# PATIENTS' UNDERSTANDING OF HEART DISEASE:

# RELATIONSHIPS WITH DECISIONS TO SEEK HELP WITH ACUTE SYMPTOMS, AND WITH ADHERENCE TO TREATMENT

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**Abstract**

Pre-hospital delay in seeking help for acute chest pain in patients with coronary heart disease is a major impediment to prompt thrombolysis. Failure to adhere to medication, attend cardiac rehabilitation where appropriate, and change lifestyle, all impair secondary prevention. This thesis examined psychological factors related to these problems, and the psychological models of illness held by patients diagnosed with acute coronary syndromes (ACS). Two main issues were investigated; firstly, what factors were associated with shorter pre-hospital delays following symptom onset; and secondly, whether cognitive models of illness predicted adherence to advice, psychological and emotional adjustment, and quality of life at 3 months and 13 months post-discharge.

Data were collected from 269 patients diagnosed with ACS within five days of hospital admission. Analyses were focussed on the total time between symptom onset and admission to hospital (pre-hospital delay). This interval was divided into two phases; time between symptom onset and decision to call for medical help (patient decision time), and time from call for help to admission (home to hospital delay). Patients were followed up 3 and 13 months later. Adherence to medical advice (lifestyle changes, adherence to medication, attendance at cardiac rehabilitation programmes), psychological distress and quality of life were measured by telephone interview and questionnaire.

A number of sociodemographic, social, clinical and psychological factors were associated with pre-hospital delay. Beliefs about the causes of heart disease made an important contribution. Cognitive representations of heart disease measured during hospital admission did not predict adherence to treatment regimens after discharge, but significantly predicted later psychological and emotional adjustment, and quality of life. Theses findings have implications for understanding the contribution of psychological factors to the experience of acute heart disease, and point to methods of more effective patient care and management.

Dedicated to my husband, Gabriel.

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# Chapter 1: Introduction

## 1.1: The prevalence of coronary heart disease

Coronary heart disease (CHD) is the most common cause of premature death in the UK. In 2002, 270 000 people suffered a heart attack (also known as an acute myocardial infarction or AMI) in the UK resulting in 117, 000 deaths, 81 000 of which occurred outside hospital. In terms of mortality from all causes, coronary heart disease alone was responsible for 22% of premature deaths in men and 17% in women (British Heart Foundation, 2002). The UK Heart Attack study reported that as many as one third of patients suffering from acute coronary events died before admission to hospital (Norris, 1998).

Despite recent improvement, the death rate from coronary heart disease in the UK remains among the highest in the world. The British Heart Foundation estimated that over 1.3 million people (about 850,000 men and 450,000 women) living in the UK had suffered from a heart attack. The prevalence of angina has been estimated at 5% of men and 3% of women. This results in huge costs to the health care system in terms of hospital care and drug treatment of around £3,500 million a year, and costs to the UK economy of about £3,100 million because of days lost due to death, illness and informal care of people with the disease (British Heart Foundation, 2005a). Coronary heart disease also has a long lasting impact on affected individuals and their families in terms of physical and psychological adjustment and quality of life.

## 1.2: Risk factors for coronary heart disease

There are a number of risk factors have been identified which predispose to the development of cardiovascular disease (CHD and stroke). Smoking is a major risk factor. It is estimated that over 30,000 deaths from cardiovascular disease (CVD) a year in the UK are caused by smoking, and that regular exposure to second-hand smoke increases the risk of CHD by about 25% (British Heart Foundation, 2005a). Approximately 28% of men and 24% of women smoke. The highest rate of smoking is among adults aged 20-34 years (declining with age) and among people from manual social groups. Poor diet (including high fat and salt intake and low consumption of fruit and vegetables) is also an important risk factor. Again, people from a poor socioeconomic background are at increased risk in the UK because they generally have a lower consumption of fruit and vegetables than people who are on higher incomes. Regular physical activity lowers the risk of CHD, but only 37% of men and 24% of women in the UK do enough exercise to meet current recommendations.

It is estimated that about 43% of men and 33% of women in England are overweight, and a further 22% of men and 23% of women are obese (British Heart Foundation, 2005). Being overweight is associated with raised blood pressure, non-insulin dependent diabetes and low levels of physical activity all of which have an increased risk of CHD. Moderate alcohol intake is associated with reduced risk of CHD, however women are advised not to drink more than 14 units per week and men not more than 21 units per week. In the UK, it is estimated that about 27% of men and 17% of women drink more than this, and binge drinking among younger age groups is currently recognised as a serious health issue. People with type 2 diabetes are at substantially higher risk of heart disease. Currently about 4% of men and 1% of women in the UK have been diagnosed with diabetes. Hypertension (blood pressure of 140/90mmHg or

more) is also an important risk factor for CHD affecting about one third of the population of England, whilst about 66% of people have blood cholesterol levels above the recommended level (5.0 mmol/l).

A number of psychological factors, such as lack of social support, work stress, depression, anxiety and personality factors (particularly hostility) have also been associated with increased risk of CHD (British Heart Foundation, 2005a). Depression has been associated with a 3-4 fold increase in cardiac mortality over the first 18 months following an MI (Frasure-Smith et al, 1995a). Life stress and social isolation along with depression are related to morbidity and mortality following diagnosis of ACS (Barefoot et al, 2000).

## 1.3: The development of coronary heart disease

This thesis will focus specifically on two acute forms of coronary heart disease, acute myocardial infarction (AMI), and unstable angina (UA) which are caused by atherosclerosis of the blood vessels and jointly described by the term 'acute coronary syndromes' (ACS). Atherosclerosis was, until recently, thought to be a slowly progressive, degenerative disease causing symptoms through its mechanical effect on blood flow, particularly in the small calibre arteries supplying the myocardium and brain. Recent research, however, has shown it to be a dynamic inflammatory process (Weissberg, 2000). The initial abnormality is a fatty streak visible macroscopically on the endothelial surface of the artery and caused by an accumulation of lipids and macrophages. This develops into a mature atherosclerotic plaque, made up of a central lipid core bounded by an endothelialized fibrous cap containing vascular smooth muscle cells and connective tissue, particularly collagen. As the plaque grows, the vessel expands preserving both the lumen diameter and the blood flow (this known as positive

remodelling) (Davies et al, 2004). Large atherosclerotic lesions can accumulate without compromising blood flow or producing symptoms, indeed angiographic findings show that most culprit lesions are not flow limiting.

Atherosclerosis remains clinically silent until one of two events. The lesion either expands to the point at which it limits blood flow, producing symptoms of reversible ischaemia during periods of exertion or demand (angina), or the fibrous plaque ruptures causing the exposure of sub-endothelial collagen and lipid. The latter initiates activation of circulating platelets and clotting cascade proteins, leading to the development of 'vulnerable blood'. This term describes a condition in which platelets are prone to activation, the coagulation system is in a prothrombotic state, and high levels of circulating inflammatory factors are present (Naghavi et al, 2003). This may result in the formation of a thrombus, composed of both fibrin and platelets, through the activation clotting factor proteins via the intrinsic and extrinsic coagulation pathways. The consequences of plaque rupture may therefore vary widely ranging from complete lysis of the thrombus by endogenous fibrinolytic pathways with the subsequent healing of the fibrous cap and overlying endothelium, to complete occlusion of the lumen by the thrombus. Severity may range from being clinically silent to producing an acute vascular event such as unstable angina, AMI, stroke or sudden death (Davies et al, 2004).

An AMI occurs when the flow of blood through the coronary arteries is reduced to such an extent that the heart muscle is damaged or dies. This often occurs suddenly, commonly producing symptoms such as central crushing chest pain, a feeling of heaviness/discomfort in the left arm, and collapse. Other symptoms patients have described may also include; shortness of breath, nausea and/or vomiting, diaphoresis,

palpitations, feeling faint, dizzy or weak, indigestion–like abdominal discomfort, stomach upset, flu-like symptoms, pain in the jaw/head/shoulder/back, or feelings of panic and/or impending doom. Early medical treatment following the onset of symptoms of AMI is essential due to the high risk of fatal arrhythmias and to effectively limit damage to the heart muscle.

## 1.4: Treatment for ACS

The advent of thrombolytic therapy has revolutionised the treatment of ACS over the last 20 years by enabling blood flow to be restored to the myocardium. Thrombolytic therapy is administered intravenously and dissolves the thrombus causing the obstruction. The impact of thrombolytic therapy on mortality and morbidity was shown to be significant by several large clinical trials (ISIS-2, 1988b; ISIS-3, 1992). Patients who received thrombolytic therapy within the first hour after symptoms began (known as the 'golden hour') were shown to be 50% more likely to survive the first year following an AMI (GISSI, 1986).

Coronary angioplasty is an alternative procedure also used to relieve obstruction or reduce narrowing in coronary arteries. It consists of a small balloon catheter inserted into the artery and advanced to the narrowing where the balloon is inflated and removed leaving in place a rigid support (stent) to keep the blood vessel open. The use of this technique as the first choice of treatment in ACS is known as primary angioplasty and some researchers have found it to be superior to treatment by thrombolysis (Andersen et al, 2003; Jacobs, 2003). The UK Government is currently planning to investigate the feasibility of introducing a national service able to provide primary angioplasty (Department of Health, 2004).

Since the therapeutic benefit diminishes over time, the effectiveness of thrombolytic therapy and primary angioplasty are dependent on the treatment being initiated as quickly as possible. Reperfusion therapy within the first hour promotes maximal myocardial salvage, but administration within 6 hours can also significantly reduce infarct size and mortality (ISIS-3, 1992), and the benefits of thrombolysis are evident up to 12 hours after symptom onset (Fibrinolytic Therapy Trialists' Collaborative Group, 1994). The Worcester Heart Attack Study, however, showed that patients arriving at hospital within one hour of symptom onset were six times more likely to receive thrombolytic therapy than patients presenting more than 6 hours after onset (Gurwitz et al, 1997).

Despite these findings, only 25% of patients suffering symptoms of ACS call for medical help within one hour of symptom onset, and 40% of patients wait more than 4 hours before seeking help (Goff, Jr. et al, 1999; Gurwitz et al, 1997; Leslie et al, 2000). There is considerable variation in the time interval between onset of symptoms and hospital admission, ranging between 1.7 and 7 hours, with longer delay times leaving patients at higher risk of fatal arrhythmias and increasing their risk of extensive myocardial damage (Ottesen et al, 2004). Most studies report median pre-hospital delay times between 2 to 4 hours (Goldberg et al, 2002; Horne et al, 2000; Leslie et al, 2000). Evidence suggests that pre-hospital delay times have remained relatively constant over the last two decades and is not adequately explained by severity of cardiac illness (Goldberg et al, 1999). Thus, despite the availability of thrombolytic treatment and more recently primary angioplasty, pre-hospital delay has remained unacceptably long.

Reducing pre-hospital delay time is now widely recognised as a crucial step in reducing mortality from AMI. The greatest impediment to shorter delays is thought to be patient

decision time, the time taken for the patient to recognise that their symptoms are serious and to decide to call for medical help. Patient decision time has been described as the 'weakest link in the chain of survival' (Penny, 2001) and accounts for up to 80% of total delay time from onset of symptoms to arrival at hospital (GISSI, 1995). Interventions to reduce delay have not been very successful to date, so it is important to understand better the factors contributing to delay.

## 1.5: Adjustment, psychological wellbeing and quality of life following ACS

Patients who survive cardiac events remain at high risk for future AMI or stroke. There is strong evidence that secondary prevention to reduce cardiovascular risk reduce morbidity and mortality. Unfortunately, risk factors remain poorly controlled in many survivors of ACS, and one of the factors which contributes to this is poor adherence to treatment recommendations by patients. There is evidence that fewer than 50% of patients recommended to attend cardiac rehabilitation programmes actually do so (Lane et al, 2001b), less than 50% of smokers quit after an AMI and adherence to other recommended lifestyle changes is poor (Wood, 2001).

Psychological adjustment following ACS may be an important factor affecting patients' quality of life following an ACS. Anxiety and depression have been found to be significant predictors of poorer quality of life in patients following AMI (Lane et al, 2001). Depression has been consistently linked to non-adherence to medical treatment recommendations (Guiry et al, 1987; Ziegelstein et al, 2000). Reviews of clinical and experimental studies have reported that mental stress (acute, sub-acute or chronic) increases the risk of ischemia, MI or death in patients with established ischemic heart disease (Januzzi, Jr. et al, 2000; Kubzansky & Kawachi, 2000). Ischemic complications

following AMI resulting from anxiety are increased from 2.5 – 5 times that of non-anxious patients (Frasure-Smith et al, 1997; Moser & Dracup, 1996). Poor quality of life has been shown to predict mortality and morbidity among cardiac patients (Rumsfeld et al, 1999). It is possible that patients' beliefs about the causes of their heart problems influence their psychological wellbeing (anxiety and depression) and impact on quality of life following ACS. Illness beliefs have been shown to predict attendance at cardiac rehabilitation programmes and return to work (Petrie et al, 1996), adherence to medication (Horne & Weinman, 1999) and may also be involved in behaviour change post AMI (Byrne et al, 2005; Weinman et al, 2000).

Patients' causal attributions may play an important role in adjustment following diagnosis of ACS. If patients have inaccurate beliefs about the causes of their heart disease and inaccurate perceptions of their personal health risks, they may underestimate the relevance of advice given to them by medical staff. Communication may be less effective if patients have different models of cause from clinicians. This may result in patients making inaccurate attributions or neglecting to make attributions to particular risk factors that affect them personally, and thus affect their response firstly to their symptoms at onset, and secondly to secondary prevention and making lifestyle changes, which in turn may affect their quality of life.

## 1.6: Outline of the two clinical problems to be investigated

The aim of this thesis is to investigate two important clinical problems;
Firstly, it will investigate the problem of pre-hospital delay by examining clinical, socio-demographic, and psychosocial characteristics of a prospective cohort of patients admitted to hospital with a diagnosis of ACS. It will also investigate patients' beliefs

about the causes of their heart problem and the role these beliefs may play in pre-hospital delay.

Secondly, it will investigate associations between patients' causal beliefs and adjustment up to 13 months following the ACS, including adherence to clinical recommendations (prescribed medication, attendance at cardiac rehabilitation programme if appropriate and lifestyle changes), psychological wellbeing (anxiety and depression) and quality of life.

## 1.7: Aims of this thesis

1. To investigate the socio-demographic and psychological factors which predict delay in contacting medical help following the onset of symptoms of ACS.
2. To investigate the relationship between patients' health beliefs and their decision to seek help following the onset of symptoms of ACS.
3. To investigate the relationship between patients' health beliefs and adherence to medical advice 3 months and 13 months after hospital discharge, and to identify factors which may predict non-adherence.
4. To investigate the relationship between patients health beliefs and adjustment and quality of life 3 months and 13 months after hospital discharge.

## 1.8: Structure of this thesis

The literature review in chapter 2 will define pre-hospital delay and its constituent phases. The common factors that have been shown to influence pre-hospital delay will be discussed, including socio-demographic and clinical factors, previous medical history, situational and psychosocial factors. It will also describe a number of intervention studies which have attempted to reduce delays. Finally, it will describe the

psychological models that have been used to help explain patients' perceptions of illness and how this may relate to pre-hospital delay. Chapter 3 describes the aims, hypotheses, and methodology of the present study, and findings relating socio-demographic, clinical and psychosocial characteristics with delays between symptom onset and hospital admission for ACS are presented.

Chapter 4 is a literature review of previous studies that have investigated causal attributions in relation to pre-hospital delay. Chapter 5 outlines the methodology used in this study to investigate patients' causal attributions and presents the results of these analyses.

Chapter 6 addresses the problems of adjustment over the months following discharge from hospital, including adherence, psychological well being and quality of life. It reviews literature about how these issues relate to causal attributions. Chapter 7 describes the methodology used to investigate specific behaviour changes, adherence, psychological adjustment (depression and anxiety), and quality of life in relation to causal attributions at 3 months and 13 months following ACS. Analyses of 3 month results are presented. Chapter 8 presents the results of analyses carried out on follow up data collected after 13 months and discuss the findings.

Chapter 9 is a general discussion of the findings of this study and their implications for research and clinical practice. It also reviews the strengths and weaknesses of the study, and suggests areas that might be developed for further research.

# Chapter 2: Literature review of pre-hospital delay

## 2.0: Introduction

The first aim of this thesis is to investigate the socio-demographic and psychological factors which predict delay in contacting medical help following the onset of symptoms of ACS. I hypothesize that shorter patients' decision time in seeking help will be associated with demographic and psychosocial variables including younger age, male gender, greater social support, higher socio-economic status, time of onset on a week day and within work hours, the presence of a bystander, attribution of symptoms to heart attack and low cardiac denial. In this chapter I will review previous research into pre-hospital delay with particular emphasis on factors that have previously been found to predict delays.

## 2.1: Trends and range of pre-hospital delay

Trends in pre-hospital delay have changed little in over a decade, despite the widespread use of thrombolytic therapies and angioplasty. Two large cross sectional American studies carried out retrospective reviews of AMI patients medical records. Goldberg et al (1999) reviewed 364 131 medical records of AMI patients from 1994 to 1997 for the Second National Registry of Myocardial Infarction and found no change in median pre-hospital delay (2.1 hours). McGinn et al (2005) reviewed 18 928 AMI patients and reported no statistically significant change in the proportion of patients delaying $\geq$4 hours from 1987 to 2000. The Worcester Heart attack study was a longitudinal study which examined trends in pre-hospital delay from 1986 to 1997 (Goldberg et al, 2000b) and also reported very little change in duration of delay during this period; the mean and median pre-hospital delays were 4.1 and 2.2 hours respectively in 1986 and 4.3 and 2.0 hours in 1997.

Previous studies have shown considerable variation in pre-hospital delay ranging between 1.7 and 7 hours (Ottesen et al, 2004). The ISIS-2 (1988a) and ISIS-3 (1992) studies showed median pre-hospital delays of 5 and 4 hours respectively, and the GUSTO Trial (1993) reported median times to treatment of 2hr 45 minutes. The UK Heart attack study reported that only 15% of patients came under hospital care within 1 hour, 54 % within 2 hours, 67% within 4 hours (Norris, 1998) . Other UK studies have also found median pre-hospital delay to be between 2-4 hours (Horne et al, 2000; Leslie et al, 2000). Thus, despite the availability of thrombolytic treatment and more recently primary angioplasty, patient delay has remained consistently unacceptably long. Longer delay times leave patients at higher risk of fatal arrhythmias, increases their risk of extensive myocardial damage and reduces the likelihood of them receiving thrombolytic therapy.

## 2.2: Definitions of pre-hospital delay and its constituent phases

The term pre-hospital delay usually refers to the time interval between the onset to symptoms and admission to hospital. Most studies have analysed pre-hospital as one time interval but a few have divided it into a number of separate phases for analysis. Labels given to these phases and definitions of the time intervals to which they refer has not been consistent between studies. Table 2.1 shows the definitions of pre-hospital delay, its constituent phases, mean and median delay times reported in previous studies.

For the purposes of this thesis, **pre-hospital delay** will be defined as the total time from onset of symptoms to admission to hospital; **patient decision delay** will describe the time interval between the patient first becoming aware of symptoms to deciding to call for medical help; **home to hospital delay** will describe the time period from the call for help to hospital admission. Mean delay times are often skewed due to a small number

of individuals who have very long delays, the median time may therefore be a more useful representation of average delay and will be quoted, when available, in this literature review. Patient decision delay has been reported by previous studies as making up 23% of the total pre-hospital delay in patients presenting within 2 hours of onset (GISSI, 1995) and from 60% to 80% in patients presenting later than this (GISSI, 1995; Schmidt & Borsch, 1990).

Time taken to for the emergency ambulance services to respond to calls for help and to transport patients to hospital has improved greatly in recent years. In the National Service Framework for Coronary Heart Disease (Department of Health, 2000), the UK Government made it a priority to reduce response time by ambulance services attending patients suffering symptoms of a heart attack victims to within 8 minutes from the call for help, and to transport patients to hospital within 30 minutes. In 2003/2004, the London Ambulance Service attended 76% of emergency calls within 8 minutes, ambulance response time is therefore unlikely to play a role in causing prolonged pre-hospital delays in treatment of cardiac patients (British Heart Foundation, 2005a).

