this was not explained by CHD severity, behavioral or biological factors.</td>
</tr>
<tr>
<td></td>
<td>(stable)</td>
<td>1015</td>
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<tr>
<td></td>
<td>106</td>
<td>No anxiety 909</td>
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<tr>
<td></td>
<td>(2010)</td>
<td>*60.0 (±11.0) 67.0</td>
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<td>84.0</td>
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<tr>
<td>Author</td>
<td>Cohort</td>
<td>Study type and country conducted</td>
<td>Assessment</td>
<td>Baseline</td>
<td>Follow-up</td>
<td>Results At follow-up (unless otherwise stated)</td>
<td>Conclusion</td>
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<tr>
<td>Srinivasan and Joseph (2004)</td>
<td>Chest pain 337</td>
<td>CHD+PD 29 (9%) PD 28 (8%) **CHD 177 (53%) No CHD/PD 81 (24%)</td>
<td>Age not provided for total cohort 72.4 Cohort India</td>
<td>Anxiety: HADS SCAN</td>
<td>During attendance at A&amp;E or as in-patient. Nil</td>
<td><strong>Psychiatric disorder</strong> (PD) (SCAN) 12.8% anxiety (8.9% panic disorder) 10.4% depression Anxiety (mean HADS scores) related to chest pain diagnosis 8.2 (±3.7) CHD+PD diagnosis 14.3 (±3.0) PD diagnosis 3.5 (±3.0) CHD only 3.3 (±2.8) no CHD or PD Significant difference in HADS anxiety scores between CHD+PD and PD groups compared to CHD alone and no CHD/PD groups (p&lt;0.001)</td>
<td>Psychiatric distress and coronary heart disease appear to coexist in patients presenting to A&amp;E for assessment of chest pain.</td>
</tr>
<tr>
<td>Fleet et al (1996)</td>
<td>Chest pain 441</td>
<td>Panic disorder 108 Non-panic disorder 333</td>
<td>56.8 (±12.4) 61.0 Cohort Canada</td>
<td>Anxiety: STAI ADIS-R Depression: BDI</td>
<td>During attendance at A&amp;E Anxiety 8.2% GAD 24.5% panic disorder (panic) <strong>Chest pain (CP) type (%)</strong> (panic vs non-PD) 80.0 panic vs 61.0 non-panic had either atypical or non-anginal CP (p=0.0004) <strong>CP diagnosis (%)</strong> (panic vs non-panic) 75.0 panic vs 52.3 non-panic= non-cardiac 25.0 panic vs 47.8 non- panic = stable/ unstable angina or MI (p&lt;0.0002)</td>
<td>Panic disorder is prevalent in a quarter of chest pain attendees at A&amp;E. Atypical or non-anginal pain is more common in panic disorder patients than those without panic.</td>
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<td>Author</td>
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<tr>
<td>Strik et al (2003)</td>
<td>STEMI (first)</td>
<td>Cohort 58.0 (±11.0) 100%</td>
<td>Anxiety and Depression: 90-item SCL-90</td>
<td>1 month post first STEMI</td>
<td>Mean 3.4 years (1-70 months)</td>
<td>Anxiety (baseline) 59.5% total cohort 59.9% invasive vs 59.2% non-invasive (p=0.907) Depression (baseline) 47.1% total cohort 41.6% invasive vs 51.4% non-invasive (p=0.085) MACE (cardiac death or MI) 7.9% (25) total cohort MACE Independent predictors of cardiac events: Anxiety HR 2.79 (p=0.029), LVEF &lt;50% HR 2.29 (p=0.047) and age&gt;58 years HR 2.44 (p=0.047). Predictor of increased healthcare use: anxiety OR 2.00 (p=0.005)</td>
<td>Symptoms of anxiety at baseline were an independent predictor of cardiac events and healthcare consumption. Conversely symptoms of depression were not associated with cardiac events or increased healthcare consumption at follow-up.</td>
</tr>
<tr>
<td>Author</td>
<td>Cohort</td>
<td>Study type and country conducted</td>
<td>Assessment</td>
<td>Baseline</td>
<td>Follow-up</td>
<td>Results At follow-up (unless otherwise stated)</td>
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<tr>
<td>Grace et al (2004) and (2005a)</td>
<td>ACS 906</td>
<td>Cohort Canada</td>
<td>Anxiety: MHQ PRIME-MD</td>
<td>2 to 5 days post ACS admission</td>
<td>6 months 12 months</td>
<td>Anxiety- elevated or sub-threshold 36.6% baseline 23.8% 6 months 19.4% 12 months Depression (BDI ≥10) 31.3% baseline 25.2% 6 months 21.7% 12 months ACS Symptoms (number on admission) 7.56 (±4.6) high anxiety vs 5.38 (±3.7) no anxiety (p&lt;0.001) 80.4% participants with panic disorder (6 months) experienced recurrent IHD at 12 months Predictive of recurrent IHD event at 12 months: Older age, greater depression (baseline), CVD family history, elevated anxiety (6 month) Anxiety and depression scores declined at 6 and 12 months. Anxious participants reported significantly more symptoms and were significantly more likely to have atypical symptoms than non-anxious participants.</td>
<td></td>
</tr>
<tr>
<td>Mortensen et al (2005)</td>
<td>STEMI 1351</td>
<td>RCT Denmark</td>
<td>Anxiety and depression: HADS</td>
<td>One month post MI Nil</td>
<td></td>
<td>Anxiety (HADS≥ 11) Lysis 10.0% vs PPCI 10.8%, p=0.71 Depression (HADS≥ 11) Lysis 7.3% vs PPCI 6.4%, p=0.58 SF-36 physical summary scale (PS) Lysis 42.8 (sd 9.5) vs PPCI 44.3 (sd 9.5), p&lt;0.01 Rose angina questionnaire Angina: lysis 75% vs PPCI 80%, p&lt;0.01 HRQoL and angina significantly better in PPCI compared to lysis group at one month. Association between variables not established as multivariate analysis not performed.</td>
<td></td>
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<tr>
<td>Author</td>
<td>Cohort</td>
<td>Study type and country conducted</td>
<td>Assessment</td>
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<td>Follow-up</td>
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</tbody>
</table>
| McGowan et al (2004)           | MI 305 | Cohort UK                        | Anxiety and Depression: HADS Vital exhaustion: MQ | Mean 3.6 (±3.4) days post MI | Nil        | **Anxiety** (mean HADS) 7.4 (±4.9) baseline  
**Depression** (mean HADS) 4.9 (±3.9) baseline  
**Depression and anxiety** were highly correlated with vital exhaustion \( (r=0.59, r=0.56, p<0.01) \) when controlling for age, gender and comorbidity. Fatigue also correlated with depression and anxiety \( (r= 0.50, r=0.47, p<0.01) \). | There was a high correlation between vital exhaustion (particularly fatigue) and depression and anxiety; this was not related to comorbid conditions. |
| Dickens et al (2004, 2007)     | MI 589 | Cohort UK                        | Anxiety and Depression: HADS | One week prior to MI 12 months 8 years (mortality) | 12 months 23.8% (140) at baseline 12.1% (71) at 12 months (new onset) 46.4% (273) no depression at any point  
**Mortality and cardiac events** 6.5% died by 12 months 23.5% died by 8 years 13.3% died of cardiac cause (8 years)  
**Survival** at 8 years was equivalent between those depressed one week before MI (mean 89.2 months, 95% CI 84.7 to 93.8) and those not depressed (89.9 months, CI 87.4 to 92.4, \( p=0.75 \)). | Symptoms of depression one week before MI did not influence survival at 12 months or 8 years. |
<table>
<thead>
<tr>
<th>Author &amp; Year</th>
<th>Type</th>
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<table>
<thead>
<tr>
<th>Cohort</th>
<th>Age (mean yrs) (SD or range)</th>
<th>Study type and country conducted</th>
<th>Assessment</th>
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<th>Follow-up</th>
<th>Results</th>
<th>Conclusion</th>
</tr>
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<tbody>
<tr>
<td>UK</td>
<td>57.6 (±11.2) 63.0</td>
<td>Cohort</td>
<td>Depression: HADS</td>
<td>One week prior to MI</td>
<td>12 month 8 years (mortality)</td>
<td>Depression (total HADS ≥17) 16.3% (96) at baseline 12.1% (71) at 12 months (new onset) 46.4% (273) no depression at any point</td>
<td>Depression following MI is predictive of increased cardiac events at mean 8 years following MI.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author &amp; Year</th>
<th>Type</th>
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<tbody>
<tr>
<td>de Jonge et al (2006)</td>
<td>MI 468</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cohort</th>
<th>Age (mean yrs) (SD or range)</th>
<th>Study type and country conducted</th>
<th>Assessment</th>
<th>Baseline</th>
<th>Follow-up</th>
<th>Results</th>
<th>Conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Netherlands</td>
<td>57.6 (±11.2) 63.0</td>
<td>Cohort</td>
<td>Depression: CIDI</td>
<td>3 months post MI</td>
<td>12 months 2.5 years</td>
<td>Depression 25.4% (119) post MI depression of these patients: 44.5% (53) incident depression 55.4% (66) non-incident depression</td>
<td>New onset depression following MI is predictive of increased cardiac events at mean 2.5 years following MI.</td>
</tr>
<tr>
<td>Author</td>
<td>Cohort</td>
<td>Study type and country conducted</td>
<td>Assessment</td>
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<td>Follow-up</td>
<td>Results</td>
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<tr>
<td>Grace et al</td>
<td>ACS 705 MI &amp; UA</td>
<td>Cohort Canada</td>
<td>Depression: Interview BDI</td>
<td>≥2 weeks</td>
<td>2 years</td>
<td>Depression 23.2% history of depression 31.3% during admission (BDI score ≥ 10)</td>
<td>Symptoms of depression during admission led to increased likelihood of death at both 2 and 5 years post MI, whereas history of depression ≥2 weeks prior to MI did not predict increased mortality.</td>
</tr>
<tr>
<td>Van Melle et al</td>
<td>MI 6367</td>
<td>Meta-analysis (22 studies)</td>
<td>Depression and mortality</td>
<td>Mean 13.7 months</td>
<td>5 years</td>
<td>Depression was associated with all cause mortality OR 2.38 (p&lt;0.00001) cardiac mortality OR 2.59, p&lt;0.00001 and increased risk of new cardiac events OR 1.95 (p=0.0006)</td>
<td>Depression following MI was associated with increased cardiac mortality and new events.</td>
</tr>
<tr>
<td>Author</td>
<td>Cohort</td>
<td>Type number</td>
<td>Age (mean yrs)</td>
<td>Gender</td>
<td>Study type and country conducted</td>
<td>Assessment</td>
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</table>
| Frasure-Smith (1993) and (1995a) | MI 222 | 60.0 78.0 | Cohort Canada | Depression: DIS BDI | One week post MI | 6 months 18 months | **Depression at baseline** 15.8% major depression (DIS) 30.6% mild/moderate (BDI ≥10)  
**Mortality** 5.4% died (all cardiac) by 6 months 8.6% died (cardiac) by 18 months  
**At 6 months depression** was a predictor of mortality after control for other multivariate predictor of cardiac mortality (Killip class and prior MI) HR 4.29 (p=0.013).  
**At 18 months depression** was also associated with cardiac mortality after controlling for prior MI, Killip class, PVCs OR 6.64 (p=0.0026). | Depression following MI was a risk factor for mortality at 6 months and 18 months. This was at least equivalent to left ventricular dysfunction (Killip class). |

**Assessment:** Short Form 36 (SF 36), Hospital Anxiety and Depression Scale (HADS), Rose angina questionnaire, Measures Middlesex Hospital Questionnaire (MHQ)-Phobic Anxiety Subscale, the Anxiety Subscale of the PRIME-MD, Beck Depressive Inventory (BDI), 90-item Symptom Check List (SCL-90), State-Trait Anxiety Inventory (STAI), The Maastricht Questionnaire (MQ; vital exhaustion), Composite International Diagnostic Interview (CIDI), National Institute of Mental Health Diagnostic Interview Schedule (DIS), Primary Care Evaluation of Mental Disorders Brief Patient Health Questionnaire (PHQ), Seattle Angina Questionnaire (SAQ), Spielberger State Anxiety Inventory (SAI), Computerized Diagnostic Interview Schedule for DSM-IV, Schedule for Clinical Assessment in Neuropsychiatry (SCAN), the Anxiety Disorders Interview Schedule-Revised (ADIS-R).

**GAD-** general anxiety disorder  
**CVD-** cardiovascular disease  
**A&E-** accident and emergency department  
**OR-** odds ratio  
**HR-** hazard ratio  
**CI-** confidence interval  
**PVCs-** premature ventricular contractions  
◊Invasive STEMI treatment included thrombolysis, percutaneous coronary intervention, coronary bypass surgery.  
¥incident depression refers to new depression starting following MI; non-incident depression refers to either depression prior to MI or continuation of depression since MI.
## Appendix C Table of psychological health and quality of life self-report questionnaires

<table>
<thead>
<tr>
<th>Focus of Instrument</th>
<th>Name of Instrument</th>
<th>Reference</th>
</tr>
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<tbody>
<tr>
<td>Anxiety</td>
<td>The Brief Symptom Inventory</td>
<td>Derogatis and Melisaratos (1983)</td>
</tr>
<tr>
<td></td>
<td>Diagnostic Interview Schedule for DSM-IV</td>
<td>Robins et al (1981)</td>
</tr>
<tr>
<td></td>
<td>Spielberger State Anxiety Inventory (SAI)</td>
<td>Spielberger (1993)</td>
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<tr>
<td></td>
<td>Middlesex Hospital Questionnaire (MHQ)-Phobic Anxiety Subscale</td>
<td>Crown and Crisp (1996)</td>
</tr>
<tr>
<td></td>
<td>State-Trait Anxiety Inventory (STAI)</td>
<td>Spielberger (1970)</td>
</tr>
<tr>
<td>Depression</td>
<td>Beck Depressive Inventory (BDI)</td>
<td>Beck (1978)</td>
</tr>
<tr>
<td></td>
<td>National Institute of Mental Health Diagnostic Interview Schedule (DIS)</td>
<td>Robins et al (1981)</td>
</tr>
<tr>
<td></td>
<td>Montgomery and Asberg Depression Rating Scale (MADRSAS)</td>
<td>Montgomery and Asberg (1979)</td>
</tr>
<tr>
<td></td>
<td>Depression Interview and Structured Hamilton (DISH)</td>
<td>Freelander et al (2002)</td>
</tr>
<tr>
<td></td>
<td>Hamilton Rating Scale for Depression (HRSD)</td>
<td>Williams (1988)</td>
</tr>
<tr>
<td>Anxiety and depression</td>
<td>90-item Symptom Check List (SCL-90)</td>
<td>Derogatis et al (1973)</td>
</tr>
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<td></td>
<td>Hospital Anxiety and Depression Scale (HADS)</td>
<td>Zigmond and Snaith (1983)</td>
</tr>
<tr>
<td></td>
<td>Primary Care Evaluation of Mental Disorders Brief Patient Health Questionnaire (PHQ)</td>
<td>Spitzer et al (1994)</td>
</tr>
<tr>
<td>Psychological health</td>
<td>Cognitive Behaviour Assessment Hospital Form</td>
<td>Bettinard and Zotti (1995)</td>
</tr>
<tr>
<td>Quality of life</td>
<td>Medical Outcomes Study Short Form 36 (SF-36)- generic</td>
<td>Ware (1993)</td>
</tr>
<tr>
<td></td>
<td>Seattle Angina Questionnaire (SAQ)- disease specific</td>
<td>Spertus et al (1995)</td>
</tr>
</tbody>
</table>
Appendix D Research ethics approval

32 October 2006

Dr Farzin Feth-Ordoubadi
Consultant Cardiologist
Manchester Heart Centre
Manchester Royal Infirmary
Oxford Road
Manchester
M13 9WL

Dear Dr Feth-Ordoubadi

Full title of study:

**MAIN STUDY**: Acute Intervention for Myocardial Infarction: Patient Experiences and Patient-Related Outcomes

**SUB-STUDY**: An investigation of the relationship between psychosocial factors and non-physiological cardiac symptoms in post primary angioplasty patients following ST-elevation myocardial infarction

REC reference number: 06/Q1401/77

Thank you for your letter of 19 September 2006, responding to the Committee’s request for further information on the above research and submitting revised documentation.

The further information has been considered on behalf of the Committee by the Chair.

**Confirmation of ethical opinion**

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised.

Stockport Primary Care Trust
Stockport Research Ethics Committee
Room 131, 1st Floor
Gateway House
Pond Street South
Manchester
M30 7UX

Telephone: 0161 297 2100
Fax: 0161 297 2363
Email: rai.ethics@ijimh.nhs.uk

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Ethical review of research sites

The Committee has designated this study as exempt from site-specific assessment (SSA) There is no requirement for Local Research Ethics Committees to be informed or for site-specific assessment to be carried out at each site.

Conditions of approval

The favourable opinion is given provided that you comply with the conditions set out in the attached document. You are advised to study the conditions carefully.