**Table 2.1: Definition and average delay times of pre-hospital delay and its constituent phases**

| Authors | Design & sample size | Phases & definitions of delay | Average delay times |
|---|---|---|---|
| Berglin Blohm M et al (1998) Sweden | Prospective computerised timed intervals 2019 AMI pts | **Pre-hospital delay** – pain onset to hospital admission<br>**In-hospital delay**- hospital admission to CCU admission<br>**Onset of pain to thrombolysis**<br>**Door to needle time**- hospital admission to thrombolysis | Median - 2 hrs15 mins<br>55 mins<br>2h 50 mins<br>55 min |
| Bourma J et al (1999) Netherlands | Descriptive 3 centre study interview/ questionnaire 400 AMI pts under 70 years old | Pre-hospital delay time<br>**Patient delay** –onset to call for help<br>**Doctor delay** – call for help to call for ambulance<br>**Ambulance delay** – call for ambulance to hospital admission | Median values:<br>30 mins<br>38mins<br>35 mins |
| Carney R et al (2002) N.Ireland | Cross sectional 62 AMI patients | **Delay time** - symptom onset to seeking help. | Median 2hr 15 mins |
| Dracup K & Moser DK (1997) N.America | Multi centre descriptive survey 277 patients with AMI | **Delay**- time of onset to arrival at hospital | Mean 110 (±79) mins<br>Median 90 mins |
| Dracup K et al (1997) Sydney, Australia | 317 patients with AMI<br>Interview combined with review of medical records | **Pre-hospital delay** - onset of symptoms to arrival at hospital | Median 6.4 hours |
| Ell K et al (1994) Los Angeles, USA | Structured interviews 254 African American pts admitted to a public hospital & 194 African American pts admitted to private hospital for AMI | **Decision logistics phase** – time from onset to decision to seek emergency care<br>**Travel logistics phase** -decision to seek care to hospital admission<br>**Total Pre-hospital delay** – (decision phase + travel phase) | Mean 9.14h, Median 1.90<br>Mean 5.74, Median 0.75h |
| GISSI (1995) | Multi-centre case-control study 5301 pts | **Delay** – onset to hospital treatment in CCU<br>**Decision time** – onset to decision to seek help<br>**Home to hospital** – help seeking to arrival at A&E.<br>**In-hospital time** – arrival at A&E and treatment | Mean 8 hrs 15 mins<br>Median 3 hrs 50mins |
| Goldberg RJ et al (2002) N.America, Europe, Australia, NZ | Population based approach - retrospective review of medical records<br>Total:10 582 patients<br>3693 STEMI<br>2935 NSTEMI<br>3954 UA | Onset of symptoms to hospital admission0 | Median delay times<br>3.0 hrs NSTEMI<br>3.0 hrs UA<br>2.3 hrs STEMI |

| Authors | Design & sample size | Phases & definitions of delay | Average delay times |
|---|---|---|---|
| Grossman SA et al (2003) Boston, USA | Prospective observational 374 patients | **Lag time** - Onset of symptoms to hospital arrival | Mean 8.7h<br>Median delay 4h |
| Heriot AG et al (1993) London | Interview<br><br>103 MI patients under 75 years | **Total delay** - onset to hospital admission<br>**Decision time**- onset of chest pain to decision to seek help<br><br>**Response time** – decision to seek medical help to hospital arrival | Median - 2hours<br><br>30mins if contact GP<br>25 mins if go straight to A&E<br>GP involved- 160mins.<br>Ambulance – 82 mins<br>Own transport – 90 mins |
| Horne R et al (2000) UK | Structured interview 88 patients admitted to hospital with first MI | **Delay** –symptom onset to A&E arrival | Mean 7.3h (14.2)<br>Median 2.2h |
| Luepker RV et al (2000) USA | Randomised trial 59944 adults aged over 30 years with chest pain | **Patient delay** - symptom onset to hospital arrival | Median 140 mins |
| Matthews, K.A et al (1983) | Structured interview 43 patients admitted to CCU with suspected MI | **Total delay** – onset to decision to seek care<br>**Onset to illness decision**<br>**Illness decision to seeking care** | Mean & Median (in days):<br>Mean 11.1, Median 1.5day<br>Mean 10.0,Median 0.1 day<br>Mean 1.2, Median 0.1 day |
| McKinley S et al (2000) | Observational study 424 AMI patients | **Delay** - symptom onset to hospital arrival | Median 90 mins (both groups) |
| More R et al (1995) Brighton | Observational study 274 AMI patients | **Symptom onset to thrombolysis**<br>**Symptom onset to ambulance arrival;**<br>**Ambulance transport to hospital;**<br>**Hospital admission to thrombolysis** | Median;<br>142 mins<br><br>60 mins<br><br>35mins<br><br>25 mins. |
| O'Carroll RE et al (2001) Scotland | Interview & questionnaire 72 pts admitted with AMI | **Delay** – admitting doctors estimate of time of MI to recorded time of A&E arrival (confirmed by patient 3-5 days later) | Mean 474.7 mins<br>Median 167 mins |
| Ottesen MM et al (2004) Denmark | Structured interview 250 pts admitted with acute coronary syndrome | **Prehospital delay** – onset to hospital presentation<br>**Decision delay** – onset to seeking medical attention<br>**Physician delay**-seeking medical attention to arrival of ambulance/at A&E.<br>**Transportation delays** ambulance arrival to hospital presentation | Median times:<br>107 mins,<br><br>74 mins<br><br>25mins<br><br>22mins |

| Authors | Design & sample size | Phases & definitions of delay | Average delay times |
|---|---|---|---|
| Ruston A et al (1998) Canterbury, UK | Qualitative study semi structured interviews 43 cardiac patients 21 bystanders | Decision phases from onset to call for help: **Warning** **Interpretation** **Preliminary action** **Re-evaluation** **Final action** | |
| Schmidt SB et al (1990) USA | Observational – questionnaire and chart review 126 pts admitted with AMI | **Pre-hospital time** - Onset time to hospital arrival | Mean 5.9 hours (±11.0) Median 2.0 hours |
| | | **Precall time** – symptom onset to call to be taken to hospital. | Mean 5.1 (11.1) Median 1.1 |
| | | **Notification time** - onset to notification of another person. | |
| | | **Decision time** – symptom notification to time of decision to seek medical help | |
| | | **Delay time** – decision to call for medical help and calling ambulance | |
| | | **Wait time** – call for ambulance to leaving for hospital | |
| | | **Transportation time** – travel to hospital | |
| Safer MA et al (1979) | Retrospective self report | **Appraisal delay** – time to interpret symptoms | Median 4.2 days |
| | | **Illness delay** – time between recognising illness and decision to seek medical attention | Median 3.1 days |
| | | **Utilization delay** – time between decision to seek medical help and receiving it. | Median 2.5 days |
| | | **Total delay** | Median 8 days |
| Syed M et al (2000) Detroit, USA | Prospective cohort 395 MI patients Retrospective review of delay from medical notes | **Pre-hospital delay** – Symptom onset to hospital admission | Mean (SD) & median: Blacks- 6.4 (±6.0), 3.2 hrs Whites -4.8( ±5.3), 2.1 hrs |
| | | **Door to needle time** – hospital admission to thrombolysis, | Blacks - 2.1 (±1.9), 1.3 hrs Whites - 1.5 (±1.8), 1.0 hr |
| Walsh J et al (2004) Ireland | Single sample 61 AMI patients | | Median: |
| | | **Phase 1: Patient delay** – Time taken by patient to recognise symptoms & call for help | 20 mins |
| | | **Phase 2: Pre-hospital delay** – call for help to hospital arrival | 140 mins |
| | | **Phase 3: Hospital delay** – hospital arrival to treatment | Not reported |
| | | **Pre-hospital delay** –symptom onset to hospital admission | 244 mins |

## 2.3: Predictors of pre-hospital delay

Previous studies have reported a number of factors which have been found to predict pre-hospital delay. These include socio-demographic factors such as gender, age, race, socio-economic background and education, as well as clinical factors such as presenting symptoms and severity of cardiac event, past medical history and time of day/season in which the symptoms occurred. Previous studies have failed to provide a clear picture of which factors account for prolonged pre-hospital delay.

### 2.3.1: Demographic factors - Age

Most studies have found a statistically significant relationship between age and pre-hospital delay showing that older people delay up to twice as long as younger people (Berglin-Blohm et al, 1998; Dracup et al, 1995; Dracup & Moser, 1997; GISSI, 1995; Goldberg et al, 1999; Goldberg et al, 2000b; Goldberg et al, 2002; Gurwitz et al, 1997; McKinley et al, 2000; Ryan & Zerwic, 2003; Schmidt & Borsch, 1990; Walsh et al, 2004). These studies suggest that increasing age is significantly associated with delays of over 2 hours and that patients aged over 70 year are more likely to delay over 12 hours than younger patients. In a large study of 102 339 patients aged over 65 years carried out in the USA by the Cooperative Cardiovascular Project, 29.4% arrived 6 or more hours after symptom onset and of these 17.8% arrived after 12 hours (Sheifer et al, 2000). A large retrospective American study of 18 928 patient found that prolonged pre-hospital delays of more than 4 hours were more common among patients aged 65 or greater (McGinn et al, 2005). There are a few studies reporting no association between age and pre-hospital delay (Goldberg et al, 1992; Matthews et al, 1983; Wielgosz et al, 1988), however, as Dracup et al (1995) argue these are mainly small studies which lack the statistical power to detect differences.

Research by McMechan and Adgey (1998) showed age to be the single most important predictor of mortality resulting from AMI. Rask-Madsen et al (1997) reported that patients over 70 years old account for between a third and half of patients suffering AMI and approximately 80% of deaths due to AMI occur in those over 65 years, with 60% of these occurring in people aged over 75years. This is also supported in a study by Gurwitz et al (1997), who found that patients who did not have a living spouse, who lived alone, who lived in a long term nursing home, or who were retired were also more likely to have longer pre-hospital delays.

It has been suggested that older patients have longer delays because they have more difficulty in interpreting their symptoms due to the presence of other underlying illnesses with which the symptoms may be confused (Ryan & Zerwic, 2003). Older people may also associate milder symptoms, such as fatigue, with normal physiological processes of aging, even when these symptoms are severe. Previous history of AMI, heart failure, cardiogenic shock, atrio-ventricular block, and atrial fibrillation or flutter is more common among older patients, and this may interfere with their interpretation of acute symptoms of AMI (White et al, 1996). Ryan & Zerwic (2003) suggest that these chronic illness symptoms, which are more common in elderly patients, mask or create "background noise" in the presence of acute symptoms making interpretation more difficult.

Rate of symptom onset may also be an important factor influencing how quickly older people in particular respond to their symptoms. The slower the rate of onset, the more likely it is that symptoms will not be considered serious and will be seen as a normal part of aging (Leventhal & Diefenbach, 1991). This may be especially true if patients experience prodromal symptoms, the most common being chest discomfort (of recent

onset or a change in status of chronic angina), dyspnoea, palpitations, diaphoresis, fatigue, weakness, and depression. Up to 61% of people have been found to experience prodromal symptoms prior to suffering AMI (Hofgren et al, 1995).

### 2.3.2: Gender

The majority of studies have found women to have longer pre-hospital delay times than men. A large retrospective American study which reviewed the medical notes of 18 928 AMI patients found that prolonged pre-hospital delays of more than 4 hours were more common among women and black patients (McGinn et al, 2005). Similarly, in a large cross sectional study of 364 131 patients included in the second National Registry of Myocardial Infarction, Goldberg et al (2000b) found that women were more likely than men to wait over 2 hours before contacting medical help. A retrospective chart review of 145 male and 166 female patients with chest pain found that women were more likely than men to delay over 6 hours after the onset of chest pain before hospital presentation (Lehmann et al, 1996). Other studies have produced similar findings (Goldberg et al, 1999; Gurwitz et al, 1997; Sheifer et al, 2000; Syed et al, 2000).

There is, however, some conflicting evidence concerning the influence of gender on pre-hospital delay. Zerwic (1999) argues that the influence of gender on delay during ACS has not been clearly established because historically women were either not recruited into these studies or included in substantially smaller numbers than men. Indeed, a few studies have found no association between gender and pre-hospital delay. A study of 1097 MI patients (246 of which were female) found no evidence that women have longer pre-hospital delay than men (Kudenchuk et al, 1996). Similarly, a study of 1360 AMI patients (810 male and 550 female) by Goldberg et al (1998) found no

gender differences in delay. This is also supported by evidence from other studies (Dracup & Moser, 1997; Moser et al, 2005; Zerwic et al, 2003).

Zerwic (1999) argues that many studies examining the effects of gender have not consistently controlled for the effect of age, which is an important consideration given that women are usually older than men at the time of infarction. It is important to determine whether any effect of gender is an independent predictor of delay or if it is confounded by age. She also comments that since women usually access health services more often and more readily than men, it is unclear why their delaying behaviour during AMI is so inconsistent with usual health care seeking behaviour, unless women experience a different pattern of symptoms than that exhibited by men or do not believe that AMI is a disease experienced by women.

There is some evidence to suggest that women may experience more atypical symptoms than men making diagnosis more difficult. Meishke et al (1998) reported that women experience more nausea and shortness of breath than men. Women have been found to suffer significantly more back and jaw pain, nausea and/or vomiting, dyspnoea, indigestion dizziness, fatigue loss of appetite, syncope and palpitations compared with men who were more likely to experience chest pain and diaphoresis (DeVon & Zerwic, 2003; Goldberg et al, 2000a). A large American study of over 10 000 adults presenting to A&E departments with cardiac symptoms also found that women were more likely than men to present with symptoms of heart failure and that they were less likely to report chest pain as their chief complaint (Zucker et al, 1997). In contrast, the Myocardial Infarction and Triage Intervention project (MITI), reported that 99% of subjects with confirmed AMI reported chest pain and that there were no gender differences in patients presenting with dyspnoea, diaphoresis, nausea and epigastric pain

(Kudenchuk et al, 1996). Evidence from these studies is therefore not entirely consistent regarding gender differences in symptoms of AMI.

Meishke et al (1999) investigated the how women label symptoms of AMI and argued that many women mis-label their symptoms at onset causing them to delay seeking medical help earlier. Women who recognised common and less common symptoms as cardiac were more likely to say they would seek medical help immediately than those who did not. Awareness of atypical symptoms, information seeking, and perceptions of personal risk were significant predictors of labelling atypical symptoms as cardiac. Some studies have found that women experience more prodromal (early) symptoms than men (Hofgren et al, 1995). Women are usually significantly older than men when they present, the average age for women presenting with AMI was 61 years compared with men at 57 years (Zucker et al, 1997). Women were also found to have a higher incidence of diabetes and hypertension, both of which have been shown to increase delay times in some studies, and it is possible that combination of these factors may influence delay time in women.

Delays in treatment may be compounded when patient initially contact the general practitioner (GP). In a Danish study by Ottesen et al (2004) of 250 patients admitted with ACS, women were found to have atypical symptoms more frequently and greater pre-hospital delay time than men due to prolonged physician delay (69 minutes versus 16 minutes). Bouma et al (1999) investigated patient delay in 400 consecutive patients admitted with AMI in the Netherlands and found substantially longer pre-hospital delay in women (52 minutes versus 36 minutes) caused by the GP taking longer to form a diagnosis in women. A British study by Heriot et al (1993) also reported similar findings. Studies suggest that because atypical symptoms may be more common in

women, doctors find it more difficult to make a diagnosis and this leads to longer delay. Dracup et al (1995) argue that GP's prolong delay by not perceiving the symptoms as cardiac, making recommendations for self-medication, and also due to inappropriate counselling by reception staff. The inability of patients to contact GPs immediately or arrange appointments at short notice may also be a factor. Patients in the UK are advised to call emergency services directly since this is known to reduce delay time. In spite of this, however, many patients prefer to contact their GP initially. Leslie et al (2000) reported that 55% of patients in a Glasgow study with cardiac symptoms consulted a GP prior to making the decision to attend hospital, and Pattenden et al (2002) reported similar findings in a study in North Yorkshire.

Most studies investigating heart disease have reported a lower proportion of women suffering AMI. Indeed, a prospective study of 8488 patients presenting at hospital with cardiac symptoms found that AMI is almost twice as common in men as women (Zucker et al, 1997). In a large European study of gender differences, Rosengren et al (2004a) found that younger women (<65 years) were less likely than men to present with ST elevation and more likely to be discharged with a diagnosis of unstable angina (UA), while there was no difference in older patients. Among patients who underwent coronary angiography, both younger and older women were less likely than men to have 3-vessel disease or main stem disease. Women in the Framingham study also presented more often than men with angina over a twenty year follow up period (Lerner & Kannel, 1986). This may have contributed to a mistaken belief amongst some women that heart disease is a male problem (Dempsey et al, 1995; Finnegan, Jr. et al, 2000). Although twice as many women died from heart diseases than cancer in 1998 in the USA, a telephone survey of 1000 women aged more than 25 years reported that most women did not perceive heart disease to be a substantial health concern and only

8% perceived coronary heart disease as their greatest health threat compared to cancer (Mosca et al, 2000).

Although women are less likely to suffer an AMI than men, coronary heart disease remains a significant problem among women. In 2002, 17% of all premature deaths in women were caused by coronary heart disease and this is the cause of more deaths among women each year than breast cancer (British Heart Foundation, 2002). The risk of hospital mortality in women is almost twice that of men (Kudenchuk et al, 1996). Findings from the GUSTO-trial also showed that women had more non-fatal complications after treatment, including shock, congestive heart failure, serious bleeding, and reinfarction (Weaver et al, 1996). However, although AMI in women results in a less favourable outcomes, it has traditionally been treated less aggressively in women than men (Kudenchuk et al, 1996; Lehmann et al, 1996). Women are less likley to undergo cardiac catheterisation, angioplasty and surgery for coronary artery bypass grafts (CABG's).

### 2.3.3: Ethnicity

Relatively little is known about pre-hospital delay in minority groups since most studies have focused on white men. Some studies from the USA have found that people with a non-white racial background are likely to delay longer in seeking help for symptoms of AMI. In an American study of survival rates and pre-hospital delay among black patients, Cooper et al (1986) found pre-hospital average median delays of 6.4 hours, approximately twice as long as studies of predominately white population. Most of

these patients, however, did not call an ambulance to take them to hospital and a prolonged transport time may have contributed to this. More recently, Ell et al (1994) described an association between pre-hospital delay and lower socio-economic status, lower education level, female sex, poor symptom perception and decreased access to health care among African Americans. The time taken to decide symptoms are serious and travel time to hospital among African Americans may be influenced by multiple factors including structural access to care, persistence and degree of perceived severity of symptoms, consultations with a lay person as well as medical professionals, and mode of transportation. Possession of health insurance may have been a factor influencing pre-hospital delay in this study since people from lower socio-economic status are less likely to have made provision for health insurance and may therefore have been reluctant initially to seek medical help due to the financial implications. Zerwic et al (2003) also reported that African Americans delayed significantly longer than non-Hispanic Whites (3.25h vs 2.0h) and race was found to be a significant predictor variable in whether or not participants sought treatment within the first hour after symptom onset.

The investigators of the Second National Registry of Myocardial Infarction, which included 1624 AMI patients, found delay times more prolonged in non-white patients including African Americans, Hispanics, Asians and American Indians (Goldberg et al, 1999). They reported American Indians had the highest median delay times of all ethnic groups examined; on average they delayed 2.5 hours longer than white patients. This is supported by evidence from a study by Syed et al (2000) which investigated the effect of delay times on racial differences in thrombolysis for AMI. Of 395 patients with a first myocardial infarction included in the study, 33% were African American. Delay times were determined retrospectively by review of medical records. Pre-hospital

patient delay from onset of symptoms to presenting at the emergency department, and door to needle times were significantly longer in African Americans, and African American patients received thrombolysis less often than white patients (47% vs 68%, $p < 0.001$). African Americans were more likely to present at Emergency Department later than 2 hours after symptom onset. The increased pre-hospital delay time in African American patients strongly influenced whether they received thrombolysis. They also found that that in their sample African Americans were more often female, more likely to have hypertension and diabetes mellitus, and more likely to have non-Q-wave AMI.

Significant ethnic differences in the reporting of symptoms and prolonged pre-hospital delay have been observed in other studies. In a large study of 10,469 African Americans with AMI enrolled in the National Registry of Myocardial Infarction-2, the under utilization of reperfusion therapy was investigated in relation to atypical presentation, patient and hospital delay (Manhapra et al, 2001). As many as 47% of eligible African American patients in this study did not receive reperfusion therapy. Progressive delays in hospital arrival and evaluation were associated with reduced likelihood of early reperfusion. The investigators suggested that this was due to atypical presentation (absence of chest pain) and delays in seeking medical help.

It is possible different ethnic groups experience different symptoms of ACS. Studies have reported that Mexican Americans are likely than Whites to experience upper back pain and palpitations, jaw and arm pain in addition to classic symptoms such as chest pain (Meshack et al, 1998) and African Americans are more likely than white patients to present with atypical symptoms such as absence of chest pain (Manhapra et al, 2001). In a study investigating delayed presentation in the Hispanic population in Los Angeles, Latinos were found to delay a median average of 9.2 hours, and Asians delayed a

median average of 12 hours whilst African Americans had average delays 3.5 hours, which were similar to those of Caucasians at 3.2 hours (Henderson et al, 2002). A combination of socio-economic status, language and cultural practices may act as barriers to medical care in these populations.

Most of the studies investigating race have been done on population samples in the USA. This may give some indication of differences in pre-hospital delay due to racial background but the differences in health services and access to emergency care may also influence patients' responses to symptom onset where health insurance required. There are known to be racial differences in the prevalence of certain risk factors for coronary artery disease. Hypertension and diabetes mellitus are more common in black people than whites.

In the UK, Barakat et al (2003) found no significant differences between Bangladeshi and white patients in onset of symptoms and arrival at hospital (64.5 mins vs 63.0 mins). South Asians were also found to be more likely than white Europeans to seek immediate medical care for pain identified as cardiac in origin (Chaturvedi et al, 1997). It took almost twice as long, however, for Bangladeshis to receive thrombolysis. They were significantly less likely than whites to complain of central chest pain and more likely to offer non-classic descriptions of pain such as left sided pain and which was pinching or burning in nature (Barakat et al, 2003).

### 2.3.4: Social support

Lack of social support and having fewer social contacts has been strongly associated increased mortality (Rosengren et al, 2004b; Welin et al, 2000) and morbidity (Dickens et al, 2004). Studies have shown contradictory findings concerning marital status.

Marital status has been found to predict shorter pre-hospital delays in some studies (Burnett et al, 1995) whilst other studies have reported no association (Dracup & Moser, 1997; Gurwitz et al, 1997). However, social support and social networks have not been studied in detail in relation to pre-hospital delays. These variables are potentially important, so were included in the present study. Based on the known protective effects of social networks and social support, one might expect them to be associated with shorter delays.

### 2.3.5: Socioeconomic factors

There is some evidence to suggest that patients with lower socioeconomic status have longer pre-hospital delay. One of the markers of socioeconomic status (SES) is years of education. In a study by McKinley et al (2000) of 424 North American and Australian patients with AMI, patients with lower incomes and fewer years of education had longer pre-hospital delay. Similar findings have been reported by other studies (Dracup et al, 1997; Ell et al, 1994; Kentsch et al, 2002).

Annual household income is another frequently used marker of SES. Zerwic et al (2003) reported that patients in the USA with a yearly income of less than $20,000 experienced significantly longer delays than those who earned more than this in a study of 212 patients admitted to hospital with AMI in the mid-west USA. This is supported by a number of other studies (Dracup & Moser, 1997; Meischke et al, 1995; Schmidt & Borsch, 1990; Sheifer et al, 2000). Somewhat contradictory findings were reported by Ruston et al (1998) in a British study, however, using occupational class to measure SES they found that people in manual occupations were less likely to have prolonged pre-hospital delays.

It is possible that perceptions about the ability to pay for health care and differences in accessing medical services may form barriers to seeking medical help in countries where medical care is dependent on private provision of health insurance. Pre-hospital delays, however, in countries where health care is free at the point of delivery such as the UK, Australia and other parts of Europe are comparable to the USA so evidence does not support this proposition (Dracup et al, 1997).

General measures of access to resources often incorporate measures of education, income and type of employment as well as a range of other measures. The Townsend Index has been used widely as a measure of deprivation in medical and social policy research. This provides a material measure of deprivation and disadvantage based on 4 variables taken from the 1991 census which combine to form an overall score. Higher scores indicate higher levels of deprivation and disadvantage (Townsend, 1993). Higher levels of deprivation and social inequality have been associated with higher mortality and poor health in general (Kreiger et al, 1997), and increased risk of heart disease (Malmstrom et al, 1999; Rutledge et al, 2003). Deprivation may impact on pre-hospital delay in a variety of different ways, including lowered self esteem, poor communication skills, less access to information and resources such as having a telephone or owning a car, and greater barriers when trying to access health care (Ell et al, 1994). Sheifer et al (2000) reviewed the charts of 102 339 patients admitted to hospital with MI and reported that patients who lived in an impoverished area were significantly more likely than patients who lived in more affluent areas to present with their symptoms more than 6 hours after onset.

### 2.3.6: Contextual factors

#### *2.3.6.1: Time, day and season of symptom onset*

Almost 25% of AMI's occur within the first 3 hours of waking and there is an approximate 3-fold increased risk of having an AMI during this time (Willich et al, 1991). Some studies have found time of symptom onset to influence pre-hospital delay. Goldberg et al (2000b) found onset of symptoms at the weekend, afternoon and evening, or early morning to be significantly associated with delays of over 2 hours. This finding is supported by Gurwitz et al (1997) who found that the greatest risk for pre-hospital delay was symptom onset in the evening and early morning hours, from 6pm to 6am. Similarly, Bouma et al (1999) reported prolonged pre-hospital delay if symptoms started outside of office hours (134 minutes compared with 111 minutes) due to a reluctance of the patient to disturb the doctor. Indeed, Sheifer et al (2000) reported that many patients who presented during the day, had experienced their first symptoms the previous night. Other studies have shown that patients who experienced the onset of their symptoms at the weekend also delayed longer in contacting medical help (Goldberg et al, 2000b; Pattenden et al, 2002).

#### *2.3.6.2: Presence of a bystander*

Studies have shown contradictory findings regarding the helpfulness of bystander involvement in help seeking behaviour. Ell et al (1994) found an association between being alone when symptoms started and longer patient decision time. Perry et al (2001) also found that shorter delays were associated with the presence of a bystander at symptom onset, talking to another person and talking to a family member. Dracup et al (1995) also reported that most patients arrive at the decision to seek medical help with the involvement of a family member, but that patients who make this decision alone have shorter delay times. In a later study however, Dracup and Moser (1997) found no

significant differences based on whether the patient was alone or not when symptoms began. In contrast to these findings, McKinley et al (2000) found that informing a spouse or other family member resulted in considerable delays, while the presence of a bystander who was unrelated to the patient such as a friend, co-worker or stranger resulted in shorter delays. Dracup and Moser (1997) reported that patients who were at home when symptoms began delayed longer than those who were elsewhere . They suggested that family members may be more likely to share the denial of the patient where as a co-worker may be less willing to bear the responsibility of a wrong decision. Evidence from a German study of 739 patients post AMI partially supported this in that although asking others for advice generally reduced the risk of delay, in 2.8% of participants, there was an almost 7-fold higher risk of longer patient delay time because bystanders encouraged a "wait and see" approach (Kentsch et al, 2002).