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

<table>
<thead>
<tr>
<th>Document</th>
<th>Version</th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Application</td>
<td>s.1</td>
<td>03 August 2009</td>
</tr>
<tr>
<td>Investigator CV (Dr Farzin Foro-Ordoubadi)</td>
<td></td>
<td>03 August 2009</td>
</tr>
<tr>
<td>Investigator CV (Dr Linda McGowan)</td>
<td></td>
<td>02 August 2008</td>
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<tr>
<td>Protocol</td>
<td>1.2</td>
<td>02 August 2008</td>
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<tr>
<td>Covering Letter</td>
<td></td>
<td>02 August 2008</td>
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<tr>
<td>Summary/Synopsis (Appendix B)</td>
<td>1.2</td>
<td>02 August 2008</td>
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<td>Questionnaire: SAD (Appendix A)</td>
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<td>Questionnaire: EBS (Appendix A)</td>
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<td>Questionnaire: TADS (Appendix A)</td>
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</tr>
<tr>
<td>Questionnaire: ISO (Appendix A)</td>
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<td>Questionnaire re: SH-28 (Appendix A)</td>
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<tr>
<td>Letter to invited participant</td>
<td>1</td>
<td>02 August 2008</td>
</tr>
<tr>
<td>Participant Information Sheet</td>
<td>1</td>
<td>02 August 2008</td>
</tr>
<tr>
<td>Participant Consent Form</td>
<td>2</td>
<td>13 September 2008</td>
</tr>
<tr>
<td>Response in Request for Further Information</td>
<td></td>
<td>19 September 2006</td>
</tr>
<tr>
<td>Email re: Funding Details</td>
<td></td>
<td>15 July 2009</td>
</tr>
<tr>
<td>E-Mail re: Sponsorship Agreement</td>
<td></td>
<td>01 August 2008</td>
</tr>
<tr>
<td>List of Publications (Appendix F)</td>
<td>1.1</td>
<td>30 May 2009</td>
</tr>
<tr>
<td>E-Red Paper (Appendix E)</td>
<td>1.2</td>
<td>02 August 2008</td>
</tr>
<tr>
<td>Sum Study Protocol (Appendix D)</td>
<td>1</td>
<td>13 April 2008</td>
</tr>
<tr>
<td>List of Coatings (Appendix C)</td>
<td></td>
<td>01 June 2008</td>
</tr>
</tbody>
</table>

Research governance approval

You should arrange for the R&D department at all relevant NHS care organisations to be notified that the research will be taking place, and provide a copy of the R&D application, the protocol and this letter.

All researchers and research collaborators who will be participating in the research must obtain final research governance approval before commencing any research procedures. Where a substantive contract is not held with the care organisation, it may be necessary for an honorary contract to be issued before approval for the research can be given.
Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

06/Q1401/77 Please quote this number on all correspondence

With the Committee's best wishes for the success of this project.

Yours sincerely,

Dr Effi Eyong
Chair

Email: m.flatt.bowcock@northwest.nhs.uk

Encl.

Standard approval conditions

Copy to:
Prof Henry Kitchener
R&D Office
1st Floor
Postgraduate Centre
Manchester Royal Infirmary
Oxford Road
Manchester
M13 9WL

Mrs Heather Kee-Smith
Research Co-ordinator
Manchester Heart Centre
Manchester Royal Infirmary
Oxford Road
Manchester
M13 9WL

An advisory Committee to NW North West
Appendix E Patient information sheet and consent form

Central Manchester and Manchester Children’s University Hospitals

Patient Information Sheet

Title of Project:

Title of main study:
Acute Intervention for Myocardial Infarction: Patient Experiences and Patient-Related Outcomes

Title of sub-study:
An Investigation of the Relationship between Psychosocial Factors and No Physiological Cardiac Symptoms in Primary Percutaneous Coronary Intervention Patients following ST-Elevation Myocardial Infarction

Investigators:
Dr Fath-Ordoubadi, Sister Isles-Smith, Professor Denton and Dr McGowan

Introduction
You are being invited to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish, this includes your cardiologist (heart doctor). Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part.

This research study is being carried out by the Manchester Heart Centre, Manchester Royal Infirmary and the School of Nursing, Midwifery and Social Work, University of Manchester.

The doctor responsible for the study is Dr Fath-Ordoubadi, Consultant Cardiologist, Manchester Heart Centre. The study is being supported by Professor Denton and Dr McGowan from The University of Manchester.

A sub-study is also being conducted by Heather Isles-Smith a PhD student from the University of Manchester, who is being supervised by Professor Denton and Dr McGowan.

Why is this study being carried out?
As you are now aware you have recently suffered a heart attack. You have received treatment for this in the form of either angioplasty or a clot busting drug known as thrombolysis. Thrombolysis is a well established treatment and is used across the United Kingdom. Angioplasty, during a heart attack, is known as primary angioplasty and is a newer treatment.
Primary angioplasty is a developing service so doctors and nurses are unsure of how the support services, such as cardiac rehabilitation need to be adapted for patients receiving this treatment. Cardiac rehabilitation has been available to cardiac patients such as those who receive a bypass operation, angioplasty or suffer a heart attack for many years. It is very effective at helping individuals to recover from their illness and help them to adapt their lifestyles to prevent further cardiac problems.

Primary angioplasty is an invasive life saving treatment which is carried out in an emergency situation. In many circumstances patients may need to be transferred to a hospital other than their local one to receive treatment. Many doctors and nurses believe that the after care and rehabilitation service may need to be adapted for patients who receive primary angioplasty, to allow for these circumstances. It is important that changes to these services meet the needs of patients and are informed by patient feedback. It is the intention of this study to collect information from individuals who suffer a heart attack and are treated with either thrombolysis or primary angioplasty, so that their experiences can be compared and fed back into service development.

In the sub-study patients who have received primary angioplasty and have suffered chest pain and/or breathlessness since their treatment will be followed up. This will help doctors and nurses to discover the reasons for these symptoms and the number of people who are effected. The study is being undertaken by Heather Iles-Smith as part of a PhD at the University of Manchester and is being supervised by Professor Deaton and Dr McGowan.

**What are the aims of the main study?**
The aims of the study are to:
- Investigate how people who have had a heart attack adjust to life afterwards and compare people who have received thrombolysis or angioplasty treatment
- Look at how anxiety, depression, social life and the way people cope following a heart attack affects recovery.

**Why have I been invited to take part?**
You have been invited to take part in this study because you have recently suffered a heart attack and been treated with either thrombolysis or primary angioplasty. A total of 350 patients, 175 angioplasty and 175 thrombolysis patients will be asked to take part.

**Do I have to take part?**
It is up to you to decide whether or not to take part. If you decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part you are still free to withdraw at any time and without giving a reason. A decision to withdraw at any time, or a decision not to take part, will not affect the standard of care you receive.
What will I be asked to do as part of the study?
You will be asked to complete four questionnaires on three occasions. The first occasion will be within a week of your discharge home from hospital. The second and third occasions will be at 6 and 12 months after your heart attack. On both these occasions the questionnaires will be posted to you and a study nurse or researcher will telephone you to answer any queries you have regarding completing the questionnaires. The questionnaires will take approximately 10 minutes each to complete.
We will contact your GP to inform them of your participation in the study. We may also contact your GP at 6 and 12 months after your heart attack to see if you have been admitted to hospital.

What is the sub-study?
The sub-study only involves patients who have received primary angioplasty as treatment for their heart attack.
The aim of the study is to discover how many of these patients suffer cardiac like symptoms for one year following their angioplasty and the cause of symptoms.

Will I be asked to take part in the sub-study?
You will only be asked if you would like to take part in the sub-study if you received primary angioplasty as your treatment at the time of your heart attack.

What will I be asked to do as part of the sub-study?
If you agree to take part in the sub-study, the researcher will ask you some questions during the each of the three telephone calls mentioned earlier. There will not be extra telephone calls. The questions will relate to any cardiac sounding symptoms that you may have experienced. You will be asked if you have made any visits to your GP or to hospital. Your hospital records will also be examined to collect information relating to any admissions during the year follow up. Twenty of the patients participating in the sub-study will also be interviewed in depth about their experiences of angioplasty and any cardiac sounding symptoms that they may have had. Generally the interviews will take place in the patients home or otherwise at the hospital.

How long will the study last?
The study will last for 12 months.

Are there any benefits to participating in the study?
This study may not directly benefit you but the results will provide information, which may benefit other patients undergoing the same treatment in the future.

Will there be any risks?
You will not experience any risks through taking part in this study. However the information you give through the questionnaires may show that you are suffering from anxiety and/or depression. If this is the case the researcher will discuss the matter with you end with your permission will refer you to your GP for treatment.
What about confidentiality of information?
The information you give during the study will be treated in confidence. During the study you will only be referred to by a study code and your initials. Your name or other information, which may identify you, will not appear in any of the final study reports.

You will be asked to sign a consent form before the start of the study to show that you have received information, had the opportunity to ask questions, think about and discuss the study with others.

Who should I ask if I want more information?
You should ask the doctor responsible for the study Dr Fath-Ordoubadi on 0161 276 5570 or the Research Coordinator, Sister Iles-Smith on 0161 276 6195 if you are unsure of anything, have any questions or are worried about taking part in the study for any reason.
PATIENT INFORMED CONSENT FORM

Title of Project:
Acute Intervention for ST-Elevation Myocardial Infarction: Patient Experiences and Patient Related Outcomes Study

Name of Researchers:
Dr Fath-Ordoubadi, Sister Iles-Smith, Professor Deaton and Dr McGowan

Please initial boxes

1. I confirm that I have read and understand the information sheet dated 16th November 2007 (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 Manchester Heart Centre, the University of Manchester 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.

5. I give consent for my GP to be contacted.

6. I agree to take part in the sub-study:
   An investigation of the Relationship between Psychosocial Factors and Non Physiological Cardiac Symptoms in Poel PCI Patients Following ST-Elevation Myocardial Infarction.

__________________________________________  ____________________________  ____________________________
Name of Patient                                  Date                                    Patient’s Signature

__________________________________________  ____________________________  ____________________________
Name of Researcher                               Date                                    Researcher’s Signature

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

Acute Intervention for ST-Elevation Myocardial Infarction: Patient Experiences and Patient Related Outcomes Study
Version 3                                     19/11/2007
Page 1 of 1
Appendix F Patient pack (at baseline)

Date filled in: __________  Patient study ID no. ___

Background Information

Firstly we would like to ask some questions about you. These details will be kept confidential. If you do not wish to answer some of the questions you do not have to.

What is your sex?  
Male  
Female

What is your age and date of birth?

What is your occupational status?  
Employed full-time  
Employed part-time  
Self-employed  
Unemployed  
Retired  
Housework

What is your occupation?  
(Previous occupation if retired)

What is the highest level of education you have obtained?  
left school without certificates, O’Level, A’level, degree etc

What are your living arrangements?  
Live alone  
Live with others at home

What is your ethnic group?  
White  
UK  
Irish  
Other European  
Other  
Mixed  
White & Black Caribbean  
White & Black African  
White & Asian  
Other

Black  
Caribbean  
African  
Other

Asian  
Indian  
Pakistani  
Bangladeshi  
Other

If “other” please specify below

Thank you for taking the time to complete these questionnaires  
Please return them in the stamped addressed envelope provided
Hospital Anxiety and Depression Scale

Doctors are aware that emotions play an important part in most illnesses. This questionnaire is designed to help your doctor to know how you are feeling.

Please read each item, and then place a tick in the box, which comes closest to how you have been feeling during the past week. Don’t take too long over your replies; your immediate reaction to each item will probably be more accurate than a long thought out response.

<table>
<thead>
<tr>
<th>I feel tense or wound up</th>
<th>I feel cheerful</th>
</tr>
</thead>
<tbody>
<tr>
<td>Most of the time</td>
<td>Not at all</td>
</tr>
<tr>
<td>A lot of the time</td>
<td>Not often</td>
</tr>
<tr>
<td>Time to time, occasionally</td>
<td>Sometimes</td>
</tr>
<tr>
<td>Not at all</td>
<td>Most of the time</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>I feel restless as if I have to be on the move</th>
<th>I still enjoy the things I used to enjoy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Very much indeed</td>
<td>Definitely as much</td>
</tr>
<tr>
<td>Quite a lot</td>
<td>Not quite as much</td>
</tr>
<tr>
<td>Not very much</td>
<td>Only a little</td>
</tr>
<tr>
<td>Not at all</td>
<td>Hardly at all</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>I can sit at ease and feel relaxed</th>
<th>I look forward with enjoyment to things</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definitely</td>
<td>As much as I ever did</td>
</tr>
<tr>
<td>Usually</td>
<td>Rather less than I used to</td>
</tr>
<tr>
<td>Not often</td>
<td>Definitely less than I used to</td>
</tr>
<tr>
<td>Not at all</td>
<td>Hardly at all</td>
</tr>
<tr>
<td>I get a sort of frightened feeling as if something awful is about to happen</td>
<td>I feel as if I’m slowed down</td>
</tr>
<tr>
<td>I get sudden feelings of panic</td>
<td>I can laugh and see the funny side of things</td>
</tr>
<tr>
<td>--------------------------------</td>
<td>---------------------------------------------</td>
</tr>
<tr>
<td>Very often Indeed</td>
<td>As much as I always could</td>
</tr>
<tr>
<td>Quite often</td>
<td>Not quite so much now</td>
</tr>
<tr>
<td>Not very often</td>
<td>Definitely not so much now</td>
</tr>
<tr>
<td>Not at all</td>
<td>Not at all</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>I get a sort of frightened feeling like ‘butterflies’ in the stomach</th>
<th>I can enjoy a good book or radio or TV programme</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not at all</td>
<td>Often</td>
</tr>
<tr>
<td>Occasionally</td>
<td>Sometimes</td>
</tr>
<tr>
<td>Quite often</td>
<td>Not often</td>
</tr>
<tr>
<td>Very often</td>
<td>Very seldom</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Worrying thoughts go through my mind</th>
<th>I have lost interest in my appearance</th>
</tr>
</thead>
<tbody>
<tr>
<td>A great deal of the time</td>
<td>Definitely</td>
</tr>
<tr>
<td>A lot of the time</td>
<td>I don’t take so much care as I should</td>
</tr>
<tr>
<td>From time to time but not too often</td>
<td>I may not take quite as much care</td>
</tr>
<tr>
<td>Only occasionally</td>
<td>I take just as much care as ever</td>
</tr>
</tbody>
</table>
The Seattle Angina Questionnaire

1. The following is a list of activities that people often do during the week. Although for some people with several medical problems it is difficult to determine what it is that limits them, please go over the activities listed below and indicate how much limitation you have had due to chest pain, chest tightness or angina over the past 4 weeks.

   Place a tick in one box on each line

<table>
<thead>
<tr>
<th>Activity</th>
<th>Severely limited</th>
<th>Moderately limited</th>
<th>Somewhat limited</th>
<th>A little limited</th>
<th>Not limited</th>
<th>Limited or did not do for other reasons</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dressing yourself</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Walking indoors on level ground</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Showering</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Climbing a hill or a flight of stairs without stopping</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Gardening, vacuuming or carrying groceries</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Walking more than a block at a brisk pace</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACTIVITY</td>
<td>Severely limited</td>
<td>Moderately limited</td>
<td>Somewhat limited</td>
<td>A little limited</td>
<td>Not limited</td>
<td>Limited or did not do for other reasons</td>
</tr>
<tr>
<td>---------------------------------------------------</td>
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<td>------------------</td>
<td>-------------</td>
<td>----------------------------------------</td>
</tr>
<tr>
<td>Running or jogging</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lifting or moving heavy objects (e.g. furniture, children)</td>
<td></td>
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<tr>
<td>Participating in strenuous sports (e.g. swimming, tennis)</td>
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</tbody>
</table>

2. **Compared with 4 weeks ago**, how often do you have chest pain, chest tightness, or angina when doing your most strenuous level of activity?

I have chest pain, chest tightness or angina...

- Much more often
- Slightly more often
- About the same
- Slightly less often
- Much less often

3. **Over the past 4 weeks**, on average, how many times have you had chest pain, chest tightness or angina?

I have chest pain, chest tightness or angina...

- 4 or more times per day
- 1-3 times per day
- 3 or more times per week but not every day
- 1-2 times per week
- Less than once a week
- None over the past 4 weeks

Page 359
4. **Over the past 4 weeks**, on average, how many times have you had to take 
nitros (nitroglycerin tablets) for your chest pain, chest tightness or angina?

I take nitros...

<table>
<thead>
<tr>
<th>4 or more times per day</th>
<th>1-3 times per day</th>
<th>3 or more times per week but not every day</th>
<th>1-2 times per week</th>
<th>Less than once a week</th>
<th>None over the past 4 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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</tr>
</tbody>
</table>

5. How bothersome is it for you to take your pills for chest pain, chest tightness or angina as prescribed?

<table>
<thead>
<tr>
<th>Very bothersome</th>
<th>Moderately bothersome</th>
<th>Somewhat bothersome</th>
<th>A little bothersome</th>
<th>Not bothersome at all</th>
<th>My doctor has not prescribed pills</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td></td>
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</tr>
</tbody>
</table>

6. How satisfied are you that everything possible is being done to treat your chest pain, chest tightness or angina?

<table>
<thead>
<tr>
<th>Not satisfied at all</th>
<th>Mostly dissatisfied</th>
<th>Somewhat satisfied</th>
<th>Mostly satisfied</th>
<th>Highly satisfied</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

7. How satisfied are you with the explanations your doctor has given you about your chest pain, chest tightness or angina?

<table>
<thead>
<tr>
<th>Not satisfied at all</th>
<th>Mostly dissatisfied</th>
<th>Somewhat satisfied</th>
<th>Mostly satisfied</th>
<th>Highly satisfied</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>
8. Overall, how satisfied are you with the current treatment of your chest pain, chest tightness or angina?

<table>
<thead>
<tr>
<th>Satisfied at all</th>
<th>Mostly dissatisfied</th>
<th>Somewhat dissatisfied</th>
<th>Mostly satisfied</th>
<th>Highly satisfied</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

9. Over the past 4 weeks, how much has your chest pain, chest tightness or angina interfered with your enjoyment of life?