#### *2.3.6.3: .Type of assistance sought*

Patients who contact the ambulance service initially when they seek help have shorter delays than patients who seek help from other sources such as family members, friends or their GP (Ell et al, 1994; Heriot et al, 1993; McGinn et al, 2005; Schmidt & Borsch, 1990). Using the emergency ambulance service also has other advantages, such as the immediate provision life support skills should the patient develop cardiac arrythmias or cardiac arrest, and medical assessment prior to hospital arrival which may accelerate the commencement of thrombolysis. Evidence from several studies, however, shows that patients gave a number of reasons for their reluctantance to call an ambulance; they believed their symptoms were not serious (Leslie et al, 2000), were afraid of wasting NHS time and resources (Pattenden et al, 2002), were worried about troubling others, feared the consequences of seeking help, decided to wait for symptoms to go away and did not recognise the importance of their symptoms (Dracup & Moser, 1997).

Meischke et al (2000) found that 50% of patients call their GP first rather than the ambulance service after developing symptoms of ACS. Carney et al (2002) also found that three quarters of patients in their study called the GP first. Leslie et al (2000) also reported that most patients in their study called their GP initially after the onset of cardiac symptoms and only one quarter call for an ambulance service.

### 2.3.7: Clinical factors

#### *2.3.7.1: Cardiac risk factors and previous medical history*

The evidence concerning pre-hospital delay and patients past medical history is contradictory. Intuitively, one would expect patients with a history of cardiac problems or cardiac risk factors to have shorter pre-hospital delays following the onset of cardiac symptoms. However, this is not the case. Several studies have shown that patients with a previous cardiac history are likely to delay as long or longer than those than those with no previous cardiac history (Dracup et al, 1995; Dracup & Moser, 1997; Goldberg et al, 2000b; McKinley et al, 2000; Pattenden et al, 2002; Walsh et al, 2004). The Worcester Heart Attack Study, a longitudinal study of 3837 patients hospitalised with AMI, reported that patients with a prior medical history of angina, diabetes or recent hospitalisation within the previous year were significantly more likely to delay more than 2 hours (Goldberg et al, 2000b). Other studies have also found that a history of diabetes and angina significantly increases the likelihood of a longer delay (Dracup & Moser, 1997; Goldberg et al, 2000b; McKinley et al, 2000; Sheifer et al, 2000). Patients with a history of hypertension have also been found to be more likely to have increased delay times than normotensive patients (Berglin-Blohm et al, 1998; Goldberg et al, 1999; Gurwitz et al, 1997).

It is possible that patients may be initially unable to distinguish their symptoms from more common complaints in the presence of other co-morbidities such as heart failure, angina or diabetes. It has been suggested that diabetic patients may have an altered perception of pain (Nesto & Phillips, 1986; Sheifer et al, 2000) and that denial may play a greater role in the reaction of patients who have a previous cardiac history or higher risk factors (Goldberg et al, 2002). These patients may be reluctant to face the possibility of having another ACS, and the implications of this combined with other co-morbidities. Alternatively, they may experience a different set of symptoms to the first time and not recognise them as cardiac symptoms.

Research evidence is, however, somewhat contradictory. Sheifer et al (2000) reported that in a study of 102 339 older subjects aged 65 years and over, although a prior history of diabetes and angina predicted significantly longer delay, previous AMI, bypass surgery, angioplasty and cardiac arrest predicted early presentation . The second National Registry of Myocardial Infarction in the USA (a large cross-sectional study of 364 131 patients) also showed that patients with previous AMI or who had undergone previous coronary angioplasty had shorter delays (Goldberg et al, 1999). Gurwitz et al (1997) reported that a history of mechanical revascularization reduced the risk of prolonged delay in a retrospective chart review study of 2409 patients admitted with AMI . This would seem to indicate that specific prior experience of heart attack and specific types of treatment does indicate shorter delay, while the presence of other more general risk factors that increase vulnerability to ACS may not.

#### *2.3.7.2: Attribution of symptoms*

Prodromal angina pectoris occurring shortly before the onset of AMI has been associated with smaller infarct size and better both long term and short term survival in

non-elderly patients (Ishihara et al, 2000). As many as 58 to 61% of patients admitted to hospital with symptoms of MI report prodromal symptoms (Hofgren et al, 1995; Horne et al, 2000; O'Carroll et al, 2001). Patients who experienced a slower onset of symptoms with prodromal heart related symptoms have been found to have significantly longer delay times (Horne et al, 2000).

Patients' experience of particular clinical symptoms have been shown to affect pre-hospital delay. Unsurprisingly, severe chest pain of sudden onset is usually associated with shorter delays. Goldberg et al (2002) reported that patients later diagnosed with an ST elevation AMI had shorter delays (median 2.3 hours) than those with a non ST elevation MI (median 3.0 hours). This is also supported by other studies (Steg et al, 2002). Patients with severe symptoms such as cardiogenic shock, haemodynamic instability and larger infarcts have been shown to delay less than patients with smaller or non-Q-wave AMI (Goldberg et al, 1999; Leizorovicz et al, 1997). It is possible therefore that the pattern of symptoms is slightly different. It is interesting, however, that there does not seem to be a clear association between pre-hospital delay and intensity of pain. O'Carroll et al (2001) found no association, while Ell et al (1994) reported that patients who perceived their pain to be continuous and incapacitating had significantly shorter delays in seeking treatment. Horne et al (2000) reported a weak association with severe pain predicting shorter delays.

The pattern of symptoms may be an important factor in pre-hospital delay. Although Leslie et al (2000) reported that chest pain is the only symptom in 35% of cases, Cameron et al (2005) found that patients experience an average of 6.43 symptoms. They found that the most common symptoms reported by newly diagnosed MI patients questioned within 2 days of their admission were fatigue, chest pain, loss of strength

and increased perspiration. Golberg et al (2000a) reported that, in addition to chest pain, dyspnoea, arm pain, sweating, and nausea were the most common symptoms, although women were more likely than men to report nausea, vomiting and back, neck, and jaw pain. Several studies have reported that patients who experience atypical symptoms delay longer in seeking help. Australian patients who reported heartburn, shortness of breath or intermittent symptoms had prolonged delay times (Dracup et al, 1997). Women may be more likely to present with atypical symptoms and may also experience prolonged physician delay because of this (Ottesen et al, 2004). Grossman et al (2003) also found that symptoms such as shortness of breath, nausea, or weakness predict delay, although these results may have been confounded by age in this study. Older patients are also more likely to have longer delays and to present with atypical or non-chest pain symptoms and are more likely to have co-morbidities such as diabetes, arthritis, previous cardiac problems or a degree of cognitive impairment, and may be less able to discern the onset of symptoms.

Many studies have shown that the belief that one is having a heart attack is associated with prompt hospital attendance (Carney et al, 2002; Leslie et al, 2000; Meischke et al, 1995; O'Carroll et al, 2001). Carney et al (2002) reported that the odds pre-hospital delay of less than 60 minutes in patients who attribute their symptoms to heart pain is approximately four times higher than in patients who attribute their symptoms to some other cause. Ruston et al (1998) reported that non-delayers knew about a wider range of symptoms while delayers knew only about chest and arm pain, and extended delayers were unsure about sympotms.

Patients often misinterptret their symptoms. In a study by O'Carroll et al (2001), just 17% of patients believed their symptoms were those of a heart attack, 42% thought they

were experiencing heartburn or indigestion. Carney et al (2002) also reported that patients most commonly attributed their cardiac symptoms to indigestion. Patients often fail to recognise chest pain as as symptom of heart attack when it is gradual in onset or moderate in severity (Ell et al, 1994; Finnegan, Jr. et al, 2000).

Several studies have demonstrated that the general public, and AMI patients in particular, are knowledgeable about AMI symptoms, however, this does not appear to lead to quicker recognition at the time of the event (Finnegan, Jr. et al, 2000; Goff, Jr. et al, 1998; Goff, Jr. et al, 1999; Horne et al, 2000; Pattenden et al, 2002). In a study of 200 AMI patients by Clark et al (1992) although 95% of patients knew at least one major symptom of AMI, only 50% believed their own symptoms were heart related . A qualitative study of 22 patients admitted to hospital following a second, third or fourth heart attack, showed that knowledge of symptoms from a previous heart attack did not result in shorter decision time (Pattenden et al, 2002). Six themes were identified that may have influenced patients decision making, including symptom appraisal, perception of risk, previous experience, and psychological factors such as fear and denial.

Other studies have shown that patients' prior expectations of AMI symptoms were associated with delay time. Patients whose actual symptoms matched those they would have expected to experience prior to their AMI sought help significantly faster than those whose symptoms did not match their expectations (Johnson & King, 1995; Ruston et al, 1998). In a study by Zerwic et al (2003), 57% of women and 49% of men experienced this mismatch in their symptoms. Similarly, Horne et al (2000) reported that 58% of patients experiencing their first AMI also experienced mismatch between expected and experienced symptoms, and that these patients were more likely to delay

and more likely to have a third party make the decision to call for help. Other studies have also supported this finding (Perry et al, 2001; Ruston et al, 1998).

Several studies have investigated patients' explanations for delays in seeking treatment for cardiac symptoms. Reasons given for delaying included; waiting for symptoms to go away, symptoms were not regarded as serious, worry about troubling others, symptoms not recognised as cardiac, and feared of seeking help, possibly due to the implications of being admitted to hospital and the perceived consequences of this (Dracup & Moser, 1997; McKinley et al, 2000). Patients who used coping strategies such as self- medication and rest also delayed longer. Kentsch et al (2002) reported prolonged decision time of over 1 hour was associated with patients saying that they tried to distract themselves, took analgesics, thought the cause of symptoms to be an illness or organ other than the heart, and who did not take the symptoms seriously . Zerwic et al (2003) also found that strategies such as resting, using over the counter medications, having a hot drink or attempts to consult a healthcare provider resulted in increased pre-hospital.

### 2.3.8: Psychological factors

Patients' responses to cardiac symptoms are not straight forward. Despite being relatively well informed about the signs and symptoms of heart attack, they tend misattribute their own symptoms to a cause other than cardiac and use delaying strategies at symptom onset. These various strategies may be simply a way of denying a serious threat to their health for as long as possible in the face of mounting evidence.

### *2.3.8.1: Cardiac denial of impact*

Denial could be a crucial factor in the first few hours following the onset of symptoms of ACS (Wielgosz et al, 1988; Wielgosz & Nolan, 1991). Previous research into the use of denial as a coping strategy in cardiac patients has found that most cardiac patients engage in denial to some extent (Dracup et al, 1995). It has been linked to both positive and negative consequences. The presence of denial in the first few days of following AMI has been generally accepted as a beneficial coping strategy that may protect the patient from distressing emotions such as anxiety and depression (Lewin, 1995; Sarantidis et al, 1997). In patients diagnosed with ACS, it has also been associated with shorter hospitalization periods, higher rates of return to work (Julkunen & Saarinen, 1994) and increased mortality (Havik & Maeland, 1988) and morbidity (Levenson et al, 1989).

Inattention to pain or maladaptive coping behaviours, however, may increase the time required to decide to seek medical help, lead to a reduced ability to retain information and poor adherence to medical advice post discharge (Wielgosz & Nolan, 1991). Patients who are not attentive to the severity of their symptoms may attribute their pain to indigestion and therefore delay in seeking help, which may then have life threatening consequences. A study by Kenyon et al (1991) found an association between delay and low awareness of emotions in 157 patients post AMI, suggesting that patients who delay in seeking help may be less attuned to physiological and emotional reactivity to cardiac symptoms. Using measures of somatic and emotional awareness (the Modified Somatic Perception Questionnaire and Toronto Alexithymia Scale) they found that patients who were both emotionally and somatically under-aware evidenced by far the greatest mean delay (over 29 hours) in responding to symptoms of AMI than any other group. Alternatively, it is also possible that patients who are very emotionally aware might be

distracted by the diversity or intensity of their symptoms, also resulting in prolonged delay. High emotional arousal has been correlated with increased delay (Wielgosz & Nolan, 1991).

A number of studies have examined the influence of denial on patients decision delay and it has been suggested than previous MI may induce post-traumatic stress disorder so that when symptoms reoccur, patients may try to suppress or avoid stimuli that remind them of the initial trauma (Alonzo & Reynolds, 1998). Meischke et al (2000) reported that fear and denial were frequently experienced during cardiac emergency . Flowers (1992) developed the Cardiac Denial of Impact Scale, a short self report measure designed to focus on the denial of the impact or consequences associated with cardiac illness. Higher scores on this scale have been significantly associated with longer pre-hospital delays of more than 4 hours (O'Carroll et al, 2001). Carney et al (2002) also investigated a possible association between denial and pre-hospital delay in sample of 62 patients post AMI using the Cardiac Denial of Impact Scale. No association was found, although this was a relatively small sample size. Psychological factors that were associated with delay included health value, pre-disposition to report symptoms, internal and chance locus of control, and whether or not symptoms were attributed to heart pain.

## 2.4 Intervention Studies

A few intervention studies have attempted to reduce pre-hospital patient delay but results have been mixed. Intervention studies reviewed in this section are summarized in Table 2.2. One early intervention campaign was conducted in Nottingham and aimed to increase early reporting of cardiac symptoms among 13 000 patients aged over 40 years registered at 3 General Practices. Patients received instructions to call a special emergency hospital number if they experienced chest pain for longer than10 minutes.

Results showed that patients in the intervention practice reported chest pain earlier than the 10 comparison practices and there was a lower rate of definite and probable AMIs among calls received by the special telephone line than calls received by patients' own GP (Rowley & Mitchell, 1982).

Most interventions have used multi media public educational campaigns aimed at increasing knowledge about symptoms of ACS and giving advice about the appropriate actions to take. A study in Götenborg, Sweden, showed a significant reduction in median delay time from 180 to 138 minutes among patients with confirmed AMI following a 12 month education campaign aimed at the general public (Herlitz et al, 1992). Similarly, a 'before and after' study based in Geneva, Switzerland, was also successful in significantly reducing median delay from 180 to 155 minutes using a mass media public awareness campaign involving television, radio, newspapers, advertisements, posters and leaflets which were distributed to every household over the period of 1 year (Gaspoz et al, 1996). Visits to the A&E per week for ACS also significantly increased.

Other studies have been less successful. The 'Call Fast, Call 911' campaign study was randomised controlled trial (RCT) in the USA which used a 10 month mass media radio and television campaign accompanied by a direct mail campaign (Meischke et al, 1997). The aims to increase the percentage of patients calling the emergency number "911", and to decrease patients' delay time. Based on the stages of delay proposed by Safer (1979), the intervention firstly addressed appraisal delay by providing information about the signs and symptoms of ACS; secondly, it addressed illness delay by emphasizing the importance of fast action; and thirdly it addressed utilization delay by eliminating uncertainty about the curability of ACS by stressing the importance of early treatment

and use of emergency medical services (EMS). The intervention started with a 7 week mass media campaign including television and radio. A direct mail campaign then targeted all households in which the head of the household was 50 years of age or older. This intervention, however, had no impact on pre-hospital delay time, although there was a non-significant increase in use of the emergency "911" number.

The Rapid Early Action for Coronary Heart Treatment (REACT) trial was also a large RCT which used a community intervention aiming to reduce patient delay and increase use of EMS (Luepker et al, 2000). The study took place over a 4-year period and included 20 communities (10 matched pairs) in the USA. The intervention consisted of a local advisory group, public education via television, radio and direct mail targeting all residents of the intervention communities with an 18 month programme of raising general awareness of symptoms of ACS and appropriate action to take, professional education aimed at medical staff, and patient education for those with a history of CHD or risk factors. This intervention did not significantly reduce patient delay time although there was an increased appropriate use of EMS in intervention communities.

Berglin Blohm et al (1998) evaluated the results of 9 public education campaigns designed to shorten patient decision delay and pre-hospital delay. This included one Canadian study, two American studies, one Australian and four European studies. Results showed that these campaigns had either no effect at all or produced a limited reduction in pre-hospital delay. The authors concluded that there were no reports to indicate that media campaigns improved survival and that these interventions had not proved worthwhile in improving pre-hospital delay.

In a recent systematic review of eleven studies by Kainth et al (2004), the authors concluded that there was little evidence that media/public information campaigns or one to one educational interventions reduced pre-hospital delay and they may have resulted in an increase in calls made to emergency switchboards. The eleven studies included in the review consisted of the two RCTs, a controlled trial and eight 'before and after studies'. They commented that the methodological quality of these studies was generally poor making it difficult to draw firm conclusions. They suggested that future research should emphasize the importance of thrombolytic therapy, make people feel it is acceptable to call the emergency services without fear they are wasting NHS resources, evaluate the decision making process and target education at high risk groups (including family). They also recommended the measurement of mortality as an outcome measure in such studies. This supports conclusions from another earlier systematic review of mass media interventions aiming to reduce help seeking delay among AMI patients (Caldwell & Miaskowski, 2002). This review critiqued eight mass media intervention studies, only three of which were successful in reducing delay but since they did not utilise experimental design causal inference could not be established. The authors highlighted poor methodology used in many of these studies and concluded that media messages should do more than create awareness, rather they should target high risk groups, provide gender specific information, emphasize symptom evaluation, problem solving and decision making skills and address problems of denial.

**Table 2.2: Intervention studies aiming to reduce pre-hospital delay**

| Authors | Description of study population and intervention | Outcomes | Results of intervention |
|---|---|---|---|
| Gaspoz,J.M et al (1996) | Population based prospective observational study<br><br>Population – population of Geneva, Switzerland (380 000)<br>2477 patients with chest pain who presented at A&E during 12 months prior to and during study<br><br>12 month multimedia campaign | 1) Time of pre-hospital delay | 1)Mean pre-hospital delay reduced from 7h 50mins (median 180 mins) to 4h 54 mins (median 155 mins) P<0.001<br><br>2)Significant increase in A&E attendance for ACS<br><br>3)Significant increase in calls to central switchboard<br><br>4)No change in ambulance use |
| Herlitz,J et al (1992) | Study based in Göttenborg, Sweden<br>12 month public education campaign | 1) Time from symptom onset to A&E arrival | 1)Reduction in median delay from 180 mins to 138 mins. |
| Ho, M.T et al (1989) | Public media education campaign<br>Population – population of King County, Washington, USA<br><br>401 AMI patients interviewed pre-campaign and 489 AMI patients interviewed post campaign | 1)Time of pre-hospital delay<br><br>2)EMS use | 1)No significant change in pre-hospital delay<br><br>2) No significant change in use of EMS |
| Luepker,R.V et al (2000) | Rapid Early Action for Coronary Treatment (REACT) Trial<br><br>Design: 10 matched pairs of US cities.<br>Population 55777-238912<br>18 months duration<br><br>Target - mass media via radio, television, newspaper, direct mail, brochures.<br><br>4 strategies:<br>1)Community organization<br>2)Public education<br>3)Professional further education -<br>4)Patient education | 1) Time from symptom onset to A&E arrival<br><br>2)Use of EMS | 1)Non-significant over all difference between intervention and reference communities<br><br>2) 20% increased appropriate use of EMS in intervention communities (P<0.005)<br><br>3)Non-significant increase in ambulance use in intervention communities. |
| Meischke,H et al (1997) | ''Call fast, call 911"<br>Prospective randomized controlled trial<br>3 intervention groups & 1 control group<br><br>7 week mass media campaign followed by 10 month direct mail campaign<br><br>Population – population of King County, Washington, USA<br>5447 pts suffered a cardiac event | Time of pre-hospital delay | 1)No intervention effects observed<br><br>2)Non significant increase in use of " 911" in intervention groups |

| Authors | Description of study population and intervention | Outcomes | Results of intervention |
|---|---|---|---|
| Rowley,J.M et al (1982) | Study population – 13000 patients aged over 40 years from 3 General Practices<br><br>Instructions given to call a special telephone number if developed chest pain lasting more than 10 mins<br><br>Convenience comparison groups recruited from 10 General practices | Early reporting of symptoms | 1) Earlier reporting of chest pain but to own GP rather than special telephone number |

## 2.5: Theoretical models

It has been recognised for many years that patients' beliefs about their illness affect their responses to symptoms and decisions about treatment (Mechanic, 1972). Over recent years various models of health behaviour have been developed to try to explain various ways individuals try to make sense of the problems that arise following the onset illness. The two most widely used in relation to cardiac patients and pre-hospital delay are outlined below.

### 2.5.1: The Health Belief Model

The health belief model has been used to examine the behaviour of AMI patients, particularly in relation to adherence (Becker et al, 1972). This model is attractive to researchers because it is based on a relatively uncomplicated cost/benefit framework. The model suggests the patient who experiences chest pain is likely to make the decision to seek medical help depending on two variables:

1) The amount of threat perceived by the patient in relation to the symptoms. This will determine how vulnerable s/he feels to cardiac disease and general illness, and is confirmed by the presence of symptoms, previous experience of symptoms, degree of disruption to social roles etc.

2) The attractiveness or value of the action in question, ie contacting medical services. This is based on the probability that, in the patients' view, the decision to go to hospital

will reduce the threat and will not be too costly in terms of time, money or emotional energy.

Other factors such as demographic characteristics, treatment related issues, the patients' own attitudes and physician-patient interactions may also have an influence on these variables.

There are three main criticism of using the health belief model to understand pre-hospital delay. Firstly, it does not reflect the important role that bystanders may play in the decision to seek medical help (Dracup et al, 1995). Secondly, the model seems to be more accurate at describing preventive behaviours such as use of seat-belts than predicting care seeking behaviours (Kasl,1975). Thirdly, prospective studies have reported weak relationships within the model structure indicating that a significant portion of the variance in delay is not explained by the model alone (Haynes, 1976).

### 2.5.2 The Common-Sense Model of Self Regulation

The Common-Sense Model (CSM) is a self-regulatory model of illness behaviour developed by Leventhal & Diefenbach (1991) and may be particularly useful in the investigation of pre-hospital delay. The model views patients as active problem-solvers who organize their knowledge about illness in complex memory structures, or cognitive representations. These cognitive representations are constantly updated by new information and experience. It is these cognitive representations that are thought to determine the behaviours the patient chooses at the onset of symptoms such as seeking appropriate treatment. These illness representations are also thought to determine how patients will cope in terms of adhering to medical advice following ACS and emotional adjustment.

The model proposes that patients' beliefs have five basic components or themes that serve to define the nature of the health threat for the individual:

1) The illness identity comprises of the label that the individual uses to describe the condition (such as heart attack) and the associated symptoms (chest pain, breathlessness, pain in the left arm, nausea etc).

2) Beliefs about the cause of the illness.

3) Expectations about the duration of the illness and time needed for treatment.

4) The consequences of the illness in terms of its physical, social, emotional and financial implications.

5) Understanding of the controllability of the illness, whether or not it is curable and extent to which it requires professional medical intervention.

The model also describes three stages of coping following the onset of symptoms. In the first stage the health threat is assessed and labelled. The patient becomes aware of the symptoms, assesses possible causes and identifies the nature of the threat in the context of passing time. In the second stage, the patient develops coping strategies to deal with the threat and formulates an action plan. This may include the decision to wait and see what happens or to seek immediate medical help. In the third stage, the patient evaluates the success of this plan and reassesses the cognitive representation. All three stages are influenced by the patients' abstract knowledge (such as typical symptoms of heart attack, risk factors and personal vulnerability) and previous experiences.

Patients' illness representations may vary considerably within any illness population, determining not only their choice of coping behaviour but also forming a conceptual framework for making sense of information from health care professionals and

evaluating recommended treatment or advice. For most people, the onset of symptoms of ACS would be a very frightening experience. Leventhal (1970) explains the role of emotions in care seeking behaviour using the idea of parallel processing which proposes that people typically make simultaneous cognitive and emotional representations of their illness (see Figure 2.1). The illness representations therefore not only consist of the cognitive elements described above, but also of emotional representations (of fear and anxiety) which may be important determinants of emotional outcomes (such as anxiety and depression). Health threats are thought to generate both emotional states of fear and distress and a corresponding need for procedures to manage these emotions as well as a cognitive representation of the threat and a corresponding need for procedures for managing these threats (Leventhal et al, 2003).

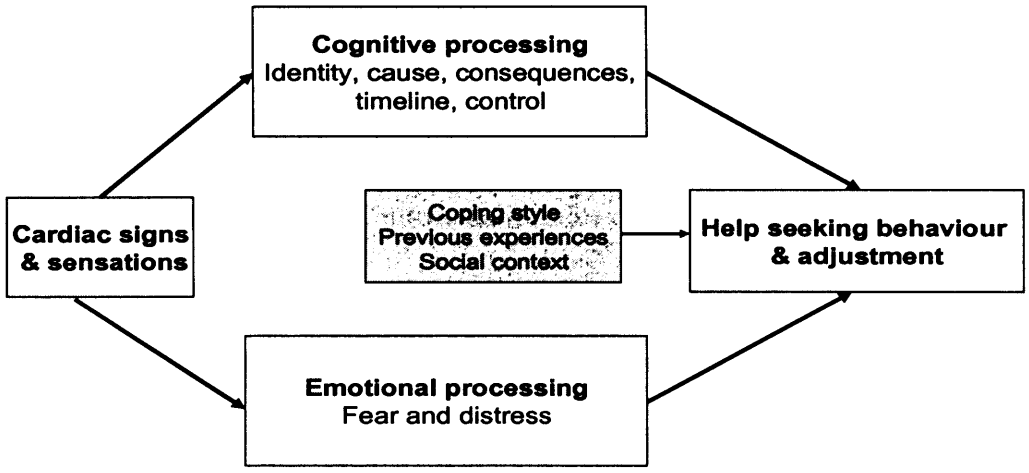


**Figure 2.1: Parallel processing (Leventhal, 2001)**

The CSM has been used in a number of studies investigating the influence of patients' perceptions of illness in various different diseases and health issues, and has been particularly useful in examining health behaviours among cardiac patients. For example, Meishke et al (1995) found that the model provided a useful framework for understanding processes involved in the decision to use the emergency medical services at the onset of symptoms . Pattenden et al (2002) and Walsh et al (2004) used the CSM to help understand aspects of pre-hospital delay. Petrie et al (2002) developed an intervention based on the CSM which was successful in causing positive changes in patients' attitudes towards their AMI, resulting in faster return to work and lower rates of angina symptoms at 3 months follow up. Negative illness representations have been found to predict complications during recovery from AMI (Cherrington et al, 2004) and in predicting attendance at cardiac rehabilitation programmes (Cooper et al, 1999). Dracup et al (1995) developed an integrated model of treatment seeking for AMI symptoms using the CSM and interactionist role theory (symbolic interactionism) to explain the role bystanders play in the decision to seek help or delay treatment.

The CSM and patients' illness representations may therefore be useful in understanding pre-hospital patient delay, adherence and adjustment in patients following diagnosis of ACS. This thesis will use the CSM as a framework to focus in particular on beliefs about illness identity and cause, which may be important factors involved in patients' decisions to seek help following the onset of symptoms, and attributions concerning the cause of the ACS, which may impact on adherence to medical advice and emotional adjustment post hospital discharge. Illness representations may also be associated with quality of life following diagnosis of ACS.

## 2.6: Summary

This literature review has discussed a range of factors have been found to predict pre-hospital delay in previous research (summarized in Figure 2.2). This thesis will investigate the socio-demographic and psychological factors which predict delay in contacting medical help following the onset of symptoms of ACS, based on the hypothesis that shorter patients' decision time in seeking help will be associated with demographic and psychosocial variables including younger age, male gender, greater social support, higher socio-economic status, time of onset on a week day and within work hours, the presence of a bystander, attribution of symptoms to heart attack and low cardiac denial.

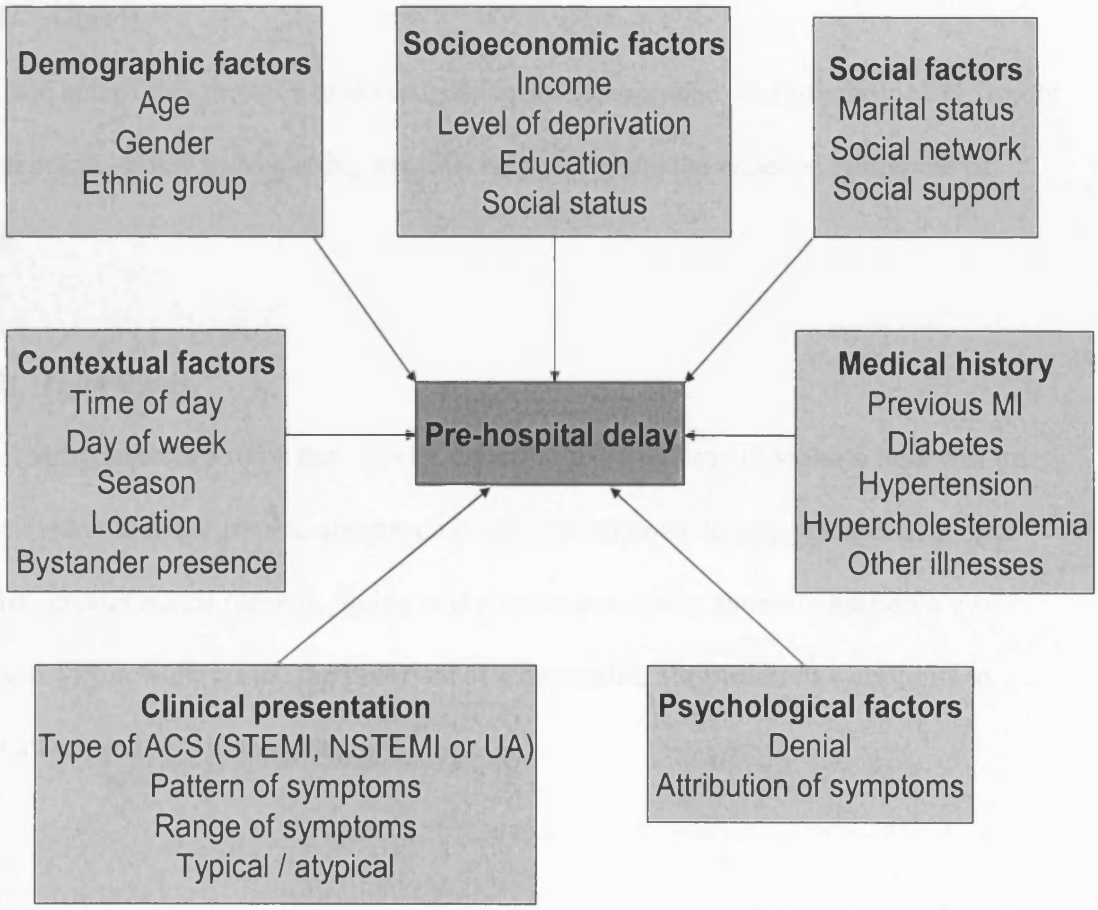


**Figure 2.2: Factors predicting pre-hospital delay in previous research**

# Chapter 3: Investigation of factors which predict pre-hospital delay

## 3.0: Introduction

Cardiac patients vary in the speed with which they seek medical help following the onset of acute chest pain. This is important because the effectiveness of treatment to limit the extent of damage to the heart muscle and to restore blood flow depends on prompt treatment. Fatality from cardiac arrhythmias is also reduced if treatment is commenced early.

## 3.1: Aims and Hypotheses

### 3.1.1: Aims

The first aim of this thesis is to investigate socio-demographic and psychological factors which predict delay in contacting medical help following the onset of symptoms of ACS.

### 3.1.2: Hypothesis

The first hypothesis will be that shorter patients' decision time in seeking help will be associated with demographic and psychosocial variables including younger age, male gender, greater social support, higher socio-economic status, time of onset on a week day and within work hours, the presence of a bystander, attribution of symptoms to heart attack and low cardiac denial.

## 3.2: Methodology

### 3.2.1: Participants

This study took place in the context of a larger study, the ACCENT study, which was investigating emotional and behavioural triggers for chest pain as a symptom of acute myocardial infarction (AMI) and unstable angina (UA). It was funded by the British Heart Foundation. Participants were recruited from 5 separate hospitals including University College Hospital, The Heart Hospital, St Georges' Hospital (all London hospitals), Kingston Hospital and Southend Hospital. A total of 375 patients were potentially eligible for the study, however 49 (12.8%) were discharged or transferred to other hospitals before interview could take place, and 32 (8.5%) declined the invitation. A total of 294 patients were recruited for the ACCENT study. For this thesis, data will be analysed on a study population of 269 of these patients. This will include 2 sub-samples which overlap (Appendix 1), those patients for whom information on pre-hospital delay was available (N = 228, see below) and patients returned and completed the measure for causal attributions ( N = 165, see chapter 5).

Of the 294 patients recruited to the ACCENT study, admission time data was missing in 66 cases where patients had been transferred into one of the participating hospitals from another hospital and information on the primary admission time was not documented in transfer notes. A study population of 228 participants on whom admission time data were complete were analysed for the first part of this thesis which will focus on pre-hospital delay. The second part of the thesis will focus on causal attributions for heart disease, adherence to medical advice, adjustment and quality of life and will include a study sample of 165 participants who returned self-completed questionnaires and provided adequate data for this stage of the analysis.

Approval was granted from the relevant Local Research Ethics Committees (LRECs), and Research and Development Departments. Consecutive patients admitted to the coronary care units between 2nd October 2001 and 30th September 2004 were considered suitable to enter the study providing they were admitted with chest pain and a diagnosis of ACS. Diagnosis of ACS was made by a cardiology consultant or registrar and based on the presence of chest pain verified either by progressive ECG changes (ST segment elevation in 2 contiguous leads of at least 1mm, ST segment depression of at least 1mm in 2 contiguous leads, new left bundle branch block, or dynamic T wave inversion), raised level of troponin T >0.1$\mu$l (Ammann et al, 2004), or creatine kinase (CK) measurement of over twice the upper reference limit (The Joint European Society of Cardiology/American College of Cardiology Committee, 2000). It was important that patients could identify the time of symptom onset, time they contacted medical services and were able to recall events leading up to their admission clearly.

### 3.2.2: Exclusion criteria

Patients were excluded according to criteria required for the ACCENT study as follows;

- Unable to clearly recall time of symptom onset and to describe events leading up to hospital admission.
- Unable to understand or complete a structured interview.
- Not fluent in English.
- Presence of on-going critical ischaemia or ventricular tachyarrythmias.
- Too ill for interview.
- Serious psychiatric illness.
- Decline to participate.
- Under 18 years of age.

- Short/medium outlook compromised by other medical conditions or co-morbidities such as renal failure, cancer, stroke, any inflammatory illnesses and any illnesses that may have influenced mood and symptom presentation.

(The last criterion is specific to the requirements of the ACCENT study for reasons not relevant to this thesis).

### 3.2.3: Design

This study used an observational prospective cohort design using structured interview and questionnaires, with follow up at 3 months and 12 months.

### 3.2.4: Procedure

All patients invited to participate were given a patient information sheet which explained the rationale for the study, implications of their participation and specified the main investigators involved (Appendix 2). Patients were allowed as much time as they needed to read through the information, ask questions and to make a decision on their participation within the initial 5 day period of their hospital admission. Those who agreed to participate were then asked to give written consent (Appendix 3) and a time was arranged for interview. I personally recruited and interviewed 157 patients in this study.

Psychosocial data were collected by means of a semi-structured face-to-face interview (Appendix 4) by one of five trained interviewers (including this author) in a private area free of interruptions as soon after their ACS as possible and within 5 days of admission. All information was treated as confidential to the patient and researcher for the purposes of this study. Interviews lasted for 45 to 90 minutes. The time the acute symptoms of ACS began was taken as the time of onset. For patients who had experienced

premonitory symptoms over preceding days or weeks, only those could specify a time point at which their symptoms had worsened significantly were included. Information concerning occupation, education, and income was collected, and participants were asked to give details about events surrounding the onset of symptoms and prior to hospital admission, such as time of call for medical help, whether the patient was alone or whether another person (a bystander) was present, who had made the decision, self medication of analgesics or indigestion remedies, which medical services were contacted (GP, ambulance, NHS Direct) and at what time, the range of symptoms experienced etc. Patients were also asked what they thought was happening when their symptoms started, ie whether they thought it was a heart problem, angina, indigestion or something else. Intensity of chest pain was measured using a numerical rating scale whereby patients were asked to give a score from 0 (none) to 10 (worst pain ever experienced) to their chest pain at the onset of their symptoms. This is an assessment tool which is often used in clinical areas to estimate the level of pain or discomfort experienced by patients (Herlitz et al, 1986; O'Connor, 1995).

Clinical information was taken from patients' medical records, such as analysis of ECG, presenting symptoms and time of onset, diagnosis, severity of ACS, medical treatment, any complications etc. Time of hospital admission was taken from the Accident and Emergency (A/E) department admission form attached to patients' medical notes (Appendix 4).

### 3.2.5: Psychosocial measures

Patients were asked to complete a set of questionnaires containing psychological and social measures. These included:

### *3.2.5.1: Socio-economic position*

Socio-economic position was assessed at baseline using a number of measures contained in the interview (Appendix 4). Patients were asked for details of their level of educational attainment. This is a widely used indicator of socio-economic position which is easily measured, applicable to people not in the active labour force as well as those in employment and stable over time. The level of reported educational attainment was categorised into three groups; no educational qualifications, up to O'level, and A'level or higher. Participants' level of yearly income was classified into three categories; less than £20 000, £20 – 40 000, and over £40 000. A deprivation index was also computed based on four criteria: living in a crowded household (defined as one or more person per room), not having access to a car or van, renting as opposed to owning their home, and being in receipt of state benefits. Participants were classified as low deprivation (negative on all items), medium deprivation (1 positive score) and high deprivation (2-4 positive items). The deprivation index gives a broader measure of social deprivation and access to resources and is based on the scale developed by Townsend (Townsend et al, 1990). Previous research had shown strong associations between social inequalities such as those mentioned above and health (Kreiger et al, 1997).

### *3.2.5.2: Social network*

Social networks at baseline were measured using the Social Network Index developed by Cohen et al (1997) as an index of the diversity of social interactions (Appendix 5). Participants were asked about the frequency of their interactions within a typical fortnightly period with 12 sets of contacts (e.g. children, friends, work colleagues). Greater values represented more diverse social networks, and scores could range from 0 to 12 (higher scores indicating larger social networks). For the purposes of analysis,

participants were categorised into three groups; small social network (0 to 3 social contacts), medium social network (4 to 5 social contacts) and large social networks (6 or more).

Social support was assessed using a scale previously shown to predict survival in elderly patients following myocardial infarction (Berkman et al, 1992). Participants were asked how many people they could count on for emotional support, and responses were allocated to four categories: no support, 1 person, 2-3 people, 4 or more people (Appendix 4, question 10, page 331).

#### *3.2.5.3: Cardiac denial of impact scale*

This 8 item questionnaire (Appendix 6) was used to measure denial of the consequences or impact associated with cardiac illness at baseline and was developed by Flowers (1992). Items such as; "I was not at all afraid when my symptoms first occurred", and "I very seldom take unnecessary risks", were scored on a 4 point Likert scale ranging from strongly disagree (1), disagree (2), agree (3), to strongly disagree (4), so that scores could range between 8 and 32. A study of 91 cardiac rehabilitation patients showed good internal reliability, indicating an alpha co-efficient of 0.72, and a 3 week test-retest reliability of 0.71 (Flowers, 1992). As noted in Chapter 2 (section 2.3.8.1), scores on the cardiac denial of impact scale have previously been associated with cardiac illness and pre-hospital delays of more than 4 hours (Flowers, 1992; O'Carroll et al, 2001).

### 3.2.6: Data Storage

All data collected was treated as confidential. Interview data, consent forms, questionnaires and follow up data were kept in a locked filing cabinet with access restricted to researchers. Data was anonymised and entered onto a computerised database which was password protected.

### 3.2.7: Statistical analyses

Data were collected using a sample of 228 participants. The data were analysed using the Statistical Package for Social Sciences (SPSS). The total pre-hospital delay period was analysed using three variables; onset of symptoms to hospital admission (pre-hospital delay), onset of symptoms to time of calling for medical help (patient decision time), and time of calling for medical help to hospital admission (home to hospital delay). Each of these variables was highly skewed, as shown below in Figures 3.1, 3.2 and 3.3. Different approaches to analysis were considered, including Spearman rank correlations but I eventually decided that the most helpful method of analysis was to divide each interval into categories (long and short), and to assess the characteristics associated with each of these. Pre-hospital delay was analysed using two categorisations, one comparing very short delays (less than 60 minutes) with longer delays, and the second comparing delays longer and shorter than the average (120 minutes). The rationale for these time intervals is presented below in section 3.3.4. Patient decision delay was analysed by comparing individuals with decision times of less than or greater than 60 minutes. Home to hospital delay was analysed by comparing delays of less than or greater than 120 minutes. Various other categorisations were tested, and the results were essentially the same as those presented here.

Information about the presence of a bystander and intensity of pain at symptom onset was available from a subset of 176 patients. The questionnaire measures detailed in section 3.2.5 were returned by 178 patients. Data were analysed using non-parametric tests including chi squared tests and logistic regression. The basic methodology was to compare the proportion of the participants in each category of delay (e.g. less than 60 minutes or more than 60 minutes) across categories of each of the independent variables using Chi squared tests of association. When effects were significant, I ran a logistic regression on the delay variables, with age and gender as covariates. In the logistic regression, the reference group was the 'longer' delay category (eg greater than 60 minutes, or greater than 120 minutes). The adjusted odds of a short delay with 95% confidence intervals are presented.

## 3.3: Results

### 3.3.1: Characteristics of the complete study population

The general characteristics of the sample population ( N = 269) are summarised in Tables 3.1 and 3.2 Patients were predominantly male, white, married, with a low level of education and a mean age of 60 ± 11 years. Just under half of the study sample (45.5%) had a yearly income of less than £20 000 and 30.5% were categorised as very deprived. Only 10.1% had a history of MI, and the majority of patients (70.3%) were admitted with a STEMI, with relatively few NSTEMI or UA (29.7%). High ratings for pain intensity of 8 or more on the 10-point scale were given by 38.9% of patients, and 45.7% had experienced premonitory symptoms in the 4 days prior to admission. Angiogram results showed that patients had an average of 1.74 ± 0.87 diseased vessels. The level of risk factors present in this sample was quite high, 45.0% had a previous medical history of hypertension, 49.2% had high cholesterol levels and 13.4% were diabetic. Nearly half (42.4%) were current smokers and most (64.0%) had a sedentary lifestyle and did not take regular exercise. Only 25.4% of participants recognised initial their symptoms as being those of a heart attack.

**Table 3.1: General characteristics of complete sample (N=269)**

| | | N (%) or mean ± SD |
|---|---|---|
| Gender: | Men | 211 (78.4) |
| | Women | 58 (21.6) |
| Age (yrs) | | 60 (±11) |
| Age categories: | <50 years | 60 (22.3) |
| | 50-60 years | 86 (32.0) |
| | 60-70 years | 58 (21.6) |
| | > 70 years | 65 (24.2) |
| Ethnicity: | White | 218 (81.4) |
| | Black/Asian | 50 (18.6) |
| Married | | 174 (64.7) |
| Educational qualifications: | A'level plus | 85 (31.6) |
| | Up to O'level | 60 (22.7) |
| | None | 123 (45.7) |
| Level of Deprivation: | Very deprived | 82 (30.5) |
| | Moderate deprivation | 69 (25.7) |
| | Not deprived | 118 (43.9) |
| Income per year: | < £20 k | 116 (45.5) |
| | £20-40 k | 79 (31.0) |
| | > £40 k | 60 (23.5) |
| Occupational Group: | Employed | 144 (53.7) |
| | Unemployed/disabled | 20 (7.5) |
| | Retired | 104 (38.8) |

**Table 3.2: Health and psychosocial characteristics of complete sample (N = 269)**

| | | N | (%) or mean ± SD |
|---|---|---|---|
| Previous MI | | 27 | (10.1) |
| Clinical presentation: | STEMI | 189 | (70.3) |
| | NSTEMI / UA | 80 | (29.7) |
| Intensity of pain (0 – 10): | <6 | 45 | (30.2) |
| | 6-8 | 46 | (30.9) |
| | 8-10 | 58 | (38.9) |
| Premonitory symptoms | | 123 | (45.7) |
| N vessels diseased | | 1.74 | (±0.87) |
| History of hypertension | | 121 | (45.0) |
| History of hypercholesterolemia | | 129 | (49.2) |
| History of diabetes | | 36 | (13.4) |
| Smoker | | 114 | (42.4) |
| Alcohol intake: | Drinker | 169 | (63.1) |
| | Non-drinker | 99 | (36.9) |
| Physical activity: | Sedentary | 171 | (64.0) |
| | Up to 2 /week | 53 | (19.9) |
| | > 2/week | 43 | (16.1) |
| Symptoms attributed to heart attack | | 68 | (25.4) |
| Bystander present | | 101 | (54.3) |
| Social network: | Small | 56 | (25.6) |
| | Medium | 89 | (40.6) |
| | Large | 74 | (33.8) |
| Emotional support: | None | 54 | (20.1) |
| | 1 person | 72 | (26.8) |
| | 2-3 people | 72 | (26.8) |
| | > 4 people | 71 | (26.4) |
| Cardiac denial of impact (tertiles): | Low | 77 | (35.3) |
| | Middle | 68 | (31.2) |
| | High | 73 | (33.5) |

### 3.3.2: Characteristics of the study population on whom information on pre-hospital delay and admission times were available

Information regarding admission time and pre-hospital delay was available for 228 patients (as explained in section 3.2.1). Comparison of characteristics of patients on whom no delay data was available (N = 41) and those on whom information on delay was available showed no significant differences except for ethnicity and day of the week on which the symptoms started (see Table 3.3). The group of patients with no data on pre-hospital delay were significantly more likely to have a lower proportion of Black/Asian patients than the group with data ($p = 0.014$). This might be because of the relatively small number of Black of Asian patients participating in the study (N = 50), and because most these patients were recruited from one particular hospital (82%) located in an area with a high Asian and Black population and where I was the principal researcher. I was therefore able to ensure data required for pre-hospital delay analysis was collected.

The proportion of patients whose symptoms started on a week day rather than at the weekend was significantly greater in the group with no data on pre-hospital delay than in the group with data ($p = 0.035$). Patients who had data on pre-hospital delay were less likely to have their symptoms start on a weekday than at the weekend than patients without data on delay (OR 2.34, $p = 0.043$). Some of these patients may have initially been admitted to outlying hospitals and then later transferred into the participating hospital so that details concerning the initial hospital admission times were not included in the referral notes. Alternatively, it may be explained by chance.