<table>
<thead>
<tr>
<th>Severe limitation of life</th>
<th>Moderate limitation of life</th>
<th>Slight limitation of life</th>
<th>Barely limited</th>
<th>Not limited</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

10. If you had to spend the rest of your life with your chest pain, chest tightness or angina the way it is now, how would you feel about this?

<table>
<thead>
<tr>
<th>Satisfied at all</th>
<th>Mostly dissatisfied</th>
<th>Somewhat dissatisfied</th>
<th>Mostly satisfied</th>
<th>Highly satisfied</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

11. How often do you worry that you may have a heart attack or die suddenly?

<table>
<thead>
<tr>
<th>Never</th>
<th>Rarely</th>
<th>Occasionally</th>
<th>Often</th>
<th>Can't stop worrying</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Page 361
ENRICHSD Social Support questionnaire

Please circle your answer:

1. Is there someone available to whom you can count on to listen to when you need to talk?

   None of the time  A little of the time  Some of the time  Most of the time  All of the time

2. Is there someone available to give you good advice about a problem?

   None of the time  A little of the time  Some of the time  Most of the time  All of the time

3. Is there someone available who shows you love and affection?

   None of the time  A little of the time  Some of the time  Most of the time  All of the time

4. Is there someone to help with daily chores?

   None of the time  A little of the time  Some of the time  Most of the time  All of the time

Can you count on anyone to provide you with emotional support (talking over problems or helping you make a difficult decision)?

   None of the time  A little of the time  Some of the time  Most of the time  All of the time

Do you have as much contact as you would like with someone you feel close to, someone in whom you can trust and confide in?

   None of the time  A little of the time  Some of the time  Most of the time  All of the time

7. Are you currently married or living with a partner?

   Yes  No
Appendix G Telephone interview (pro forma)

Baseline telephone interview:

Patient study ID: ………………………. Date of interview: ……………………

Angina and chest pain / discomfort (CCS)
How does the patient classify their angina symptoms leading up to their heart attack?

<table>
<thead>
<tr>
<th>TICK ONE BOX</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>N/A</td>
<td>No symptoms of Angina, chest pain or tightness even during the most strenuous activities.</td>
</tr>
<tr>
<td>Class 1</td>
<td>Ordinary physical activity does not cause angina, such as walking and climbing stairs. Symptoms occur only with strenuous, rapid, or prolonged exertion at work or recreation.</td>
</tr>
<tr>
<td>Class 2</td>
<td>Slight limitation of ordinary physical activity (e.g. walking up a hill, waking or stair climbing after meals, in cold wind, or under emotional stress or only the first hours after awakening. Walking more than two blocks on the level, or climbing more than one flight of ordinary stairs at a normal pace and in normal conditions).</td>
</tr>
<tr>
<td>Class 3</td>
<td>Checked limitation of ordinary physical activity (e.g. limiting symptoms caused by walking one to two blocks on the same level or climbing one flight of stairs).</td>
</tr>
<tr>
<td>Class 4</td>
<td>Inability to carry on any physical activity without causing discomfort. Anginal symptoms may be present at rest.</td>
</tr>
</tbody>
</table>

Shortness of breath (NYHA)
How does the patient classify their shortness of breath symptoms leading up to your heart attack?

<table>
<thead>
<tr>
<th>TICK ONE BOX</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I (Mild / Nil)</td>
<td>Asymptomatic with greater than normal activity. No limitation of physical activity. Physical activity does not cause undue fatigue, palpitation, or dyspnoea (shortness of breath).</td>
</tr>
<tr>
<td>Class II (Mild)</td>
<td>Symptomatic with greater than normal activity (climbing stairs, walking up hill). Slight limitation of physical activity. Comfortable at rest, but slightly strenuous physical activity results in fatigue, palpitation, or dyspnoea.</td>
</tr>
<tr>
<td>Class III (Moderate)</td>
<td>Symptomatic with normal activity (walking). Marked limitation of physical activity. Comfortable at rest, but ordinary activity causes fatigue, palpitation, or dyspnoea (shortness of breath).</td>
</tr>
<tr>
<td>Class IV (Severe)</td>
<td>Symptomatic at rest. Unable to carry out any physical activity without discomfort. Symptoms of shortness of breath present at rest. If any physical activity is undertaken, discomfort is increased.</td>
</tr>
</tbody>
</table>

History of anxiety and /or depression

Has the patient ever:
- had any problems with anxiety? ……………………………………………………………
- seen their GP due to problems with Anxiety?………………………………………………
- had any problems with Depression?…………………………………………………………
- ever seen your GP due to problems with Depression?………………………………………

Page 363
# Charlson Co-morbidity Index (CCI)

Estimation of 10 year survival- relating to the patients co-morbidity at baseline.

Age of the patient: _________ Years

## Before index MI, did the patient have?

<table>
<thead>
<tr>
<th>Condition</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIDS?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cerebrovascular disease?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic pulmonary disease?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Congestive heart failure?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Connective tissue disease?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dementia?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemiplegia?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leukemia?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant lymphoma?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peripheral vascular disease?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ulcer disease?</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Tick the appropriate column for each condition (give only one answer per row)

<table>
<thead>
<tr>
<th>Condition</th>
<th>None</th>
<th>Without end organ damage</th>
<th>With end organ damage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes mellitus</td>
<td>None</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Liver disease</td>
<td>None</td>
<td>Mild</td>
<td>Moderate</td>
</tr>
<tr>
<td>Renal disease</td>
<td>None</td>
<td>Non-metastatic</td>
<td>Metastatic</td>
</tr>
<tr>
<td>Malignant solid tumour</td>
<td>None</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Type of Diabetes (admission):
- Type I
- Type II
- New (during/ since STEMI admission)

Pulmonary disease:
- Yes
- No

## Other information
6 month telephone interview:

Patient study ID: .......................... Date of interview: ..........................

Current angina (i.e. chest pain / discomfort) classification (CCS)

<table>
<thead>
<tr>
<th>TICK ONE BOX</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>N/A - nil</td>
<td>No symptoms of Angina, chest pain or tightness even during the most strenuous activities.</td>
</tr>
<tr>
<td>Class 1</td>
<td>Ordinary physical activity does not cause angina, such as walking and climbing stairs. Symptoms occur only with strenuous, rapid, or prolonged exertion at work or recreation.</td>
</tr>
<tr>
<td>Class 2</td>
<td>Slight limitation of ordinary physical activity (e.g. waking up a bit, waking or stair climbing after meals, in cold wind, or under emotional stress or only the first hours after awakening. Walking more than two blocks on the level, or climbing more than one flight of ordinary stairs at a normal pace and in normal conditions).</td>
</tr>
<tr>
<td>Class 3</td>
<td>Checked limitation of ordinary physical activity (e.g. limiting symptoms caused by waking one to two blocks on the same level or climbing one flight of stairs).</td>
</tr>
<tr>
<td>Class 4</td>
<td>Inability to carry on any physical activity without causing discomfort. Anginal symptoms may be present at rest.</td>
</tr>
</tbody>
</table>

Current shortness of breath classification (NYHA)

<table>
<thead>
<tr>
<th>TICK ONE BOX</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I (Mild / Nil)</td>
<td>Asymptomatic with greater than normal activity. No limitation of physical activity. Physical activity does not cause undue fatigue, palpitation, or dyspnoea (shortness of breath).</td>
</tr>
<tr>
<td>Class II (Mild)</td>
<td>Symptomatic with greater than normal activity (climbing stairs, waking up hill). Slight limitation of physical activity. Comfortable at rest, but slightly strenuous physical activity results in fatigue, palpitation, or dyspnoea.</td>
</tr>
<tr>
<td>Class III (Moderate)</td>
<td>Symptomatic with normal activity (walking). Marked limitation of physical activity. Comfortable at rest, but ordinary activity causes fatigue, palpitation, or dyspnoea (shortness of breath).</td>
</tr>
<tr>
<td>Class IV (Severe)</td>
<td>Symptomatic at rest. Unable to carry out any physical activity without discomfort. Symptoms of shortness of breath present at rest. If any physical activity is undertaken, discomfort is increased.</td>
</tr>
</tbody>
</table>

Re-presentations (to A&E or acute services)
Total number during 6 month follow-up ..........................

<table>
<thead>
<tr>
<th>Date</th>
<th>*Re-presentation details</th>
<th>Symptoms</th>
<th>Tests and investigations</th>
<th>Diagnosis</th>
<th>Hospital /doctor</th>
</tr>
</thead>
</table>

*Re-presentation details – to A&E or admission to hospital

Cardiac rehabilitation (CR)
Invited to cardiac rehabilitation Y / N
<table>
<thead>
<tr>
<th>Number of CR sessions attended</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td></td>
</tr>
<tr>
<td>One</td>
<td></td>
</tr>
<tr>
<td>Some (≥ 2 to &lt; all)</td>
<td></td>
</tr>
<tr>
<td>All</td>
<td></td>
</tr>
</tbody>
</table>

**Further revascularisation**

Number of revascularisations during 6 month follow-up ...............  

<table>
<thead>
<tr>
<th>Date</th>
<th>*Type</th>
<th>Hospital</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Type of revascularisation e.g. PCI, PPCI, CABG
Appendix H Diary card

Front cover

Acute Intervention for Myocardial Infarction: Patient Experiences and Patient Related Outcomes
Patient’s Diary

Researchers: Dr Fath-Ordoubadi, Dr McGowan, Professor Deaton, Sister Iles-Smith
Contacts: 0161 276 7958/ 6195

Inside cover

Patient Details:
Patient Study ID Surname First Name

Address

TO BE RETAINED AND COMPLETED BY THE PATIENT

First page

As a participant in the Acute Intervention for Myocardial Infarction (heart attack) we would like you to keep a record of the following visits over the next 6 months:
- Accident and Emergency (A & E) department visits
- Admissions to hospital

This diary is also developed to keep track of the major tests and all medical treatments you receive during any attendances to A & E or during any admissions to hospital during the follow-up period.
Please return the diary with the final set of completed questionnaires in approximately 6 months time.

Second page

| DIARY CARD (diary for A & E department visits and admissions to hospital) |
|---|---|---|---|---|
| Date | Reason for Visit | Where | Tests/Procedures | Doctor |
|     |                 |      |             |       |
|     |                 |      |             |       |
|     |                 |      |             |       |
|     |                 |      |             |       |
|     |                 |      |             |       |
|     |                 |      |             |       |
|     |                 |      |             |       |
|     |                 |      |             |       |

Page 367
## Appendix I  
CCI- comorbidities included

<table>
<thead>
<tr>
<th>Condition</th>
<th>Assigned Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>myocardial infarction</td>
<td>1</td>
</tr>
<tr>
<td>congestive heart failure</td>
<td>1</td>
</tr>
<tr>
<td>peripheral vascular disease</td>
<td>1</td>
</tr>
<tr>
<td>cerebrovascular disease</td>
<td>1</td>
</tr>
<tr>
<td>dementia</td>
<td>1</td>
</tr>
<tr>
<td>chronic pulmonary disease</td>
<td>1</td>
</tr>
<tr>
<td>connective tissue disease</td>
<td>1</td>
</tr>
<tr>
<td>ulcer disease</td>
<td>1</td>
</tr>
<tr>
<td>liver disease, mild</td>
<td>1</td>
</tr>
<tr>
<td>diabetes</td>
<td>1</td>
</tr>
<tr>
<td>hemiplegia</td>
<td>2</td>
</tr>
<tr>
<td>renal disease, moderate or severe</td>
<td>2</td>
</tr>
<tr>
<td>diabetes with end organ damage</td>
<td>2</td>
</tr>
<tr>
<td>any malignancy</td>
<td>2</td>
</tr>
<tr>
<td>leukemia</td>
<td>2</td>
</tr>
<tr>
<td>malignant lymphoma</td>
<td>2</td>
</tr>
<tr>
<td>liver disease, moderate or severe</td>
<td>3</td>
</tr>
<tr>
<td>metastatic solid malignancy</td>
<td>6</td>
</tr>
<tr>
<td>AIDS</td>
<td>6</td>
</tr>
</tbody>
</table>

Limitations in assigned weights:
1. "Any malignancy" was "any tumor" in the original table, which would in theory encompass benign tumors.
2. Some solid malignant tumors (seminomas for example) have a fairly good prognosis, while others are rapidly fatal.
3. Both "leukemia" and "lymphoma" have low to high risk categories.
4. AIDS survival has improved considerably since 1987.
### NYHA- Heart failure classification

<table>
<thead>
<tr>
<th>Class</th>
<th>Patient Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I (Mild)</td>
<td>No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, or dyspnea (shortness of breath).</td>
</tr>
<tr>
<td>Class II (Mild)</td>
<td>Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, or dyspnea.</td>
</tr>
<tr>
<td>Class III (Moderate)</td>
<td>Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes fatigue, palpitation, or dyspnea.</td>
</tr>
<tr>
<td>Class IV (Severe)</td>
<td>Unable to carry out any physical activity without discomfort. Symptoms of cardiac insufficiency at rest. If any physical activity is undertaken, discomfort is increased.</td>
</tr>
</tbody>
</table>
## Appendix K  CCS– GRADING OF ANGINA

<table>
<thead>
<tr>
<th>Grade Description</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade I</td>
<td>Ordinary physical activity does not cause angina, such as walking and climbing stairs. Angina with strenuous or rapid or prolonged exertion at work or recreation</td>
</tr>
<tr>
<td>Grade II</td>
<td>Slight limitation of ordinary activity. Walking or climbing stairs rapidly, walking uphill, walking or stair climbing after meals, or in cold, or in wind, or under emotional stress, or only during the few hours after awakening. Walking more than two blocks on the level and climbing more than one flight of ordinary stairs at a normal pace and in normal conditions</td>
</tr>
<tr>
<td>Grade III</td>
<td>Marked limitation of ordinary physical activity. Walking one or two blocks on the level and climbing one flight of stairs in normal conditions and at normal pace</td>
</tr>
<tr>
<td>Grade IV</td>
<td>Inability to carry on any physical activity without discomfort, anginal syndrome may be present at rest</td>
</tr>
<tr>
<td></td>
<td>Available on the Canadian Cardiovascular Society Website at <a href="http://www.ccs.ca">www.ccs.ca</a></td>
</tr>
</tbody>
</table>
Appendix L  Normality test results

Normality tests were carried out for all the key variables at both baseline and 6 months (where appropriate). The results shown below provide an example of the results of the normality test completed for HADS anxiety, for the re-presentation group at baseline. Additionally a summary of the Shapiro-Wilk normality test results for each variable are also presented in the table below. The process of analysis used is described in section 5.13.1

HADS anxiety distribution at baseline: Re-presentation group

- On histogram the visual distribution for the HADS anxiety re-presentation group at baseline was a relatively normal bell-shaped curve (Figure L-1).
- The sample skewness was positive and not twice its standard error (SE) in magnitude indicating no evidence of non-normal distribution (see Table L-1).
- The sample kurtosis was negative but less than twice its SE in magnitude, indicating no evidence of non-normal distribution.
- The Shapiro-Wilk normality test was non-significant, indicating a normal distribution.
- Overall HADS anxiety re-presentation group at baseline is normally distributed.

Table L-1 Normality test results for HADS anxiety at baseline for the re-presentation group

<table>
<thead>
<tr>
<th>Normality test</th>
<th>Test result</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skewness</td>
<td>0.08</td>
<td>0.38</td>
</tr>
<tr>
<td>Kurtosis</td>
<td>-0.89</td>
<td>0.75</td>
</tr>
<tr>
<td><strong>Normality test</strong></td>
<td><strong>Test result</strong></td>
<td><strong>df</strong></td>
</tr>
<tr>
<td>Shapiro-Wilk</td>
<td>0.96</td>
<td>38</td>
</tr>
</tbody>
</table>
Figure L-1 The histogram demonstrating the distribution of the HADS anxiety baseline data for the re-presentation group.