**Table 3.3: Comparison between patients with no data on delay time and patients with data included in delay analyses**

| | | No data N = 41 (%) | Data N = 228 (%) | Difference p-value ($\chi^2$) |
|---|---|---|---|---|
| **Demographic factors** | | | | |
| Age: | <50 years | 14.6 | 23.7 | .138 |
| | 50-60 years | 34.1 | 31.6 | |
| | 60-70 years | 14.6 | 22.8 | |
| | > 70 years | 36.6 | 21.9 | |
| Gender: | Men | 80.5 | 78.1 | .729 |
| | Women | 19.5 | 21.9 | |
| Ethnicity: | White | 95.1 | 78.9 | **.014** |
| | Other | 4.9 | 21.1 | |
| **Socio-economic factors** | | | | |
| Educational qualifications: | None | 61.0 | 43.0 | .086 |
| | Up to O'level | 19.5 | 23.2 | |
| | A'level + | 19.5 | 33.8 | |
| Deprivation: | Most deprived | 53.7 | 42.1 | .291 |
| | Moderately deprived | 17.1 | 27.2 | |
| | Least deprived | 29.3 | 30.7 | |
| Income per year: | <£20 k | 52.6 | 44.2 | .189 |
| | £20-£40 k | 18.4 | 33.2 | |
| | >£40 k | 28.9 | 22.6 | |
| Occupational group: | Employed | 48.8 | 54.6 | .506 |
| | Unemployed | 4.9 | 7.9 | |
| | Retired | 46.3 | 37.4 | |
| **Social factors** | | | | |
| Marital status: | Not married | 31.7 | 36.0 | .599 |
| | Married | 68.3 | 64.0 | |
| Social network: | Small | 29.3 | 24.7 | .747 |
| | Medium | 41.5 | 40.4 | |
| | Large | 29.3 | 34.8 | |
| **Contextual factors** | | | | |
| Time of symptom onset: | | | | |
| | Midnight – 0600 hrs | 14.6 | 22.8 | .268 |
| | 0600 hrs – midday | 36.6 | 30.7 | |
| | Midday – 1800 hrs | 36.6 | 26.3 | |
| | 1800 hrs - midnight | 12.2 | 20.2 | |
| Day of onset: | Week day | 80.5 | 63.6 | **.035** |
| | Weekend | 19.5 | 36.4 | |
| Season: | Jan - Mar | 24.4 | 28.5 | .396 |
| | Apr – June | 34.1 | 25.0 | |
| | July - Sept | 17.1 | 26.8 | |
| | Oct - Dec | 24.4 | 19.7 | |
| Presence of bystander: | Absent | 66.7 | 45.4 | .462 |
| | Present | 33.3 | 54.6 | |

(Continued on next page)

**Table 3.3: Continued**

| | | No data N = 41 (%) | Data N = 228 (%) | Difference p-value ($\chi^2$) |
|---|---|---|---|---|
| **Risk factors** | | | | |
| Previous MI: | No | 97.5 | 88.5 | .083 |
| | Yes | 2.5 | 11.5 | |
| Hypertension: | No | 65.9 | 53.1 | .130 |
| | Yes | 34.1 | 46.9 | |
| Hypercholesterolaemia: | No | 39.0 | 52.9 | .102 |
| | Yes | 61.0 | 47.1 | |
| Diabetes: | No | 92.7 | 85.5 | .215 |
| | Yes | 7.3 | 14.5 | |
| Smoker: | Non-smoker | 22.2 | 21.1 | .987 |
| | Ex-smoker | 36.6 | 37.7 | |
| | Smoker | 41.5 | 41.2 | |
| Alcohol intake: | Non-drinker | 31.7 | 37.9 | .451 |
| | Drinker | 68.3 | 62.1 | |
| Physical exercise: | Inactive | 55.0 | 65.6 | .375 |
| | Low (<2x per week) | 22.5 | 19.4 | |
| | High (>2x per week) | 22.5 | 15.0 | |
| **Clinical presentation** | | | | |
| Premonitory symptoms: | No | 56.1 | 53.9 | .799 |
| | Yes | 43.9 | 46.1 | |
| Type of ACS: | UA / NSTEMI | 31.7 | 70.6 | .765 |
| | STEMI | 68.3 | 29.4 | |
| **Psychological factors** | | | | |
| Attribution to heart attack: | No | 75.0 | 74.4 | .875 |
| | Yes | 24.4 | 25.6 | |
| Cardiac denial of impact: | Low | 31.7 | 36.2 | .846 |
| ( tertiles) | Middle | 34.1 | 30.5 | |
| | High | 34.1 | 33.3 | |

### 3.3.3 Analysis of pre-hospital delay

Patients (N = 228) were interviewed an average 2.56 ± 1.5 days after admission, with 95% being completed within 5 days of admission. The characteristics of the sample are summarised in Tables 3.4 and 3.5. Patients were predominantly male, white, married, with a low level of education and a mean age of 59.0 ± 11.2 years. Forty two per cent of participants had a yearly income of less than £20 000 and 30.7% were categorised as very deprived. Only 11.5% had a history of MI, and the majority of patients (70.6%) were admitted with a STEMI, with relatively few NSTEMI or UA (29.4%). High ratings for pain intensity of 8 or more on the 10-point scale were given by 39.2% of patients, and 46.1% had experienced premonitory symptoms in the 4 days prior to admission. Angiogram results showed that patients had an average of 1.8 ± 0.84 diseased vessels. The level of risk factors present in this sample was quite high, 46.9% had a previous medical history of hypertension, 47.1% had high cholesterol levels and 13.5% were diabetic. Nearly half (42.5%) were current smokers and most (65.6%) had a sedentary lifestyle and did not take regular exercise. Only 28.7% of participants initially recognised their symptoms as being those of a heart attack.

**Table 3.4: Characteristics of participants in the delay analyses**

| | | N (%) or mean ± SD |
|---|---|---|
| Gender: | Men | 178 (78.1) |
| | Women | 50 (29.9) |
| Age (yrs) | | 59.0 ± SD 11.2 |
| Ethnicity: | White | 180 (78.9) |
| | Black/Asian | 48 (21.1) |
| Married | | 146 (64.0) |
| Educational qualifications: | A'level plus | 77 (33.8) |
| | Up to O'level | 53 (23.2) |
| | None | 98 (43.0) |
| Level of Deprivation: | Very deprived | 70 (30.7) |
| | Moderate deprivation | 62 (27.2) |
| | Not deprived | 96 (42.1) |
| Income per year: | < £20 k | 96 (42.1) |
| | £20-40 k | 72 (30.6) |
| | > £40 k | 49 (21.5) |

**Table 3.5: Health and psychosocial characteristics of patients in the delay analyses**

| | | N | (%) or mean ± SD |
|---|---|---|---|
| Previous MI | | 26 | (11.5) |
| Clinical presentation: | STEMI | 161 | (70.6) |
| | NSTEMI / UA | 67 | (29.4) |
| Intensity of pain (0 – 10): | <6 | 45 | (30.4) |
| | 6-8 | 45 | (30.4) |
| | 8-10 | 58 | (39.2) |
| Premonitory symptoms | | 105 | (46.1) |
| N vessels diseased | | 1.8 | SD 0.84 |
| History of hypertension | | 107 | (46.9) |
| History of hypercholesterolemia | | 104 | (47.1) |
| History of diabetes | | 33 | (13.5) |
| Smoker | | 97 | (42.5) |
| Alcohol intake: | Drinker | 141 | (62.1) |
| | Non-drinker | 86 | (37.9) |
| Physical activity: | Sedentary | 149 | (65.6) |
| | Up to 2 /week | 44 | (19.4) |
| | > 2/week | 34 | (15.0) |
| Symptoms attributed to heart attack | | 58 | (25.6) |
| Bystander present | | 100 | (54.6) |
| Social network: | Small | 44 | (24.7) |
| | Medium | 72 | (40.4) |
| | Large | 62 | (34.8) |
| Emotional support: | None | 46 | (20.2) |
| | 1 person | 63 | (27.6) |
| | 2-3 people | 61 | (26.8) |
| | > 4 people | 25 | (25.4) |
| Cardiac denial of impact (tertiles): | Low | 64 | (36.2) |
| | Middle | 54 | (30.5) |
| | High | 59 | (33.3) |

### 3.3.4 Overall pattern and analysis of pre-hospital delay

The time from symptom onset to admission ranged from 10 minutes to 4.34 days. The overall mean for pre-hospital delay was 6.1 hours (±12.9 hours). As can be seen in Figure 3.1, the distribution was highly skewed. An average of 17.1% of participants were admitted to hospital within 60 minutes of symptom onset and 50% were admitted within 124 minutes. It therefore seemed reasonable to define 60 minutes as the threshold for short delays, and 120 minutes for average delays.

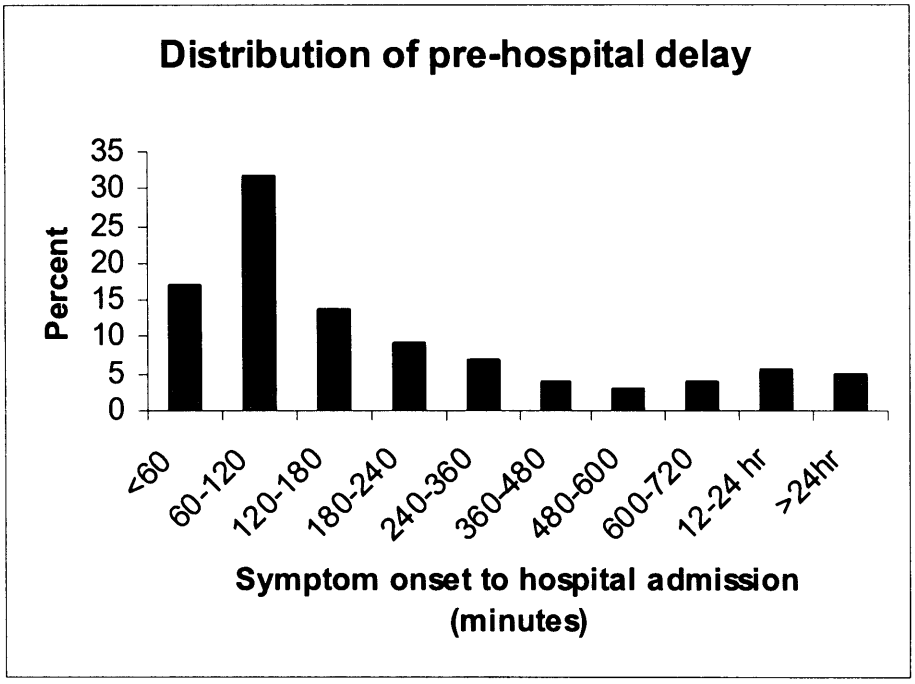


**Figure 3.1: Distribution of pre-hospital delay**

### *3.3.4.1 Distribution of patient decision time*

The time from symptom onset to decision to call for medical help ranged from 0 to 2.65 days. The overall mean for patient decision time was 3.6 hours ±7.96 hours. A total of 44.1% of participants called for medical help within 30 minutes of symptom onset and 60.8% within 60 minutes. The median decision time was 50 minutes (see Figure 3.2).

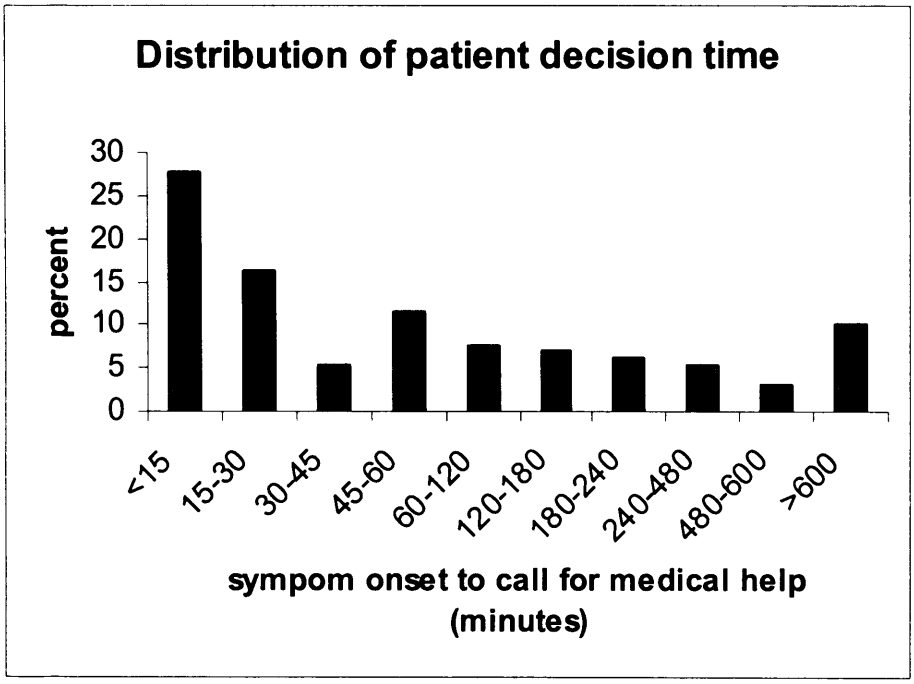


**Figure 3.2: Distribution of patient decision time**

### *3.3.4.2 Distribution of home to hospital delay*

The time from the call for help to hospital admission ranged from 2 minutes to 4.1 days. The overall mean was 2.4 hours (± 8.9 hours). The median time was 58 minutes (see Figure 3.3). Although not all patients were at home when their symptoms started, most (64.8%) were, so it seemed appropriate to term this phase of pre-hospital delay home to hospital delay.

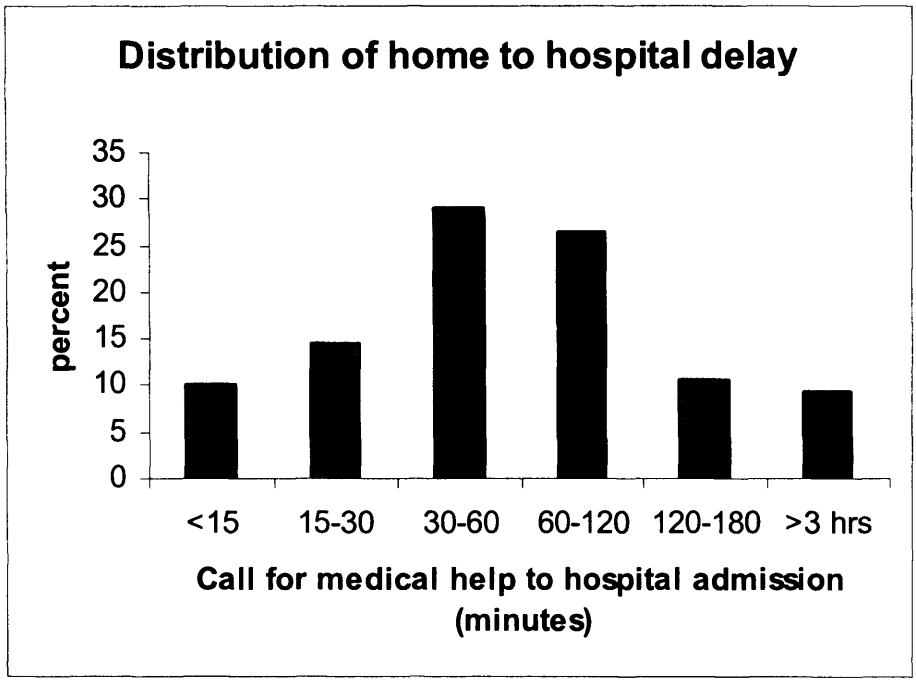


**Figure 3. 3: Distribution of home to hospital delay**

Home to hospital delay incorporated the time between calling for help (or making a definite decision to seek help in the case of patients who decided to refer themselves directly to A&E) and receiving help in a hospital (median time of 10 minutes, 8.2% of the total pre-hospital delay time), and includes time taken for medical assessment by paramedics/GP etc, and time travelling to hospital (median time 48 minutes, 31.8% of the total time)(see Figure 3.4 ). Patients were also asked to recall the time from the call for help to receiving medical assistance. This time interval ranged from zero, when a patient happened to be at a GP surgery and received treatment immediately, to 16.4

hours (50% of patients received medical treatment within 10 minutes ± 1.3 hours of calling for help). Patients were generally much less confident, however, that they could remember this time interval accurately and it has therefore not been analysed separately in further detail but included within the home to hospital delay period.

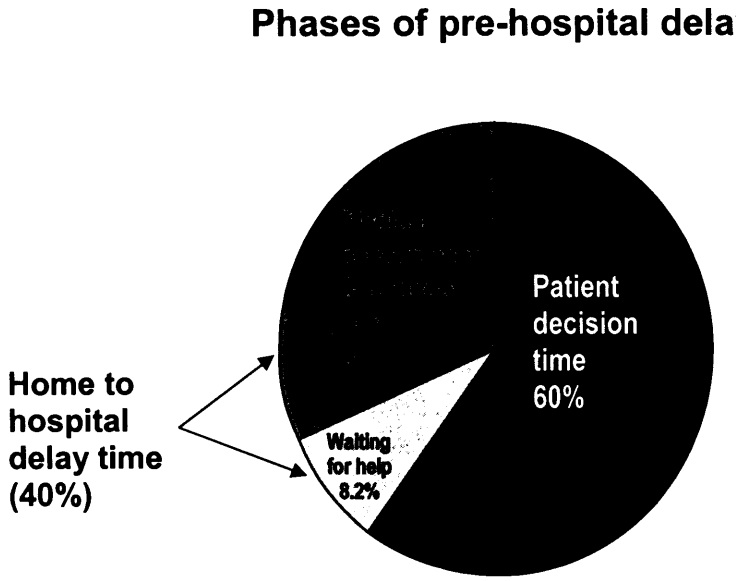


**Figure 3.4: Distribution of phases of pre-hospital delay**

### *3.3.4.3 Relationship between reported delay and interview timing*

There was a possibility that patients' recall of their delay times was distorted by the time between admission and interview. Patients who were interviewed later may have recalled different delay periods. This was tested by correlating the interval between admission and interview with the 2 phases of pre-hospital delay. None of the effects were significant, and there was no correlation between interval between admission and total pre-hospital delay ($r = -0.087$, $p = 0.189$).

### 3.3.5 Description of patients' experience during the pre-hospital delay period

As shown in Table 3.6, most patients were at home when their symptoms started (64.8%), some were outside walking to work or talking to neighbours etc (13.2%), or at work (10.2%) or in a variety other places such as on the underground train, in the car, at the gym etc (11.9%). There was little variation in the number of events depending on time of day, day of the week and season.

**Table 3.6: Location of patient, timing and attribution of symptoms at onset**

| | | N (%) |
|---|---|---|
| Location at onset: | At home | 147 (64.8) |
| | Outside | 30 (13.2) |
| | At work | 23 (10.1) |
| | Other | 27 (11.9) |
| Time of symptom onset: | Midnight – 0600 | 52 (22.8) |
| | 0600 – Midday | 70 (30.7) |
| | Midday – 1800 | 60 (26.3) |
| | 1800 – midnight | 46 (20.2) |
| Day of onset: | Weekday | 145 (63.6) |
| | Weekend | 83 (36.4) |
| Season of symptom onset: | Jan – Mar | 65 (28.5) |
| | Apr – Jun | 57 (25.0) |
| | Jul – Aug | 61 (26.8) |
| | Sept – Dec | 45 (19.7) |
| Attribution of symptoms | Indigestion | 78 (34.2) |
| | Heart attack | 58 (25.4) |
| | Angina | 23 (10.3) |
| | Other | 41 (18.0) |
| | Don't know | 28 (12.1) |

The most frequent initial attribution of symptoms was to indigestion (34.2%), followed by a heart attack (25.4%), angina (10.3%) and other causes such as muscle strain and flu (18.0%). Following the onset of symptoms, patients took a variety of actions prior to

calling for help. Patients used a variety of strategies to try to relieve their symptoms, such as resting or taking antacids before calling for help (see Figure 3.7). There were no significant associations, however, between delay time and particular type or number of strategies used. When asked why they delayed in seeking help, 68.1% of patients who answered (N=144) said that they were waiting to see if the pain would go away, 21.5% said that they did not initially think that their symptoms were serious, 6.3% said they did not want to trouble anyone and 4.2% said that they were arranging baby sitters or care of a dependent prior to contacting medical help for themselves.

**Table 3.7: Strategies used by patients to try and relieve symptoms prior to calling for medical help**

| Strategies patients tried to relieve symptoms | Percentage of patients (N=178)* |
|---|---|
| Rested – lay or sat down for a while | 66.9 |
| Took indigestion remedies | 15.7 |
| Took analgesics | 16.3 |
| Took GTN spray | 9.0 |
| Had a non-alcoholic drink (water/tea/milk) | 27.0 |
| Walked around | 18.0 |

(*Some patients used more than one coping strategy).

Having decided that their symptoms were serious, an important factor involved in total pre-hospital delay, decision time, home to hospital delay was who the patient contacted first for help. Shorter patient decision times, home to hospital delay and total pre-hospital delays were found in patients who called for an ambulance first rather than the GP/NHS Direct, went directly to an A/E department or contacted a family member or friend (see Table 3.8).

**Table 3.8: Initial call for help and average time from onset to decision to seek help, and from onset to hospital admission**

| First contact following symptom onset | Patients % (N =170) | Range of decision times (median time in mins) | Range of home to hospital delay (median time in mins) | Range of total pre-hospital delay (median delay in mins) |
|---|---|---|---|---|
| **Ambulance** | 45.0 | 2 mins - 30hrs (20) | 9 mins - 4.45 hrs (50) | 25 mins - 30.7 hrs (89) |
| **GP/NHS Direct** | 22.9 | 0 mins - 24hrs (120) | 12 mins - 3.3 days (71) | 64 mins - 104.1 hrs (204) |
| **A/E (self-referral)** | 18.8 | 5 mins - 34.2hrs (75) | 5 mins - 3hrs (37) | 10 mins - 34.4 hrs (185) |
| **Family/friend** | 13.5 | 10 mins - 46.7hrs (90) | 4 mins - 5.4 hrs (60) | 40 mins - 49.8 hrs (188) |

These effects were analysed in a series of logistic regressions summarised in Table 3.9. Patients who contacted the emergency ambulance service in the first instance for medical help were more likely to have a short pre-hospital delay of less than 120 minutes than patients who contacted other services or their family / friends. Patients who contacted their GP / NHS Direct first were less likely to have a short pre-hospital delay of less than 120 minutes than patients who contacted the Ambulance Services (OR 0.14, $p < 0.000$), referred themselves to an A&E department (OR 0.22, $p = 0.001$), or called a family member/friend (OR 0.19, $p = 0.001$). Similar results were found for patient decision time. Patients who contacted their GP/NHS Direct initially were less likely to have a short decision time of less than 60 minutes than patients who contacted the ambulance service first (OR 0.17, $p < 0.000$), referred themselves to an A&E department (OR 0.27, $p = 0.006$), or called a family member/friend (OR 0.19, $p = 0.001$). The results for home to hospital delay also reflect this pattern. Patients who contacted their GP/NHS Direct were less likely to have a short home to hospital delay than those who contacted the ambulances service first (OR 0.11, $p < 0.000$), referred

themselves to an A&E department (OR 0.28, p = 0.095), or called a family member/friend (OR 0.14, p = 0.007).

**Table 3.9: Logistic regressions for patients' first contact following onset of symptoms**

**Total pre-hospital delay**

| Initial contact for help: | Delay <120mins (%) | Delay >120 mins (%) | p-value ($\chi^2$) | Adjusted odds ratio (95% C.I.) of delay of less than 120mins | Adjusted p-value* |
|---|---|---|---|---|---|
| Ambulance | 74.0 | 26.0 | <0.001 | 1 | |
| GP/NHS Direct | 28.2 | 71.8 | | 0.14 (0.06 -0.32) | **<0.001** |
| A/E self-referral | 37.5 | 62.5 | | 0.22 (0.09 -0.53) | **.001** |
| Family/friend | 34.8 | 65.2 | | 0.19 (0.07 -0.52) | **.001** |

**Patient decision time delay**

| Initial contact for help: | Decision time <60 mins (%) | Decision time >60 mins (%) | p-value ($\chi^2$) | Adjusted odds ratio (95% C.I.) of delay of less than 60 mins | Adjusted p-value* |
|---|---|---|---|---|---|
| Ambulance | 80.3 | 19.7 | <0.001 | 1 | |
| GP/NHS Direct | 43.6 | 56.4 | | 0.17 (0.07 - 0.42) | **<0.001** |
| A/E self-referral | 50.0 | 50.0 | | 0.27 (0.11 - 0.68) | **.006** |
| Family/friend | 43.5 | 56.5 | | 0.19 (0.07 - 0.52) | **.001** |

**Home to hospital delay**

| Initial contact for help: | Home to hospital delay <120 mins | Home to hospital delay >120 mins | p-value ($\chi^2$) | Adjusted odds ratio (95% C.I.) of delay of less than 120mins | Adjusted p-value* |
|---|---|---|---|---|---|
| Ambulance | 94.7 | 5.3 | <0.001 | 1 | |
| GP/NHS direct | 64.1 | 35.9 | | 0.11 (0.03 - 0.38) | **<0.001** |
| A/E self-referral | 87.5 | 12.5 | | 0.28 (0.06 - 1.25) | .095 |
| Family/friend | 73.9 | 26.1 | | 0.14 (0.03 - 058) | **.007** |

*Adjusted for age and gender.

It is notable that despite having symptoms of ACS, only 45% of patients initially sought help via the emergency ambulance services (Table 3.8), while 22.9% preferred to call their GP or NHS Direct to seek guidance, 18.8% referred themselves directly to an A&E department and 13.5% called a family member or friend.

### 3.3.6: Factors associated with total pre-hospital delay

#### *3.3.6.1: Factors associated with a very short pre-hospital delay (less than 60 minutes).*

Socio-demographic factors such as age, gender, ethnicity, and marital status did not show any significant associations with pre-hospital delay of less than 60 minutes, although level of income approached significance (see Table 3.10). A non-linear association with education was observed. Participants who had received education up to O'level were more likely to have short delays compared with those with no qualifications. Logistic regression analysis indicated that the odds ratio (OR) adjusted for age and gender for admission to hospital within 60 minutes of symptom onset for participants with O'levels compared with participants with no educational qualifications was 2.87 ($p = 0.019$). Participants who had low scores on the deprivation index and did not experience deprivation were more likely to have short pre-hospital delays. The OR of pre-hospital delay of less than 60 minutes was 3.07 ($p = 0.017$) for the least deprived group, adjusted for age and gender, compared with those who had high scores and were classified as deprived. Participants who had a large social network also had shorter pre-hospital delays, OR 3.99 ($p = 0.040$) than those with small social networks.

**Table 3.10: Very short (<60 min) total pre-hospital delays and sociodemographic factors**

| | | Delay <60 mins (%) | Delay >60 mins (%) | p-value ($\chi^2$) | Odds ratio (95% C.I.) of delay < 60 min adjusted for age and gender | p-value |
|---|---|---|---|---|---|---|
| **Demographic factors** | | | | | | |
| Age: | <50 years | 22.6 | 77.8 | .289 | | |
| | 50-60 years | 20.8 | 79.2 | | | |
| | 60-70 years | 11.5 | 88.5 | | | |
| | > 70 years | 21.9 | 88.0 | | | |
| Gender: | Men | 17.4 | 82.6 | .814 | | |
| | Women | 16.0 | 84.0 | | | |
| Ethnicity: | White | 15.6 | 84.4 | .229 | | |
| | Other | 22.9 | 77.1 | | | |
| **Socio-economic factors** | | | | | | |
| Educational qualifications: | None | 11.2 | 88.8 | **.029** | 1 | |
| | Up to O'level | 28.3 | 71.7 | | 2.87 (1.23–7.72) | **.019** |
| | A'level + | 16.9 | 83.1 | | 1.49 (0.62 – 3.59) | .375 |
| Deprivation: | Most deprived | 10.0 | 90.0 | **.051** | 1 | |
| | Moderately deprived | 14.5 | 85.5 | | 1.52 (0.52 – 4.39) | .444 |
| | Least deprived | 24.0 | 76.0 | | 3.07 (1.23–7.72) | **.017** |
| Income per year: | <£20 k | 11.5 | 88.5 | .071 | 1 | |
| | £20-£40 k | 18.1 | 81.9 | | 1.53 (0.62 – 3.78) | .350 |
| | >£40 k | 26.5 | 73.5 | | 2.46 (0.93 – 6.48) | .069 |
| **Social factors** | | | | | | |
| Marital status: | Not married | 12.2 | 87.8 | .140 | | |
| | Married | 19.9 | 80.1 | | | |
| Social network: | Small | 6.8 | 93.2 | **.046** | 1 | |
| | Medium | 13.9 | 86.1 | | 2.24 (0.58 – 8.71) | .244 |
| | Large | 24.2 | 75.8 | | 3.99 (1.06-4.83) | **.040** |
| **Contextual factors** | | | | | | |
| Time of symptom onset: | | | | | | |
| | Midnight - 0600 hrs | 15.4 | 84.6 | .594 | | |
| | 0600 hrs - midday | 15.7 | 84.3 | | | |
| | Midday - 1800 hrs | 15.0 | 85.0 | | | |
| | 1800 hrs - midnight | 23.9 | 76.1 | | | |
| Day of onset: | Week day | 17.9 | 82.1 | .662 | | |
| | Weekend | 15.7 | 84.3 | | | |
| Season: | Jan - Mar | 21.5 | 78.5 | .644 | | |
| | Apr - June | 17.5 | 82.5 | | | |
| | July - Sept | 13.1 | 86.9 | | | |
| | Oct - Dec | 15.6 | 84.4 | | | |
| Presence of bystander: | Absent | 13.3 | 86.7 | .226 | | |
| | Present | 20.0 | 80.0 | | | |

Additionally, patients who attributed their symptoms to a heart attack (see Table 3.11) delayed less, OR 2.25 ($p = 0.012$) compared with participants who did not recognise their symptoms. Patients who had higher scores on the cardiac denial of impact scale were significantly less likely to be admitted to hospital within 60 minutes of symptom onset, the ORs for patients in the middle and upper tertiles of this scale were 0.30 ($p = 0.029$) and 0.34 ($p = 0.038$) respectively, compared to patients who had scores within the lowest tertile of this scale.

Other risk factors such as history of hypertension, diabetes, smoking, level of physical exercise or type of ACS i.e. unstable angina (UA) or non-ST segment elevation MI (NSTEMI) or ST segment elevation MI (STEMI) did not show any significant association with pre-hospital delays of less than 60 minutes. Neither were temporal factors such as time of day of onset, day of week or season related to very short total pre-hospital delays.

**Table 3.11: Very short (<60 min) total pre-hospital delays and clinical factors**

| | | Delay <60 mins (%) | Delay >60 mins (%) | p-value ($\chi^2$) | Odds ratio (95% C.I.) of delay < 60 min adjusted for age and gender | p-value |
|---|---|---|---|---|---|---|
| **Risk factors** | | | | | | |
| Previous MI: | No | 16.4 | 83.6 | .397 | | |
| | Yes | 23.1 | 76.9 | | | |
| Hypertension: | No | 18.2 | 81.8 | .646 | | |
| | Yes | 15.9 | 84.1 | | | |
| Hypercholesterolaemia: | No | 13.7 | 86.3 | .100 | | |
| | Yes | 22.1 | 77.9 | | | |
| Diabetes: | No | 16.9 | 3.1 | .859 | | |
| | Yes | 18.2 | 81.8 | | | |
| Smoker: | Non-smoker | 18.8 | 81.3 | .821 | | |
| | Ex-smoker | 15.1 | 84.9 | | | |
| | Smoker | 18.1 | 81.9 | | | |
| Alcohol intake: | Non-drinker | 20.9 | 79.1 | .242 | | |
| | Drinker | 14.9 | 85.1 | | | |
| Physical exercise: | Inactive | 14.8 | 85.2 | .399 | | |
| | Low(<2x per week) | 22.7 | 77.3 | | | |
| | High (>2x per week) | 20.6 | 79.4 | | | |
| **Clinical presentation** | | | | | | |
| Premonitory symptoms: | No | 18.7 | 81.3 | .489 | | |
| | Yes | 15.2 | 84.8 | | | |
| Type of ACS: | UA / NSTEMI | 11.9 | 88.1 | .187 | | |
| | STEMI | 19.3 | 80.7 | | | |
| Intensity of pain | < 6 | 17.8 | 82.2 | .253 | | |
| | 6-8 | 22.2 | 77.8 | | | |
| | 8-10 | 10.3 | 89.7 | | | |
| **Psychological factors** | | | | | | |
| Attribution to heart attack: | No | 13.0 | 87.0 | **.010** | 1 | **.012** |
| | Yes | 27.6 | 72.4 | | 2.25 (1.24–6.40) | |
| Cardiac denial of impact: | Low | 26.6 | 73.4 | **.013** | 1 | |
| (tertiles) | Middle | 9.3 | 90.7 | | .30 (0.10 – 0.88) | **.029** |
| | High | 10.2 | 89.8 | | .34 (0.12 – 0.94) | **.038** |

### *3.3.6.2 Factors associated with less than average pre-hospital delay (120 minutes).*

The second set of analyses compared patients with pre-hospital delays greater or less than average (120 minutes). Socio-demographic factors such as age, gender, ethnicity, education, and level of deprivation did not show any significant association with pre-hospital delays of less than 120 minutes (see Table 3.12). Married participants, however, were more likely to have short pre-hospital delays compared with participants who were not married. Logistic regression indicated that the OR, adjusted for age and gender, of being admitted to hospital in within 120 minutes of symptom onset for married participants compared with participants who were not married was 1.77 ($p = 0.048$). Participants whose symptoms began in the afternoon were more likely to have a shorter than average pre-hospital delay. The OR of pre-hospital delay of less than 120 minutes was 2.76 ($p = 0.009$) for those whose symptoms began in the afternoon, adjusted for age and gender, compared with those whose symptoms started at night. Day of the week and season did not show any significant association.

Patients with a previous history of MI were more likely to be admitted to hospital within 120 minutes of symptom onset, OR 2.80 ($p = 0.023$) compared with participants with no such previous medical history. Patients who had an ST segment elevation AMI also had a shorter than average delay, OR 1.93 ($p = 0.028$) compared with patients who had either NSTEMI or unstable angina (see Table 3.13). Patients who attributed their symptoms to a heart attack delayed less, OR 2.00 ($p = 0.040$) compared with those who did not recognise their symptoms. If the participant was with a bystander, they were also more likely to be admitted within 120 minutes, OR 1.97 ($p = 0.042$) compared with participants who were alone at the time of symptom onset. Other risk factors such as history of hypertension, diabetes, smoking, level of physical exercise did not show any significant association with pre-hospital delays of less than 120 minutes.

**Table 3.12: Less than average (<120 minutes) pre-hospital delays & sociodemographic factors**

| | | Delay<120 mins (%) | Delay>120 mins (%) | p-value ($\chi^2$) | Adjusted odds ratio (95% C.I.)*of delay of less than 120mins | Adjusted p-value* |
|---|---|---|---|---|---|---|
| **Demographic factors** | | | | | | |
| Age: | <50 years | 48.1 | 1.9 | .695 | | |
| | 50-60 years | 54.2 | 45.8 | | | |
| | 60-70 years | 46.2 | 53.8 | | | |
| | >70 years | 44.0 | 56.0 | | | |
| Gender: | Men | 48.3 | 51.7 | .833 | | |
| | Women | 50.0 | 50.0 | | | |
| Ethnicity: | White | 54.2 | 45.8 | .392 | | |
| | Other | 47.2 | 52.8 | | | |
| **Socio-economic factors** | | | | | | |
| Educational qualifications: | | | | | | |
| | None | 44.9 | 55.1 | .593 | | |
| | Up to O'level | 52.8 | 47.2 | | | |
| | A'level + | 50.6 | 49.4 | | | |
| Deprivation: | Most deprived | 47.1 | 2.9 | .094 | | |
| | Moderately deprived | 38.7 | 61.3 | | | |
| | Least deprived | 56.3 | 43.8 | | | |
| Income per year: | <£20 k | 43.8 | 56.3 | .386 | | |
| | £20-40 k | 54.2 | 45.8 | | | |
| | > £40 k | 51.0 | 49.0 | | | |
| **Social factors** | | | | | | |
| Marital status: | Not married | 40.2 | 59.8 | **.056** | 1 | **.048** |
| | Married | 53.4 | 46.6 | | 1.77 (1.01–3.11) | |
| Social network: | Small | 5.5 | 4.5 | .821 | | |
| | Medium | 48.6 | 51.4 | | | |
| | Large | 51.6 | 48.4 | | | |
| **Contextual factors** | | | | | | |
| Time of symptom onset: | | | | | | |
| | Midnight – 0600 hrs | 38.5 | 61.5 | **.029** | 1 | |
| | 0600 hrs – midday | 41.4 | 58.6 | | 1.15 (0.55–2.42) | .704 |
| | Midday – 1800 hrs | 63.3 | 36.7 | | 2.76 (1.28–5.96) | **.009** |
| | 1800 hrs - midnight | 52.2 | 47.8 | | 1.76 (0.78–3.93) | .171 |
| Day of week of onset | | | | | | |
| | Week day | 47.6 | 52.4 | .661 | | |
| | Weekend | 50.6 | 49.4 | | | |
| Season: | Jan – Mar | 50.8 | 49.2 | .873 | | |
| | Apr – June | 49.1 | 50.9 | | | |
| | July – Sept | 44.3 | 55.7 | | | |
| | Oct - Dec | 51.1 | 48.9 | | | |
| Presence of bystander: | Absent | 42.2 | 57.8 | **.041** | 1 | |
| | Present | 58.9 | 41.1 | | 1.97 (1.03–3.79) | **.042** |
| Who made decision to call for help: | Patient | 39.8 | 60.2 | **.006** | 1 | **.006** |
| | Bystander | 60.0 | 40.0 | | 2.29 (1.26– 4.15) | |

*Adjusted for age and gender

There were also some interesting results regarding the pattern of symptoms, as summarised in Table 3.13. Patients presenting with 4 or more symptoms as well as chest pain (such as pains in their arms, jaw or back etc) were more likely to have a shorter than average total pre-hospital delay, (OR 3.03, $p = 0.007$) compared with patients who experienced only chest pain. Patients who reported that they suffered from more than 3 types of non-pain symptoms (such a shortness of breath, nausea and vomiting, dizziness) were also more likely to have shorter than average delays compared with those who had no symptoms other than chest pain (OR 3.49, $p = 0.002$).

**Table 3.13 Less than average (<120 minutes) pre-hospital delays and clinical factors**

| | | Delay <120 mins (%) | Delay >120 mins (%) | p-value ($\chi^2$) | Adjusted odds ratio (95% C.I.) delay <120 mins | Adjusted p-value* |
|---|---|---|---|---|---|---|
| **Risk factors** | | | | | | |
| Previous MI: | No | 46.3 | 53.7 | **.028** | 1 | |
| | Yes | 69.2 | 30.8 | | 2.80 (1.15–6.82) | **.023** |
| Hypertension: | No | 52.9 | 47.1 | .176 | | |
| | Yes | 43.9 | 56.1 | | | |
| Hypercholesterolemia: | No | 44.4 | 55.6 | .068 | 1 | |
| | Yes | 56.7 | 43.3 | | 1.65 ( .97–2.81) | .067 |
| Diabetes: | No | 49.2 | 50.8 | .688 | | |
| | Yes | 45.5 | 54.5 | | | |
| Smoker: | Non-smoker | 50.0 | 50.0 | .730 | | |
| | Ex-smoker | 45.3 | 54.7 | | | |
| | Smoker | 51.1 | 48.9 | | | |
| Alcohol intake: | Non-drinker | 55.8 | 44.0 | | | |
| | Drinker | 44.2 | 56.0 | .083 | | |
| Physical exercise: | Inactive | 47.0 | 53.0 | .152 | | |
| | Low (<2x per week) | 61.4 | 38.6 | | | |
| | High (>2x per week) | 41.2 | 58.8 | | | |
| **Clinical presentation** | | | | | | |
| Type of ACS: | UA/NSTEMI | 37.3 | 62.7 | **.027** | 1 | |
| | STEMI | 53.4 | 46.6 | | 1.93 (1.03–3.90) | **.028** |
| Premonitory symptoms: | No | 52.0 | 48.0 | .274 | | |
| | Yes | 44.8 | 55.2 | | | |
| Intensity of pain**: | <6 | 46.7 | 53.3 | .677 | | |
| | <6-8 | 53.5 | 46.7 | | | |
| | 8-10 | 55.2 | 44.8 | | | |
| **Symptoms at onset** | | | | | | |
| Number of non-chest pain symptoms: | None | 37.1 | 62.9 | **.005** | 1 | |
| | 1-3 | 43.3 | 56.7 | | 1.34 (0.62-2.93) | .460 |
| | 4-8 | 65.2 | 34.8 | | 3.03 (1.39-7.86) | **.007** |
| Number of non-pain symptoms: | None | 36.2 | 63.8 | **.001** | 1 | |
| | 1-2 | 42.6 | 57.4 | | 1.33 (0.65- 2.73) | .429 |
| | 3-6 | 65.8 | 34.2 | | 3.49 (1.60-7.58) | **.002** |
| **Psychological factors** | | | | | | |
| Attribution to heart attack: | | | | | | |
| . | No | 44.4 | 55.6 | **.036** | 1 | |
| | Yes | 60.3 | 39.7 | | 1.90 (1.03 - 3.49) | **.039** |
| Cardiac denial of impact: | | | | | | |
| | Low | 60.9 | 39.1 | **.058** | 1 | |
| | Middle | 40.7 | 59.3 | | 0.46 (0.22 – 0.96) | **.039** |
| | High | 44.1 | 55.9 | | 0.54 (0.26 – 1.12) | **.095** |
| | Lowest tertile | 60.9 | 39.1 | **.018** | 1 | |
| | Higher tertiles | 42.5 | 57.5 | | .50 (0.26 - 0.94) | **.030** |

*Adjusted for age and gender. **Pain intensity scored on visual scale from 1 to 10 (mild to worst pain ever felt).

As mentioned previously (see section 3.3.5) patients who contacted the ambulances service in the first instance for medical help were more likely to have a shorter than average pre-hospital delay (less than 120 minutes) than patients who contacted other services or their family / friends (see Table 3.9). Patients who contacted their GP / NHS Direct first were less likely to have a pre-hospital delay of less than average than patients who contacted the emergency ambulance services (OR 0.14, $p < 0.001$), referred themselves to an A&E department (OR 0.22, $p = 0.001$), or called a family member/friend (OR 0.19, $p = 0.001$).

There were also some interesting results regarding cardiac denial of impact. Data were analysed firstly by dividing patients' scores into tertiles. The association between the three groups and pre-hospital delay of less than average (120 minutes) approached significance ($p = 0.058$). Logistic regression analyses showed that compared with the patients in the lowest tertile (lowest scores), patients in the middle tertile were less likely to have a pre-hospital delay below average, OR 0.46 ($p = 0.039$), while no association was found with patients in the highest tertile 0.54 ( $p = 0.095$), adjusted for age and gender. During further analysis of this data, however, the middle and highest tertiles were combined and compared with patients who had scores in the lowest tertile. This revealed that patients with higher scores on the cardiac denial of impact scale were significantly less likely than patients with scores in the lowest tertile to have a pre-hospital delay of less than average, OR 0.50 ($p = 0.030$), adjusted for age and gender.

In summary, very short pre-hospital delays of less than 60 minutes were associated with having an education to at least O level, not being deprived, recognising the symptoms as being those of a heart attack, having a larger social network and scoring within the lowest tertile of cardiac denial of impact scale. Pre-hospital delays of less than average

(120 minutes) were associated with being married, having symptom onset in the afternoon, having a previous history of MI, having a ST segment elevation AMI, recognising the symptoms as being those of a heart attack, having a bystander present, suffering from a greater number and range of symptoms and having a lower level of cardiac denial. Patients who contacted the ambulance service to call for help at the first instance were also more likely to have a shorter than average pre-hospital delay.

### 3.3.7 Factors associated with short patient decision times (less than 60 minutes)

The associations between socio-demographic factors and decision times of less than 60 minutes are summarised in Table 3.14. Factors such as age, gender, ethnicity, educational qualifications, deprivation index, time of onset and size of social network did not show any significant association with decision time of less than 60 minutes. Patients who were married, however, were more likely to have a decision time of less than 60 minutes, OR 1.85 ($p = .034$) than those who were not married. Patients who were with a bystander were also more likely to have a short delay of less than 60 minutes, OR 2.35 ($p=.006$), compared with those who were alone, and if the bystander was not a family member the decision time was more likely to be shorter than 60 minutes , OR 2.36 ($p = 0.04$) than if the bystander was a family member.

**Table 3.14 Patient decision time and sociodemographic factors**

| | | Decision time<60mins (%) | Decision time>60mins (%) | p-value ($\chi^2$) | Adjusted odds ratio (95% C.I.) of delay of less than 60 mins | Adjusted p-value* |
|---|---|---|---|---|---|---|
| **Demographic factors** | | | | | | |
| Age: | <50 years | 57.4 | 42.6 | .712 | | |
| | 50 – 60 | 58.3 | 41.7 | | | |
| | 60 – 70 | 67.3 | 32.7 | | | |
| | > 70 years | 61.2 | 38.8 | | | |
| Gender: | Men | 59.9 | 40.1 | | | |
| | Women | 64.0 | 36.0 | .599 | | |
| Ethnicity: | White | 60.9 | 39.6 | .952 | | |
| | Black/Asian | 60.4 | 39.1 | | | |
| **Socio-economic factors** | | | | | | |
| Educational qualifications: | | | | | | |
| | None | 58.2 | 41.8 | .775 | | |
| | Up to O'level | 62.3 | 37.7 | | | |
| | A'level + | 63.2 | 36.8 | | | |
| Deprivation: | Most deprived | 63.2 | 36.8 | .775 | | |
| | Moderately deprived | 62.3 | 37.7 | | | |
| | Least deprived | 58.2 | 41.8 | | | |
| Income per year: | <£20 k | 58.9 | 41.1 | .662 | | |
| | £20 – £40k | 55.6 | 44.4 | | | |
| | >£40k | 64.3 | 35.7 | | | |
| **Social factors** | | | | | | |
| Marital status: | Not married | 52.4 | 47.6 | **.058** | 1 | |
| | Married | 65.5 | 34.5 | | 1.85 (1.05 – 3.28) | **.034** |
| Social Network: | Small | 51.2 | 48.8 | .482 | | |
| | Medium | 62.5 | 37.5 | | | |
| | Large | 59.7 | 40.3 | | | |
| **Contextual factors** | | | | | | |
| Time of onset: | | | | | | |
| | Midnight – 0600 | 50.0 | 50.0 | .155 | | |
| | 0600 – midday | 60.0 | 40.0 | | | |
| | Midday to 1800 | 71.2 | 28.8 | | | |
| | 1800 - midnight | 60.9 | 39.1 | | | |
| Day of the week: | Weekday | 60.4 | 39.6 | .878 | | |
| | Weekend | 61.4 | 38.6 | | | |
| Season of onset: | Jan – Mar | 57.8 | 42.2 | .952 | | |
| | Apr – Jun | 61.4 | 38.6 | | | |
| | Jul – Sept | 62.3 | 37.7 | | | |
| | Oct – Dec | 62.2 | 37.8 | | | |
| Presence of a bystander: | Absent | 49.4 | 50.6 | **.005** | 1 | |
| | Present | 69.7 | 30.3 | | 2.35 (1.27-4.31) | **.006** |
| Relationship to bystander: | | | | | | |
| | Relative | 59.0 | 41.0 | **.061** | 1 | |
| | Other | 75.6 | 24.4 | | 2.36 (1.02 – 5.44) | **.044** |

*Adjusted for age and gender.