Histogram

for potiHDrepdic = yes

HADba

Mean = 9.53
Std. Dev. = 4.836
N = 38
Table L-2 Summary of the Shapiro-Wilk normality test results for the key variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Time point</th>
<th>Re-presentation group</th>
<th>Summary of normality results</th>
<th>Non-representation group</th>
<th>Summary of normality results</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>W</td>
<td>df</td>
<td>p value</td>
<td>Normality results</td>
</tr>
<tr>
<td>HADS anxiety</td>
<td>baseline</td>
<td>0.96</td>
<td>38</td>
<td>0.241</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td>6 months</td>
<td>0.98</td>
<td>37</td>
<td>0.601</td>
<td>Normal</td>
</tr>
<tr>
<td>HADS anxiety change scores</td>
<td>baseline to 6 months</td>
<td>0.96</td>
<td>37</td>
<td>0.141</td>
<td>Normal</td>
</tr>
<tr>
<td>HADS depression</td>
<td>baseline</td>
<td>0.96</td>
<td>38</td>
<td>0.199</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td>6 months</td>
<td>0.94</td>
<td>37</td>
<td>0.034</td>
<td>Non-normal</td>
</tr>
<tr>
<td>HADS depression change scores</td>
<td>baseline to 6 months</td>
<td>0.98</td>
<td>37</td>
<td>0.792</td>
<td>Normal</td>
</tr>
<tr>
<td>HADS psychological distress</td>
<td>baseline</td>
<td>0.96</td>
<td>38</td>
<td>0.197</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td>6 months</td>
<td>0.96</td>
<td>37</td>
<td>0.157</td>
<td>Normal</td>
</tr>
<tr>
<td>HADS psychological distress change scores</td>
<td>baseline to 6 months</td>
<td>0.64</td>
<td>37</td>
<td>0.573</td>
<td>Normal</td>
</tr>
<tr>
<td>SAQ angina stability</td>
<td>baseline</td>
<td>0.83</td>
<td>37</td>
<td>&lt;0.001</td>
<td>Non-normal</td>
</tr>
<tr>
<td></td>
<td>6 months</td>
<td>0.77</td>
<td>34</td>
<td>&lt;0.001</td>
<td>Non-normal</td>
</tr>
<tr>
<td>SAQ angina stability change scores</td>
<td>baseline to 6 months</td>
<td>0.95</td>
<td>37</td>
<td>0.125</td>
<td>Normal</td>
</tr>
<tr>
<td>SAQ angina frequency</td>
<td>baseline</td>
<td>0.88</td>
<td>37</td>
<td>&lt;0.001</td>
<td>Non-normal</td>
</tr>
<tr>
<td></td>
<td>6 months</td>
<td>0.81</td>
<td>34</td>
<td>&lt;0.001</td>
<td>Non-normal</td>
</tr>
<tr>
<td>SAQ angina frequency change scores</td>
<td>baseline to 6 months</td>
<td>0.92</td>
<td>34</td>
<td>0.014</td>
<td>Non-normal</td>
</tr>
<tr>
<td>SAQ physical limitation</td>
<td>baseline</td>
<td>0.93</td>
<td>37</td>
<td>0.028</td>
<td>Non-normal</td>
</tr>
<tr>
<td></td>
<td>6 months</td>
<td>0.96</td>
<td>34</td>
<td>0.179</td>
<td>Normal</td>
</tr>
<tr>
<td>SAQ physical limitation change scores</td>
<td>baseline to 6 months</td>
<td>0.95</td>
<td>34</td>
<td>0.097</td>
<td>Normal</td>
</tr>
<tr>
<td>SAQ treatment satisfaction</td>
<td>baseline</td>
<td>0.79</td>
<td>38</td>
<td>&lt;0.001</td>
<td>Non-normal</td>
</tr>
<tr>
<td></td>
<td>6 months</td>
<td>0.78</td>
<td>37</td>
<td>&lt;0.001</td>
<td>Non-normal</td>
</tr>
<tr>
<td>SAQ treatment satisfaction change scores</td>
<td>baseline to 6 months</td>
<td>0.93</td>
<td>34</td>
<td>0.028</td>
<td>Normal</td>
</tr>
<tr>
<td>SAQ quality of life</td>
<td>baseline</td>
<td>0.96</td>
<td>38</td>
<td>0.176</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td>6 months</td>
<td>0.96</td>
<td>37</td>
<td>0.186</td>
<td>Normal</td>
</tr>
<tr>
<td>SAQ quality of life change scores</td>
<td>baseline to 6 months</td>
<td>0.98</td>
<td>34</td>
<td>0.686</td>
<td>Normal</td>
</tr>
<tr>
<td>Age</td>
<td>baseline</td>
<td>0.97</td>
<td>31</td>
<td>0.638</td>
<td>Normal</td>
</tr>
<tr>
<td>Troponin 'T'</td>
<td>baseline</td>
<td>0.75</td>
<td>31</td>
<td>&lt;0.001</td>
<td>Non-normal</td>
</tr>
<tr>
<td>GRACE score</td>
<td>baseline</td>
<td>0.89</td>
<td>31</td>
<td>0.005</td>
<td>Normal</td>
</tr>
<tr>
<td>ESSI score</td>
<td>baseline</td>
<td>0.83</td>
<td>38</td>
<td>&lt;0.001</td>
<td>Non-normal</td>
</tr>
<tr>
<td>CCI score</td>
<td>baseline</td>
<td>0.73</td>
<td>31</td>
<td>&lt;0.001</td>
<td>Non-normal</td>
</tr>
</tbody>
</table>

*Re-presentation group  
**Non-representation group
Appendix M Interview schedule

TOPIC AREAS TO BE DISCUSSED:

Experience of re-presentation to acute services or hospitalisation:
- Route of representation (acute services)
- How did they feel at the time of representation
- What they believed to be the cause of symptoms
- How did symptoms compare to STEMI
- Who called for help
- If someone else called for help would they have done so and why?
- Investigations
- How long admitted for?
- Diagnosis

The Future:
- Could they differentiate between symptoms again in the future?
- How do they think the staff viewed them?
- What do they think would have helped them to differentiate between symptoms?
- Information received relating to symptoms and diagnosis
- Were they given advice for the future?
- Attitudes towards final diagnosis and participants level of understanding.

Support received post PPCI:
- Did anyone give them verbal or written info in hospital?
- Were they invited to cardiac rehab?
- Did they attend?
- If so was it helpful or why didn’t they attend?

Is there anything that I have missed in our discussion that you consider to be important?
Interview 14, patient 184:

M1  Err, Sunday night started feeling a bit dodgy, erm, woke up on the Monday morning to go to work, I thought it's, you know, it'd be something or nothing, woke up on the Monday morning ready to go to work and I just felt lousy, I felt like I was having another heart attack.

F1  Right okay.

M1  Err, so I phoned the ambulance up, they took me in and what had happened, apparently when I had the initial heart attack, there was a major artery and one of the minor arteries that had been blocked…

F1  Mm.

M1  … and they opened both of those and the minor artery is blocked again and that's what it was.

F1  Aha.

M1  I've since had an angiogram and er they've decided not to do anything with it because it's not life threatening……….but it's very debilitating.

F1  Ah right.

M1  You know, I've not been able to go back to work and to top it all, two weeks ago, they just stopped the benefits…………so I've got no money at all.

F1  Oh.

M1  So I've had… it's been, erm, this may seem a bit and I mean, I've gone from, like, in February from somebody who used to go to the gym three times a week to not being able to work, to taking tonnes of bloody pills a day…

F1  Mm.

M1  … to losing my business.

F1  Mm.

M1  Erm, having credit card companies chasing me, to having my car repossessed, to having my benefits stopped completely and I just… and now I'm actually on bloody antidepressants from the doctor as well now so, you know, it's just been… it may seem ungrateful and initially I was really grateful that, you know, the fantastic efforts of everybody and the technology involved and the expertise involved saved my life.
Appendix O Interview notes (excerpt)

Interview 14, patient 184 M-C

Age: 49
Gender: Male
Interview place: Patients home
Marital status: Lives alone
Family members: Mother, father, son and daughter (doesn't cohabit with any of them).

Prior to Interview

During the 6 month follow-up phone call with the participant, the individual spoke of having suffered a readmission to hospital due to chest pain. He stated that he thought that he was having another heart attack. He also spoke about suffering an enormous amount of stress due to financial difficulties which had occurred since his MI and particularly the re-presentation. The participant reported starting medication for the treatment of depression and experiencing palpitations and chest pains which he thought may be related to the stress that he was under.

Environment

The participant’s home was in a rundown part of the city in large tower block of flats. Arriving at the flats was quite intimidating and it did not feel particularly safe. I was fearful of leaving my car in the car park despite the car park being behind steel electronic gates. Inside the flat it was poorly decorated with bare plaster on the walls. The flat smelt strongly of smoke.

Co morbidity

The participant did not have any comorbid conditions.

Interview

Initially I felt uncomfortable as the individual seemed very angry about his plight. During the interview he constantly tried to steer the topic back to his financial problems. This included the difficulties and injustices that he had suffered at the hands of the Department of Work and Pensions (DWP) and the insurance company that had refused to pay-out on an insurance policy (to cover sickness) that he had taken out a number of years earlier because he was self-employed.

The participant stated that initially after discharge following his heart attack he had felt elated and was grateful for receiving lifesaving treatment. He was very positive about attending cardiac rehab and made a steady and successful recover and this was despite some financial problems. However he was not unduly troubled by his finances as he believed that once he returned to work he would very quickly clear his debts. Four months following his heart attack he felt well and was ready to return to his work as a laminate floor fitter. He described this as
a physically demanding job requiring the ability to carry heavy materials, to
crouch and the need to physically exert himself for long periods of time.

At the point that he was about to return to work he suffered an episode of chest
pain, which he believed to be another heart attack. He presented to A&E via the
emergency ambulance service and was admitted to hospital. Following
investigations he was given the diagnosis ‘angina’.