Risk factors such as previous history of MI, hypertension, diabetes, and smoking were not significantly associated with decision delays of less than 60 minutes, and neither were presence of premonitory symptoms or level of emotional support available (see Table 3.15). Type of ACS was an important factor, however, and shows a similar pattern of results to that for shorter than average pre-hospital delay above. Patients who had an STEMI were more likely to make the decision to seek medical help within 60 minutes after the symptoms started compared with those who had UA or NSTEMI. Logistic regression analysis indicated that the OR, adjusted for age and gender, for a decision to seek medical help within 60 minutes of symptom onset for patients who had an STEMI compared with those who had a NSTEMI or UA was 2.26 (p=.006) (see Table 3.15). Patients who attributed their symptoms to a heart attack had shorter decision delays, OR 3.24 (p=0.001) compared with those who did not recognise their symptoms.

Furthermore, the number of symptoms other than chest pain experienced by patients at onset was associated with a short decision time of less than 60 minutes ($p = 0.017$) but the association was non-linear and when analysed using logistic regression did not show a significant relationship between groups. Patients with three or more different non-pain symptoms such a nausea or shortness of breath were also more likely to have a decision time of less than 60 minutes ($p = 0.018$) although again this association was non-linear. When compared with patients presenting with no other non-pain symptoms using logistic regression the association was close to significance (0R 2.04, $p = 0.065$).

## Table 3.15: Patient decision time and clinical and psychological factors

| | | Decision time<60mins (%) | Decision time>60mins (%) | p-value ($\chi^2$) | Adjusted odds ratio (95% C.I.) of delay of less than 60 mins | Adjusted p-value* |
|---|---|---|---|---|---|---|
| **Risk factors** | | | | | | |
| Previous MI: | No | 59.2 | 40.8 | .104 | | |
| | Yes | 76.0 | 24.0 | | | |
| Hypertension: | No | 62.5 | 37.5 | .577 | | |
| | Yes | 58.9 | 58.9 | | | |
| Hypercholesterolemia: | No | 58.1 | 41.9 | .176 | | |
| | Yes | 67.0 | 33.0 | | | |
| Diabetes: | No | 60.8 | 39.2 | .981 | | |
| | Yes | 60.6 | 39.4 | | | |
| Smoker: | Non- smoker | 58.5 | 41.5 | .405 | | |
| | Smoker | 63.9 | 36.1 | | | |
| Alcohol intake | Non-drinker | 64.7 | 35.3 | .329 | | |
| | Drinker | 58.2 | 41.8 | | | |
| Physical exercise: | Inactive | 58.4 | 41.6 | .257 | | |
| | Exercise <2x per wk | 72.1 | 27.9 | | | |
| | Exercise >2x per wk | 58.1 | 41.2 | | | |
| **Clinical presentation** | | | | | | |
| Premonitory symptoms: | No | 61.0 | 39.0 | .964 | | |
| | Yes | 60.7 | 39.3 | | | |
| Type of ACS: | UA/NSTEMI | 47.0 | 53.0 | **.006** | 1 | |
| | STEMI | 66.5 | 33.5 | | 2.26 (1.26-4.06) | **.006** |
| Intensity of pain: | <6 | 60.0 | 40.0 | .796 | | |
| | 6 - 8 | 60.0 | 40.0 | | | |
| | >8 | 65.5 | 34.5 | | | |
| **Symptoms at onset** | | | | | | |
| Number of non-chest pain symptoms: | None | 62.9 | 37.1 | **.017** | 1 | |
| | 1-3 | 53.2 | 46.8 | | 0.65 (.30-1.41) | .27 |
| | 4-8 | 74.2 | 25.8 | | 1.63 (.67-3.98) | .28 |
| Number of non-pain symptoms: | None | 57.4 | 42.6 | **.018** | 1 | |
| | 1-2 | 53.3 | 46.7 | | 0.82 (.41-1.66) | .585 |
| | 3-6 | 74.0 | 26.0 | | 2.04 (.93-4.49) | .065 |
| **Psychological factors** | | | | | | |
| Emotional support: | None | 48.9 | 51.1 | .165 | | |
| | One | 68.3 | 31.7 | | | |
| | 2-3 | 65.6 | 34.4 | | | |
| | 4 + | 56.9 | 43.1 | | | |
| Attribution to heart attack: | | | | | | |
| . | No | 55.2 | 45.8 | **.001** | 1 | **.001** |
| | Yes | 79.3 | 20.7 | | 3.24 (1.60-6.56) | |
| Cardiac denial of impact: | | | | | | |
| | Lowest tertile | 68.8 | 31.3 | .087 | 1 | |
| | Middle tertile | 49.1 | 50.9 | | 0.44 (0.20 – 0.93) | **.032** |
| | Higher tertiles | 55.9 | 44.1 | | .057 (0.27 – 1.21) | .146 |
| Cardiac denial of impact: | | | | | | |
| . | Lowest tertile | 68.8 | 31.3 | **.037** | 1 | |
| | Higher tertiles | 52.7 | 47.3 | | .502 (.261-.965) | **.039** |

*Adjusted for age & gender.**Pain intensity scored on visual scale from 1 to 10 (mild to worst pain ever).

Patients who contacted their GP/NHS Direct initially were less likely to have a short decision time of less than 60 minutes than patients who contacted the ambulance service first (OR 0.17, $p < 0.001$), referred themselves to an A&E department (OR 0.27, $p = 0.006$), or called a family member/friend (OR 0.19, $p = 0.001$) (see Table 3.9).

As before, results regarding cardiac denial of impact showed that patients who scored in the higher tertiles (combined) on this scale were less likely to have a short decision time of less than 60 minutes (OR 0.50 ($p = 0.039$) than patients who scored in the lowest tertiles.

In summary, a short decision time of less than 60 minutes was associated with being married, having an STEMI, recognising the symptoms as being an indication of a heart attack, and having a bystander present. Similar results (not shown) were found for analysis of patient decision time of less than or greater than 30 minutes. Patients who contacted the ambulance service to call for help at the first instance were also more likely to have a short decision time. Patients with a larger number of symptoms (other than chest pain), more non-pain symptoms such as nausea, and patients with lower scores on the cardiac denial scale were more likely to have a short decision time of less than 60 minutes. This does not then support the theory discussed in Chapter 2 (section 2.3.1) that acute symptoms are masked by the theory of back ground noise (Ryan & Zerwic, 2003)

### 3.3.8: Factors associated with home to hospital delay of less than 120 minutes

Socio-demographic factors such as gender, ethnicity and education did not show any significant association with home to hospital delays. Significant predictors of home to hospital delay are shown in Table 3.16. Patients with an income of £20 to £40 000 were

more likely to have a home to hospital delay of less than 120 minutes than patients with an income of less than £20 000 (OR 2.30, p = 0.042), however this association did not remain significant once adjusted for age and gender. Younger patients however, who were under the age of 60, were more likely to have a short home to hospital delay compared with participants who were older. Logistic regression indicated that the OR, adjusted for gender, of having a home to hospital delay of less than 120 minutes from the call for help to hospital admission for participants aged less than 50 years was 3.63 (p = 0.014) compared to patients who were aged over 70 years. Similarly, patients aged between 50 and 60 years were also more likely to have a short home to hospital delay compared with participants who were aged over 70 years (OR 2.93, p = 0.020).

Patients who had an ST segment elevation AMI also had a short home to hospital delay, OR 2.74 (p=0.004) compared with patients who had either NSTEMI or unstable angina (see Table 3.16), although attribution of symptoms to those of a heart attack was not significantly associated with home to hospital delay. Patients who experienced more than 3 types of non-pain symptoms as well as chest pain (nausea, shortness of breath, dizziness etc) were more likely to have a short home to hospital delay than patients who had no other symptoms except chest pain (OR 3.04, p = 0.022). Similarly, patients with more than 4 non-chest pain symptoms (shoulder pain, arm pain, numbness, fainting etc) were more likely to have a short home to hospital delay than patients who had no such symptoms except chest pain. Intensity of pain, however, was not significantly associated with short home to hospital delay.

**Table 3.16 Characteristics of patients by length of home to hospital delay**

| | | Home to hospital delay <120 mins (%) | Home to hospital delay >120 mins (%) | p-value ($\chi^2$) | Adjusted odds ratio (95% C.I.) of delay of less than 120mins | Adjusted p-value* |
|---|---|---|---|---|---|---|
| **Demographic factors** | | | | | | |
| Age **: | >70 years | 67.3 | 32.7 | **.053** | 1 | |
| | 60 – 70 | 78.8 | 21.2 | | 1.88 (0.76 - 4.65) | .169 |
| | 50 – 60 | 84.7 | 15.3 | | 2.93 (1.19 - 7.23) | **.020** |
| | <50 years | 87.0 | 13.0 | | 3.63 (1.30 - 10.17) | **.014** |
| Income per year: | <£20k | 72.9 | 27.1 | .061 | 1 | |
| | £20–£40k | 86.1 | 13.9 | | 1.93 (0.83 – 4.48) | .126 |
| | £40k | 85.4 | 14.6 | | 1.69 (0.63 – 4.55) | .299 |
| **Risk factors** | | | | | | |
| Physical exercise: | Inactive | 79.9 | 20.1 | **.042** | 1 | |
| | Exercise up to 2x week | 90.7 | 9.3 | | 2.09 (.68-6.41) | .197 |
| | More than 2x week | 67.6 | 32.4 | | .47 (.20-1.09) | .079 |
| **Clinical presentation** | | | | | | |
| Type of ACS: | | | | | | |
| | UA/NSTEMI | 68.2 | 31.8 | **.004** | 1 | |
| | STEMI | 85.1 | 14.9 | | 2.74 (1.37-5.47) | **.004** |
| Pain intensity:*** | <6 | 77.8 | 22.2 | .148 | | |
| | 6-8 | 82.2 | 17.8 | | | |
| | >8 | 91.4 | 8.6 | | | |
| **Symptoms at onset** | | | | | | |
| Number of non-pain symptoms: | None | 70.2 | 29.8 | .097 | 1 | |
| | 1-2 | 80.4 | 19.6 | | 1.93 (0.85-4.36) | .114 |
| | 3-6 | 86.3 | 13.7 | | 3.04 (1.18-7.83) | **.022** |
| Number of non-chest pain symptoms: | None | 68.6 | 31.4 | .141 | 1 | |
| | 1-3 | 81.0 | 19.0 | | 2.34 (0.97-5.63) | .058 |
| | 4-8 | 84.8 | 15.2 | | 3.09 (1.11-8.60) | **.031** |

*Adjusted for age and gender. **Age adjusted for gender only. ***Pain intensity scored on visual scale.

As shown in Table 3.9, patients who contacted their GP/NHS Direct were less likely to have a short home to hospital delay than those who contacted the ambulances service first (OR 0.11, $p < 0.000$), referred themselves to an A&E department (OR 0.28, $p = 0.095$), or called a family member/friend (OR 0.14, $p = 0.007$). Other socio-demographic factors such ethnicity, risk factors such as previous MI, hypertension and diabetes were not associated with short home to hospital delay of less than 120 minutes.

In summary, home to hospital delays from making the call for help to hospital admission of less than 120 minutes were associated younger age, having an STEMI, experiencing 3 or more types of other non-pain symptoms (nausea, breathlessness etc), experiencing more symptoms other than chest pain (shoulder pain, arm pain, fainting etc) and contacting the ambulance service in the first instance for medical assistance.

A summary of significant findings relating to pre-hospital delay and it two component phases, patient decision time and home to hospital delay is shown in table 3.17.

**Table 3.17: Summary of results related to pre-hospital delay**

**Factors significantly associated with:**

| Very short pre-hospital delays | Less than average pre-hospital delays | Short patient decision times | Short home to hospital delay times |
|---|---|---|---|
| Attribution of symptoms to heart attack | Attribution of symptoms to heart attack | Attribution of symptoms to heart attack | |
| | Ambulance first call for help | Ambulance first call for help | Ambulance first call for help |
| | ST segment MI | ST segment MI | ST segment MI |
| | Bystander present | Bystander present | |
| Low cardiac denial | Low cardiac denial | Low cardiac denial | |
| | Married | Married | |
| | More non-chest pain symptoms & more-non pain symptoms | | More non-chest pain symptoms & More non pain symptoms |
| Low deprivation | | | |
| Education (up to O'levels) | | | |
| Large social network | | | |
| | | | (Higher income - unadjusted for age & gender) |
| | Afternoon onset | | |
| | History of MI | | |
| | | | Younger age |

## 3.4: Discussion

The first aim of this study was to investigate the socio-demographic and psychological factors which predict delay in contacting medical help following the onset of symptoms of ACS. The main results of these analyses will be discussed in relation to previous literature and the first hypotheses, which stated that shorter patients' decision time in seeking help would be associated with demographic and psychosocial variables including younger age, male gender, greater social support, higher socio-economic status, time of onset on a week day and within work hours, the presence of a bystander, attribution of symptoms to heart attack and low cardiac denial.

Results reported in this thesis showed that 50% of patients had a total pre-hospital delay of less than 120 minutes, and patient decision delay accounted for 60% of the total pre-hospital delay. This is comparable to most other recent studies (see Table 2.1).

### 3.4.1: Socio-demographic predictors of pre-hospital delay

The demographic characteristics and risk factor profile of patients in this sample are broadly similar to other studies which have investigated pre-hospital delay in cardiac patients. However, the mean age of patients in this study was only 59.0 years. This is lower than that seen in some studies of patients with ACS, such as the GRACE registry which reported a mean age of 66.4 years (Goldberg et al, 1999). The exclusion of patients with significant co-morbidities in this study probably led to the exclusion of elderly rather than younger patients, and women rather than men, who tend to be older when they present and therefore more likely to suffer from other co-morbidities. The mean age is close, however to that reported in other studies investigating pre-hospital delay ranging from 58 to 62 years (Dracup & Moser, 1997; Ottesen et al, 2004; Walsh et al, 2004). Almost 30% of participants were women, which is slightly fewer women

than in studies such as the GRACE registry (37%), possibly for the same reasons as mentioned above. The gender component, however, is comparable to other cardiac studies and those investigating pre-hospital delay which typically have a lower proportion of women, unless selectively recruited (Dracup & Moser, 1997; Leslie et al, 2000; Ottesen et al, 2004).

Results from this study did not support the hypothesis that younger patients would have a shorter decision delay as there was no association between delay and age or gender for either total pre-hospital delay or decision delay. Younger patients were, however, significantly more likely to have a shorter home to hospital delay. This finding is supported by a study by Dracup and Moser (1997) and by evidence from the GRACE registry which also reported that older patients took longer to access medical care than younger patients (Goldberg et al, 2002). The reasons for this are not clear, and may be that other factors were involved that were not measured in this study. For example, it may take longer for medical personnel to assess older patients because they attribute their symptoms to other co-morbidities, or because older patients present with a more complex range of symptoms. Patients who contact their GP for help are likely to speak to unqualified staff prior to communicating with the GP. This is likely to lead to delays in medical assessment, waiting for the GP to call back or for an appointment. Reception staff may respond more urgently to cardiac symptoms in younger patients who may be more pro-active. Younger patients who transported themselves to an A&E department may have been more physically mobile. They are less likely to be socially isolated and less likely to have been living alone, and may therefore have received more help from friends, family or work colleagues, in terms of access to transport, care of dependents etc. It would be useful in future research into pre-hospital delay to examine this phase

of delay in more detail in order to determine the barriers to treatment following the call for medical help.

Findings from this study support the hypothesis that patients with higher socioeconomic status would have shorter pre-hospital delays. Results showed that patients with a higher yearly income of £20-£40 000 were more likely to have a home to hospital delay of less than 120 minutes than patients with a lower income, although this did not remain significant once adjusted for age and gender, possibly because women in the study were likely to be older and on lower incomes. Other studies support this finding (Sheifer et al, 2000). Patients with higher incomes may have had better access to transport, more flexibility or control over work commitments, or better communication skills. They are also likely to be younger and less likely to be pensioners. In support of the hypothesis, patients with more years of education (up to O level) and who were not from a deprived background were also more likely to have very short pre-hospital delays (less than 60 minutes), although these associations were no longer significant for the short pre-hospital delay period (120 minutes). This was not due to a short decision delay but was associated with short home to hospital delays.

It is interesting that the socio-economic variables that were associated with very short total pre-hospital delay in this study (higher education and lower deprivation) were not associated with the time taken by patients to decide to call for medical help. The delay therefore was not in recognising that their symptoms were serious and contacting help, but rather in accessing medical help. Some other studies have reported longer pre-hospital delays in patients with fewer years of education and lower incomes (Dracup et al, 1997; McKinley et al, 2000; Schmidt & Borsch, 1990) although they do not identify at which phase of pre-hospital delay (decision time, transport time or home to hospital

delay time) the association was found. The cognitive factors associated with attribution of symptoms to heart attack and type of ACS were similar in patients who had a short decision time (within 60 minutes) as well as those whose total pre-hospital delay of less than120 minutes. This suggests, therefore, that there are factors linked to higher socio-economic position which result in shorter pre-hospital delays, which are not involved in cognitive aspect of making the decision to seek help. Rather, they are associated with the sequence of events that occur between seeking help and reaching hospital.

### 3.4.2: Previous history of MI

Only 11.5% of patients in this study had suffered a previous MI. Other studies have reported a slightly higher incidence of previous MI among participants, from 14% to 26% (Dracup & Moser, 1997; Goldberg et al, 1999; Leslie et al, 2000; O'Carroll et al, 2001; Ottesen et al, 2004; Walsh et al, 2004). Patients who had a previous history of MI were more likely to have a pre-hospital delay of less than 120 minutes in this study and, indeed, it does seem logical that these patients would recognise the symptoms more quickly from previous experience, have a better knowledge of symptoms and a greater sense of personal vulnerability to heart disease and respond promptly. Evidence from previous studies, however, is conflicting and shows that patients with a previous history of MI may take as long or longer than those having their first one (Dracup & Moser, 1997; Leslie et al, 2000; Pattenden et al, 2002; Walsh et al, 2004). The reasons for this are not clear. Although the presence of other risk factors, such as diabetes, hypertension, and hypercholesterolemia, was quite high in this study population in comparison to the general population (British Heart Foundation, 2005a), they were not associated with pre-hospital delay times. Diabetes was present in 13.5% of patients, nearly half of patients (46.9%) had prior history of hypertension and 47.1% suffered from hypercholesterolemia. This is comparable to other studies (Dracup & Moser,

1997; Goldberg et al, 1999). The presence of diabetes and hypertension has been shown to lead to longer pre-hospital delays in some studies (Goldberg et al, 2000b; McKinley et al, 2000). Previous experience of MI was therefore associated with short pre-hospital delay, while having clinical risk factors for heart disease was not.

### 3.4.3: Time and day of symptom onset

Patients were more likely to have less than average pre-hospital delay (120 minutes) if symptoms started in the afternoon. This finding is supported by results from the GRACE registry which also reported a trend for early care seeking for patients with onset of symptoms in the afternoon (Goldberg et al, 2002). Two studies, Pattenden et al (2002) and Sheifer et al (2000), found that patients were reluctant to medical seek help during the night and at weekends, preferring to wait until the following day before seeking medical help. Patients may feel anxious about contacting help 'out of hours', particularly if they choose to contact their GP initially or worry about 'causing a fuss'. Patients may feel less inhibited to seek advice during the day. Friends, relatives and co-workers may be more accessible to provide help during the afternoon. It is also possible that patients were more likely to be in a social situation during this time of day, so that a bystander was more likely to be present or to become involved, whether a work colleague, family member, friend or even a stranger. This support the hypothesis that patients are more likely to have a shorter delay if they experience symptom onset within work hours and have greater social support.

### 3.4.4: Attribution of symptoms

Patients who attributed their symptoms to those of a heart attack had both shorter decision time and shorter pre-hospital delay, thus supporting the hypothesis. Previous studies have also reported this (Dracup & Moser, 1997; O'Carroll et al, 2001). The

most common attribution of symptoms however, was to indigestion (34.2%), whilst only 25.4% of patients recognised that their symptoms were cardiac. Similar findings in other studies have reported attribution of symptoms to a cardiac cause ranging between 19% to 56% (Leslie et al, 2000; Dracup & Moser, 1997; Ottesen et al, 2004). Patients may delay in seeking help, therefore, because they misattribute their pain to some other cause such as indigestion (Wielgosz & Nolan, 1991). Pattenden et al (2002) also reported that some patients do not want to believe that they are having a heart attack, tending to play down or ignore symptoms for as long as possible. Kenyon et al (1991) suggested that patients who delay in seeking help may be less attuned to physiological and emotional reactivity to cardiac symptoms. Alternatively, patients who are very emotionally aware might be distracted by the diversity or intensity of their symptoms, also resulting in prolonged delay. High emotional arousal has been correlated with increased delay (Wielgosz & Nolan, 1991). The ACCENT study also reported an association between high emotional states involving acute anger and depression in the 2 hours before symptoms started and symptom onset (Strike et al, submitted, a; Strike et al, submitted, b). No association was found, however, between pre-hospital delay and self report measures of life stress prior to symptom onset in this study.

### 3.4.5: Severity of ACS and range of symptoms

Patients who had an STEMI were also more likely to make the decision to seek medical help and to have shorter home to hospital delays than those who had a NSTEMI or unstable angina. This is supported by evidence from the GRACE study (Steg et al, 2002). Although in this study, there was no association between age and decision time, and between age and total pre-hospital delay, home to hospital delays were more likely to be shorter than 120 minutes in younger patients. Younger patients were also reported to have shorter pre-hospital delays in the GRACE study, although the analysis of pre-

hospital delay was not divided in to stages it is not therefore possible to identify at which specific time point the difference occurred (Goldberg et al, 2002). Although pain intensity was not significantly associated with delay, it's possible that patients experienced a slightly different pattern of symptoms depending on the type of ACS. Symptoms of STEMI may perhaps follow a more classic and easily recognisable pattern. It may be easier for paramedics (and GPs) to quickly assess patients with more classic symptoms of STEMI, and this may help to explain why patients with STEMI had a shorter home to hospital delay than those with NSTEMI or unstable angina.

Patients who suffered a greater number of other symptoms (whether these were pain in other parts of the body such as the jaw or back, or symptoms such as nausea, sweating etc) as well as chest pain were more likely to have a shorter than average pre-hospital delay and a short home to hospital delay. This is supported by Ruston et al (1998) who found that non-delayers were aware of a wider range of symptoms than delayers. It is also possible, however, that this was influenced by an element of recall bias in that non-delayers were more aware of their symptoms and had better recall. Greater number of symptoms was not associated with patient decision delay, however, neither was there any association with pain intensity. The association between delay and pain intensity is not straight forward. Some previous studies have shown that patients with mild to moderate pain had significantly longer delays than patients with strong pain (GISSI, 1995) whilst others, as results presented here illustrate, show no association between pain intensity and pre-hospital delay (O'Carroll et al, 2001; Ottesen et al, 2004).