During the interview it became clear that this event completely undermined the
individuals’ confidence to the point that he no longer felt able to work. He was
fearful of suffering another heart attack that he thought would end his life, this
was to the point that he believed he would have another event and that it was
only a matter of time (when not if). Due to his inability to work his financial
situation had declined further to the point that he felt he may have to declare
himself bankrupt. Since his heart attack he had gone from living a very
comfortable lifestyle, with foreign holidays and owning a sports car to living on
£50 per week and being pursued for money by multiple sources. He explained
that the small amount of benefit that he received from the DWP had just been
stopped because the DWP had demanded that he should present himself to the
job centre as fit for work. He felt fatigued and described suffering symptoms of
anxiety and depression and did not feel able to work. He was extremely angry
and felt that he had been unfairly treated. He said that the DWP had not asked
his Cardiologist for a clinical opinion or report of his physical ability to work. At
times the individual became so angry that he raised his voice and used offensive
language to express himself. At the time of the interview he was being financially
supported by his parents. The participants spoke of the excellent medical
treatment that he had received and believed that it had been a waste of time
because he now had no life to live.

This was a difficult interview and shared some similarities with interview 12 in
that patient 192 D-C was also suffering severe financial difficulties due to her
illness and inability to work. Both participants reported the insensitivities of the
DWP and the fact that they had not been allowed to ‘be sick’. There was
commonality between the two in that they felt aggrieved not only that they had
been badly treated by the State but also that they had not been afforded the sick
role by society. Both stated that they had worked since being teenagers and had
paid into the system without ever previously needing financial help from the
State. They felt that when they did need the financial support due to a valid and
legitimate reason (a heart attack) the State had let them down. Both individuals
were extremely angry and distressed. They had also both become depressed and
were receiving medication and reported experiencing classic somatic symptoms
associated with anxiety.
Appendix P Qualitative diary (excerpt)

11 08 09- When I started to code the interviews I developed lots of codes, because I was afraid that I would miss something important and relevant later on. When you start coding obviously you only have a theoretical framework a few interviews and your intuition to base the coding framework on. Therefore initially I held off trying to shape the coding and tried to go with the data rather than restricting myself and trying to force the data to fit with the codes that I’d previously developed from coding other interviews. Very quickly, interviews one, two, three and four it was very apparent that depression, shock at having suffered a STEMI and uncertainty relating to symptoms were important themes. During interview two, anger verging on hostility became apparent and this was seen again in interviews 8 and 12.

As I am progressing through the study my interview techniques are improving. Listening to interviews after the event, has helped me to focus the questioning and develop new areas of interest and follow-up on developing topics. Initially I was apprehensive about asking about sensitive issues such as thoughts of death even though the interviewee had raised the matter by hinting at the topic. Interviewee 2 spoke of financial problems since STEMI and I decided to add this to the interview schedule. After interview 4 I have also added thoughts of death to explore this topic with subsequent interviewees.

21 03 09- I am now in the process of coding interview 17 GG and I have decided that I have far too many codes to handle! Originally I set up tree nodes under STEMI, representation and symptoms amongst others, but as I have worked my way through coding I have realised that the majority of patients do not fit into these precise ‘boxes’. Many have suffered symptoms prior to their STEMI and have gone on to associate these symptoms with their STEMI (i.e. interviewee 17 GG & interview 16 89 JP). Others have suffered multiple readmissions (interviewee 2), for elective PCI or due to symptoms that they thought were related to the heart but were indigestion etc. On a number of occasions they have been discharged from hospital and therefore I have decided to code all discharges for all admissions (pre PPCI, PPCI and other admission) under the same code (discharge) because once charted I will be able view the categories and see if there are any commonalities. This is the same for calling an ambulance.

The following recoding was done:

1. STEMI acute event- STEMI ambulance service not used merged with - Representation- Representation didn’t call for ambulance.
3. Representation- psychological cause of readmission merged with, negative cardiac investigation findings merged with, Readmission diagnosis merged with, non-cardiac representation diagnosis & merged with, No readmission diagnosis & merged with uncertain of readmission diagnosis- Title changed to Diagnosis (these were all merged because they were all related to a diagnosis).
Appendix Q  Thematic framework in Nvivo8 (screenshot)
Appendix R Subthemes in Nvivo8
(screenshot)
### Appendix S

**The anxiety and depression chart (excerpt)**

<table>
<thead>
<tr>
<th>Codes</th>
<th>Interview 21</th>
<th>Interview 22</th>
<th>Interview 23</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male, 54 years</td>
<td>Male, 27 years</td>
<td>Male, 54 years</td>
</tr>
<tr>
<td>2.01</td>
<td>Anxiety</td>
<td>- Cause of chest pain &amp; may have another STEMI whilst driving (pg 2, 46-51 &amp; pg 3, 1-29: pg 7, 32-45)</td>
<td>- Uncertain why gets chest pains &amp; tiredness; thinks it is stress (pg 17, 22-50)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Believed needed more psychological help when in-patient after CABG (pg 38, 8-38)</td>
<td>- Uncertainty cause of chest pain (cardiac or panic attack), thinks he's having a heart attack when has panic attack (pg 2, 7-49: pg 3, 25-35: pg 4, 35-49 &amp; pg 5, 1-11: pg 20, 13-20)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Worried about other STEMI whilst driving (pg 22, 1-14)</td>
<td></td>
</tr>
<tr>
<td>2.02</td>
<td>Distressed</td>
<td>- Distressed that had had another STEMI and that it might happen again (pg 18, 16-43)</td>
<td>- An event in his past causes him stress when he remembers it (pg 16, 1-16)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Pain was worse this time than his other heart attacks (pg 5, 1-9)</td>
<td>NIL</td>
</tr>
<tr>
<td>2.03</td>
<td>Fear</td>
<td>- Didn't understand sharp pain in chest (pg 18, 13-45)</td>
<td>- Afraid of heart disease. Frightened that a heart attack will kill him (pg 3, 15-25)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Thought about death early on, just after he had his heart attack (pg 10, 1-20)</td>
<td></td>
</tr>
<tr>
<td>2.04</td>
<td>Thoughts of death</td>
<td>- Post STEMI became depressed due to thoughts of what might</td>
<td>- When he has panic attack thinks he is dying from a heart attack. Worried that he might die from a STEMI (pg 4, 28-32)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Feeling disempowered</td>
<td>NIL</td>
<td>- Feels trapped. Has to stay in UK to earn money to feed Mother &amp; siblings back in Pakistan (pg 21, 1-51)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Expected not to be anxious in his work role. Believe panic attacks undignified, humiliating, can't control them (pg 4, 28-33: pg 34, 1-4)</td>
</tr>
<tr>
<td>2.06</td>
<td>Depressed</td>
<td>- Felt depressed because he had had another MI and was worried about having another one (pg 17, 10-40)</td>
<td>- Doesn't feel depressed. Feels quite happy in life (pg 29, 4-14: pg 5, 13-26)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>NIL</td>
<td></td>
</tr>
<tr>
<td>2.07</td>
<td>Feelings of failure</td>
<td>- Still smoking - unable to stop despite numerous attempts to stop (pg 47, 50-51 &amp; pg 47, 1-30)</td>
<td>- That he suffers panic attacks and that he can't control or deal with his anxieties in another way (pg 33, 1-24)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>NIL</td>
<td></td>
</tr>
<tr>
<td>2.08</td>
<td>Loss of confidence</td>
<td>- In driving, in case he has a STEMI whilst driving &quot;It's (worry of having STEMI whilst driving) stopped us doing a lot of things&quot; (pg 22, 1-14)</td>
<td>- Didn't have confidence in healthcare services (A&amp;E) &amp; discharged himself (pg 7, 15-25)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Worried about dying, isn't frightened of dying but isn't ready to die (pg 4, 28-32)</td>
<td>- Won't travel because worried about not being near a good hospital (pg 15, 12-16)</td>
</tr>
<tr>
<td>2.09</td>
<td>Treatment for depression</td>
<td>- Refused medication from GP, didn't receive any other form of treatment. Got better over time (pg 17, 10-40)</td>
<td>NIL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>NIL</td>
<td>NIL</td>
</tr>
<tr>
<td>2.10</td>
<td>Worried about the future</td>
<td>- Worries about having another STEMI whilst driving (pg 22, 1-14)</td>
<td>- Two arteries blocked; only one unblocked at time of PPCI. Worried about other blocked artery &amp; what may happen, not sure if completely or 70% blocked (pg 9, 6-49)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Worried about dying, isn't frightened of dying but isn't ready to die (pg 4, 28-32)</td>
<td>- Worries about dying, isn't frightened of dying but isn't ready to die (pg 4, 28-32)</td>
</tr>
<tr>
<td>2.11</td>
<td>Suffered anxiety and depression previously</td>
<td>- Once when out of work 20 yrs ago (pg 31, 24-38)</td>
<td>- Wife believes he suffers underlying anxiety (pg 30, 26 &amp; pg 31, 5-25)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Worried about unpleasant things that happened in the past, prior to STEMI too (pg 16, 1-16)</td>
<td>- Being too far from a hospital stops him travelling (pg 15, 9-20)</td>
</tr>
</tbody>
</table>
Appendix T Qualitative analysis- photographs demonstrating the processes undertaken

Photograph demonstrating the charting phase of framework analysis

Photograph demonstrating the mapping and interpretation phase of Framework analysis.
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