In contrast to the findings reported in this thesis, Horne et al (2000) reported that the number of symptoms experienced was not related to pre-hospital delay, rather delay was influenced by patients' interpretation of symptoms. Horne et al found that patients who

experienced atypical symptoms that that they did not expect or associate with a heart attack, had longer pre-hospital delay because they did not recognise the seriousness of their condition. They found that most patients (93%) experienced at least one atypical symptom during their cardiac event. However, shorter pre-hospital delays were associated with the presence of a greater number of typical symptoms, which were commonly perceived indicate a heart attack. This may indicate that patients, and/or medical services, were able to recognise typical symptoms more quickly, however patient decision time was not analysed separately to total pre-hospital delay and this is not possible to discern. This difference in findings may be explained by the inclusion criteria used for the study reported in this thesis; only patients presenting with chest pain were recruited, so if patients had atypical symptoms, these were experienced as well as chest pain. In the study by Horne et al, 36% of patients did not have chest pain and may have experienced only atypical symptoms.

### 3.4.6: Presence of a bystander

Finding in this study showed that patients who were accompanied by a bystander at the onset of their symptoms were more likely to have a pre-hospital delay of less than the average time (120 minutes) and a short decision time, in support of the hypothesis. Similar findings have been reported in other studies (GISSI, 1995; Perry et al, 2001). Patients often consult someone else to help them decide what to do and bystanders may play an important role in the decision making process and in helping patients travel to hospital, either by calling the ambulance or providing transport. One study reported that 93.2% of patients told someone that they were ill before travelling to hospital (Alonzo, 1986). Results in this study show that in patients with a shorter than average pre-hospital delay, it was more likely to be the bystander who made the decision to call for help (Table 3.12) and a short decision time of less than 60 minutes was more likely if

the bystander was not a relative (Table 3.14) This has also been reported in other studies (Alonzo, 1986). Dracup et al (1995) suggest that family members are more motivated to share in the denial of the patient and are less willing to confront the patients' delay while a co-worker/friend/stranger may take more pro-active approach, less willing to take responsibility for a wrong decision by taking the chance of a 'wait and see' approach and less willing to share the patients' 'wishful thinking'. The bystander may also be more able to make an objective appraisal of the symptoms and may have a better knowledge of cardiac symptoms. Having the seriousness of symptoms confirmed by a bystander may also help the patient to feel more confident in the necessity of contacting medical services. The bystander may be able to offer practical help such as phoning the GP/ambulance or providing transport to the hospital that reduce delays. Patients with a larger social network, and patients who were married were more likely to have a short decision time and pre-hospital delays possibly because this increases the likelihood of having a bystander present at onset. These patients may also have a wider range of people to consult. If the symptoms start in the afternoon, people are more likely to be at work or in some other social situation where a bystander might be present.

### 3.4.7: Type of help sought

Results of this thesis showed that the type of assistance sought by patients following the onset of their symptoms was an important factor associated with pre-hospital delay. Patients who contacted the ambulance services or referred themselves to an A&E department had both shorter decision times and home to hospital delays. Evidence from other studies support these findings (McGinn et al, 2005; Schmidt & Borsch, 1990). Studies have shown that patients are reluctant to call the ambulance services in the first instance following the onset of acute cardiac symptoms (Leslie et al, 2000; Meischke et

al, 2000; Pattenden et al, 2002). In a study by McGinn et al (2005) only 38.8% of patients used EMS.

As discussed earlier, the socio-economic variables that were associated with longer total pre-hospital delay in this study, including lower education and higher deprivation, were not associated with the time taken by patients to decide to call for medical help. The delay therefore was not in recognising that their symptoms were serious and contacting help, but rather in accessing medical help. People who are more deprived and have a lower level of education are more likely to be socially isolated. Having made the decision to seek medical help, they may face greater barriers in actually getting to hospital. They may find it more difficult to contact medical services, such as NHS Direct or their GP, because they do not have easy access to a telephone, because GP services in their area are under greater pressure, less efficient or have a more negative approach to patients. The gate keepers to such services such as reception staff may take a more negative approach to these patients, not take them seriously or be slower to take action. Emergency services and GP's may be reluctant to attend certain patients or patients living in certain areas. These patients may not communicate well with health care professionals, appear less assertive or conversely more aggressive, and this may create negative responses to them. It may also be difficult to arrange child-care or care of dependents at short notice, or to arrange for private transport to attend a GP or A&E department. A study by Heriot et al (1993) also reported that although patients had similar decision times, those who sought help from their GP rather than proceeding directly to hospital had significantly longer response times (home to hospital delay). These factors may have more of an impact on the home to hospital phase of delay than on decision time, and may lead to longer total pre-hospital delays among people with lower socio economic status.

It is not surprising that patients who initially called the ambulance service were more likely to have shorter pre-hospital delays of less than 2 hours. The ambulance service in the UK is obligated to respond quickly, preferably within 8 minutes to emergency calls involving cardiac pain and to transport them directly to hospital within 30 minutes. This study took place in an urban environment and most patients were within a reasonable distance from an ambulance centre or hospital.

A recent study investigated home to hospital delays and compared transport time to hospital between patients who referred themselves directly to A&E using private transport and those who called for an ambulance (Hutchings et al, 2004). Although time to hospital admission was slightly quicker by private transport, time to medical treatment was quicker by ambulance since paramedics were able to assess patients and commence treatment at the scene, prior to or during transportation to hospital. This has become particularly important since the recent introduction of initiation of thrombolysis by paramedics. Patients who self refer may therefore inadvertently delay treatment. Earlier studies have also found that patients who called their GP first for help had a significantly longer delay times than those who called an ambulance or referred themselves directly to an A&E department (Dracup et al, 1997; GISSI, 1995; Heriot et al, 1993).

### 3.4.8: Psychological factors

It is common for patients to experience fear and denial during cardiac emergency (Meischke et al, 2000). Denial is a transitory state and is difficult to measure since data can only be collected retrospectively, hence the measure used in this study measures denial of impact associated with cardiac illness and was collected very soon following

the ACS by self report questionnaire. Findings reported in this thesis supported the hypothesis, showing that patients with lower scores on the cardiac denial of impact scale had shorter patient decision delay and shorter than average total pre-hospital delays. This is supported by evidence from previous studies (Dracup et al, 1995; O'Carroll et al, 2001). Denial has been generally accepted as a beneficial coping strategy in the first few days following MI since it may protect the patient from distressing emotions such as anxiety and depression (Lewin, 1995; Sarantidis et al, 1997), however, inattention to pain or maladaptive coping behaviours may also increase the time required to decide to seek medical help (Wielgosz & Nolan, 1991). It has been suggested that previous history of MI may induce post-traumatic stress disorder so that when symptoms reoccur, some patients may try to suppress or avoid stimuli that remind them of the initial trauma (Alonzo & Reynolds, 1998). This is not supported by evidence from this study since patients with a previous history of MI were more likely to have a shorter rather than longer average pre-hospital delay and there was no association between previous history of MI and decision time.

## 3.5: Limitations of this study

There are a number of limitations to this study which are outlined below.

### 3.5.1: Study population

Although the demographic characteristics of patients in this study were similar to other studies investigating pre-hospital delay, this study consisted of fewer female participants than other cardiac studies in general, and the mean overall age was younger. Female participants tend to be older when they present with symptoms of ACS and often present initially with angina (Lerner & Kannel, 1986). Female patients may therefore have presented with more co-morbid conditions that would have excluded

them from this study. Due to the selection criteria for the larger study (the ACCENT study) a larger proportion of patients with STEMI were recruited compared with NSTEMI/UA than has been described in recent surveys (Rosengren et al, 2004b). This may have influenced the pattern of pre-hospital delay observed.

### 3.5.2: Limitations of inclusion criteria

Only patients whose presenting symptoms included chest pain were recruited for this study due to the need to identify specific time phases, thus patients who were diagnosed with ACS but did not suffer from chest pain or could not identify a clear onset time were not included. The exclusion criteria for this study was quite strict excluding all patients with serious psychiatric illness, on-going critical ischaemia, and other medical conditions which would compromised medium to long term outlook and influenced mood and symptom presentation. This would have probably affected mainly women and older patients. It should also be noted that pre-hospital delay was measured only in patients who survived their symptoms and who came under hospital care, they may therefore have had less serious atherosclerosis and/or less serious cardiac arrhythmias than patients who did not survive.

### 3.5.3: Limitations of the methodology

It is possible that data collected for this study was affected by recall bias. Data was collected retrospectively and patients were interviewed between 1 and 5 days after hospital admission. Some had therefore had time to develop their own theories about the causes of their illness. The self report measures used in this study were also retrospective, thus patients' reports of their pre-hospital experiences may have been affected by their efforts to understand their experience. Accurate measurement of the time intervals constituting pre-hospital delay and decision delay also depended on

patients' recall of the time their symptoms started. Although in most cases patients appeared confident in their ability to recall the time of symptom onset, it is possible that there are some inaccuracies in their recall of the timing of events prior to hospital admission.

# Chapter 4: Patients' causal attributions of ACS

## 4.0: Introduction

The beliefs that patients hold about the causes of physical symptoms or illnesses can have a profound effect on their behaviour, from the decision to seek medical help at the onset of their symptoms, to adherence to recommended treatment, and psychological adjustment to the prognosis and lifestyle changes. Understanding lay beliefs about illness may also be important in optimizing clinical management, in allowing researchers and clinicians to predict patients' behaviour more accurately, and in identifying patients who have particular difficulties in adjusting to illness and who may benefit from appropriate interventions. Previous studies have examined causal attributions in relation to gender differences (Astin & Jones, 2004; Baumann et al, 1989; Murphy et al, 2005), behaviour changes (De Valle & Norman, 1992; Weinman et al, 2000) and coping post discharge (Roesch & Weiner, 2001). There is very little previous work which has investigated the precise association between patients' beliefs about the cause of their ACS and pre-hospital delay.

This literature review will firstly discuss some of the psychological theories and concepts which have formed the background from which research into causal attributions has developed; secondly, it will discuss the main findings of studies which have investigated causal attributions specifically among cardiac patients, differences in methodologies, and how causal attributions may affect pre-hospital delay.

## 4.1 Definition of causal attributions

Attributions have been defined as post hoc interpretations or redefinitions of causes of an illness, which may be used in reconstructing basic assumptions about the world

(Sensky, 1997). Sensky argues that people do not act on the objective evidence of their illness or symptoms but on their own lay illness representations. Attributions provide a framework from which future decisions and behaviours can be made to minimize the probability of a negative outcome reoccurring. They are important because they can predict cognitions and behaviours directed towards becoming well or maintaining health after diagnosis, and motivation to perform preventive health behaviours (Roesch & Weiner, 2001).

## 4.2: The development of theories and concepts explaining causal attributions

Attribution theory was first proposed by Heider (1958) who suggested that when individuals are faced with a sudden threat, change to their environment, negative, unexpected or unusual outcomes they will spontaneously look for a cause in order to try to understand or give meaning to the threat or event. He argued that this search for causal attributions helps individuals to understand, predict and control the threat. This theory was later developed further by Weiner (1979; 1985; 1986) who focussed mainly on attributions related to achievement/failure, but it has since been used widely in other areas such as education, law, clinical psychology and mental health . The search for meaning and causal attributions may be particularly pertinent for patients suffering from a serious illness, such as an acute coronary syndrome.

Much of the work investigating the role of causal attributions and illness since the 1970's has focussed on examining what kind of attributions patients make, adjustment, and assessing the relationship between the causal attributions and subsequent recovery in terms of behaviour change. It has been hypothesised that three factors act as key mediators underlying attributional characteristics and adjustment. These include;

firstly, the preservation of self esteem (Shaver, 1970); secondly, the ability to maintain a perception of justice (Lerner, 1980); and thirdly, the maintenance of a sense of control (Heider, 1958).

Shaver (1970) proposed that individuals' reactions to negative events are influenced by their desire to avoid blame for future negative events. Defensive attributions such as attributing the event to chance or fate allow the individual to protect their self esteem and to avoid taking responsibility. Shaver argued that individuals are more likely to accept causal responsibility for positive rather than negative outcomes. This suggests that the motivation to maintain positive self-esteem makes it more likely that individuals will make attributions to external factors for negative events.

Lerners' 'Just World Theory' (1980) proposed that individuals need to believe that people deserve what happens to them so they react to negative events in such a way as to maintain that belief. This may mean that they will re-evaluate negative events and outcomes as positive, or blame themselves or others for the event in order to provide psychological consistency.

Heider (1958) argued that by attributing cause to factors which are perceived to be under the control of the individual, and therefore more easily modifiable, individuals are more able to adjust to an unexpected event and/or to the threat of it happening again. He proposed that individuals striving to attain a sense of personal control are likely to make attributions related to self (internal), while individuals who experience a lack of personal control are likely to attribute the cause of events to powerful others, luck or chance (external).

## 4.3: Types of causal attribution: dimensions and categories

Attributions have traditionally been assessed in one of two ways: dimensions or categories. Weiner (1985; 1986) proposed that causal attributions could be conceptualised and classified along three broad dimensions which have been widely used in social psychology. It is argued that knowing the dimensional locations of causal attributions rather than the attributional categories allows for more accurate prediction of the consequences of attribution (Roesch & Weiner, 2001). The first dimension is the locus of causality ('locus'). This refers to the location of a cause that reflects either the person (internal), or the environment (external). The second dimension is 'stability', which refers to the changeability of the cause over time (stable – unstable). The third dimension, 'controllability', refers to whether the cause is regarded as being under the control of the individual or not (controllable – uncontrollable). This way of classifying attributions has been used in research examining coping and adjustment following a range of negative events such as serious illness (Faller et al, 1995), death (Downey et al, 1990) and crimes such as rape (Frazier & Schauben, 1994), although there has been some disagreement and variation among researchers as to where specific causes should be located within the dimensions (Krantz & Rude, 1984).

Most studies investigating causal attributions for serious illness have assessed attributions in specific categories rather than along broad dimensions. A number of categories have been identified including the self, others, chance, the environment, heredity, life style factors or personal behaviours (such as diet, smoking, lack of exercise). More embracing constructs such as self blame have also been developed. Self blame refers to the individuals' belief that that s/he is responsible in some way for the negative event and is thus an internal attribution thought to be associated with better adjustment to negative events, since it allows individuals to believe that they have a

greater level of control over the future (Tennen & Affleck, 1990). A further distinction was later made between two subtypes of self blame; characterological and behavioural (Bulman, 1979). Characterological self blame refers to causes that are enduring aspects of individual character, and is viewed as maladaptive since character is considered to be unchangeable. Behavioural self blame refers to causes which are transient in nature and modifiable, and is viewed as adaptive because it allows patients to believe that they have some control over the future (Roesch & Weiner, 2001). Indeed, Affleck et al (1987) found that patients who attributed their MI to self blame in the form of modifiable personal behaviours were less likely to suffer another MI, and also experienced less long-term morbidity due to better adjustment to their illness.

By contrast, blaming others appears to be a specific way of making an attribution about an external cause, and has been associated with poorer adjustment in terms of emotional distress and physical impairment across a wide range of studies (Tennen & Affleck, 1990). Blaming others is thought to influence outcomes in 3 ways; by interfering with adaptive coping strategies such as problem solving and positive reappraisals; by challenging participants' deeply held world views of themselves and others; and by hindering social support.

Studies exploring the association between self blame and adjustment, however, have reported contradictory findings (Michela & Wood, 1986; Turnquist et al, 1988). A review of 65 studies investigating the association between attributions and outcomes by Hall et al (2003) showed that 'self blame' and 'blaming others' were not associated with outcomes in 76% of reported analyses, were associated with poorer outcomes in 21% and were associated with better outcomes in only 3%. Of the 10 most frequently assessed categories of attributions, characterological self-blame, blaming others and

general self blame were most often associated with poor outcomes, and no one category was associated with better outcomes. Behavioural self blame was not associated with better outcomes in this review. Hall et al suggested several explanations for the inconsistency in findings which highlight some of the difficulties in comparing attribution studies; power varied widely between studies; ways in which attributions were defined varied; the types of outcome assessed and the nature of the events differed; some studies included participants of one gender only; the time period from event to data collection was not uniform; and methods used to collect data varied between open ended questions, semi- structured interviews, questionnaires and cued responses.

## 4.4: Methodological differences between previous studies

Causal attributions have been measured in a variety of ways, but most studies have used at least one of the following methods; rating scales, cued attributions and open ended questions via a face to face interview or questionnaire. Rating scales and cued attributions have the advantage of being easier to quantify but limit the participants' choices to pre-selected items, which may also increase demand effects. Open ended questions rely on the participant being able and confident enough to make specific attributions and can be harder to analyse, but allow the subject to generate causes freely without prompting. Face to face interviews are a convenient and efficient way to obtain information but are also more likely to elicit socially desirable responses. A few studies have also attempted to assess participants' spontaneous, unprovoked thoughts about their illness but this is a more problematic method and is rarely used.

Gudmundsdottir et al (2001) compared four different assessment methods commonly used to investigate attributions in patients with heart disease in order to see if there were any differences in the pattern of causal beliefs. Patients with a confirmed MI (N = 100)

were interviewed within 2 weeks of hospital discharge. Data on spontaneous, elicited and cued attributions and ratings of the cued attributions were compared. This study was designed to be longitudinal so that changes in attributions over time (1 year) could also be assessed.

Results showed that only 21% of patients made spontaneous attributions at discharge, and of these, 'smoking', 'it's in the family', 'working' and 'stress' were the most common. These attributions did not change over time, apart from 'exertion/exercise' which was rated as initially important but less so later. Behavioural self blame was found to be the most common attributional category. Elicited attributions were made by 82% of patients at discharge and again there was no significant difference over time. Most common elicited attributions were 'stress' and 'smoking' followed by 'it's in the family' and 'worry'. Behavioural self blame was again the most common category and showed a significant increase over time. The most common cued items from the list of 34 were 'stress' and 'smoking' at all time points. At discharge this was followed by attributions to 'myself', 'worry', 'eating fatty foods' and 'it's in the family'. The order changed only slightly over the 12 month follow up period. Certain items which were consistently thought to cause MI and did not show any significant change over time, such as 'stress', 'smoking', 'eating fatty foods', 'high levels of cholesterol', 'being over weight', 'high blood pressure', 'drinking excessive amounts of alcohol', 'depression' and 'problems with my spouse', while the remaining 25 items were reported less frequently over time. Results reported for cued attributions combined scores for patients who answered 'might have' and 'yes', rather than only those who registered a definite affirmative belief, and therefore incorporate beliefs which vary in strength. Patients attributed the most important causes of their illness to 'stress' and 'smoking', followed by 'it's in the family', 'worry', and 'eating fatty foods'.

The use of different methods of attributional assessment in this study did not appear to affect the types of attributions that were made and these remained constant over 12 months. The most common attributions in all categories were 'stress', 'smoking', 'its in the family', 'eating fatty foods', and 'work items'. Patients made more attributions when the cued method was used, and, although quality was not affected, fewer attributions were made using open ended questions, possibly because the cued method alerted or reminded patients of possible causes other than those that came immediately to their mind. Most patients did not make a spontaneous attribution but they did make an elicited attribution at each time period, indicating that they had engaged in some kind of causal search. Most patients attributed their illness to 'behavioural self blame', and this seemed to reflect largely behavioural attributions. This may indicate that patients perceived that they had some control over their illness, and supports attribution theory in that people prefer to make attributions to controllable factors.

A recent review by French et al (2001) also investigated whether methodology affected the pattern of attributions made. Forty-seven studies of causal attributions were reviewed and methodologies included open ended questions, rating scales and focus groups. Sample populations varied, including patients diagnosed with heart disease and non-patients. Findings showed that chronic stress and lifestyle factors were the most common causal attributions for heart disease, and were also rated as the most important in over two thirds of studies. There was no evidence that different patterns of attribution were produced depending on whether the responder or experimenter generated the attributions, although there was evidence to show a different pattern of beliefs depending on whether respondents rated attributions dichotomously or on an interval scale. For example, attributions to stressors and fate or luck (both $p < 0.05$) were more likely to be reported where rating scales were used than in studies that used

dichotomous answers (applies/does not apply). There were no significant differences when rankings were compared dichotomously or on an interval scale.

French et al (2001) noted that in attribution studies, patient samples and non-patient samples were often asked slightly different questions, i.e. patients were asked what caused their own disease while non-patients were asked what causes heart disease in general. This is an important issue because patients may therefore have reported their own experiences and given greater weight to constructs that helped them make sense of their experiences, while non-patients answered a general question about what causes heart disease and may have focussed more on accepted medical concepts. Patients may also have interpreted this as a question about what triggered their acute event while non-patients may have interpreted the questions as being about underlying atherosclerosis.

## 4.5: Causal attributions and ACS

This review will include a number of studies which have investigated causal attributions in patients with ACS since the 1970's, summarized in Table 4.1.

**Table 4.1: Studies investigating causal attributions of heart disease in cardiac patients.**

| Authors | Subjects | Acute/non-acute phase | Method of assessment | Most common attributions |
|---|---|---|---|---|
| Affleck,G.et al (1987) | 287 MI patients | Non-acute<br><br>Interviewed at 7-8 weeks & 8 years | Interviews & questionnaires | Stress, personal behaviour (smoking, overweight, poor diet, excess alcohol, working too hard), heredity, bad luck, other people. |
| Astin,F. & Jones,K. (2004) | 140 CHD patients awaiting PTCA | Non-acute<br><br>Post discharge - time not specified. | Interviews. Open-ended questions. Quantitative | Reported by gender:<br>Women: stress, family history, cholesterol, smoking.<br>Men: poor diet, smoking, stress, family history. |
| Cameron,L.D. et al (2005) | 65 first MI patients | Acute phase – within 2 days of admission | Questionnaires | Stress, poor diet, smoking, exercise, heredity, obesity, hypertension, overwork. |
| Cowie, B. (1976) | 27 first time MI patients | Non-acute: 3 weeks post hospital admission | Interview – Open ended questions | Strain, tension, overwork. |
| Day,R. et al (2005) | 69 patients diagnosed with CHD referred for exercise stress testing. | Non-acute – not specified. | Questionnaires and 23 item checklist re:causes of MI | Genes, hypertension, cholesterol, lack of exercise, diet, stress,obesity, smoking, aging, sadness, nervous tension. |
| De Valle & Norman (1992) | 81 pre-operative patients CABG's (men only) | Non-acute. At home awaiting surgery | Questionnaires Cued list of 21 causes. | Stress, smoking, heredity, eating fatty foods.<br>Ranking: stress, work, eating fatty foods, cholesterol, smoking, heredity. |
| Fielding, R. (1987) | 148 first time MI patients. | Not specified. | Open-ended questions, plus rating of causes. | Over work, smoking, worry |
| French,D,P. (2005) | 12 first time MI patients | Acute-interviewed within 1 week of admission | Structured interview Interpretative phenomenology | Stress, heredity, smoking, diet, exercise. |
| Gudmundsdottir, H. et al (2001) | 100 MI patients | Non-acute – 2 weeks post – discharge and followed up at 2,6 &12months. | Interview: Spontaneous Open ended Cued list | Stress, smoking, myself, eating fatty food, heredity. |

| Authors | Subjects | Acute/non-acute phase | Method of assessment | Most common attributions |
|---|---|---|---|---|
| King, R. (2002) | 24 MI patients | Acute- a few days after hospital admission. | Semi-structured interview & questionnaire. Open-ended questions – phenomeno-logical design. | Stress, exercise, diet. |
| Martin,R. et al (2005) | 157 MI patients | Non-acute. Post hospital discharge. Follow up at 3 months. | Tape recorded narratives responses to 3 open ended questions | Stress, comorbid conditions, diet, smoking, heredity, lack of exercise, prior cardiac history. |
| Meyer (1983) | 30 MI patients | Not specified | Interview: open ended questions Qualitative design. | By age group: Younger patients: family history, genetics, pre-destiny. Middle-aged: patients: life, stress, work, family problems, personal overload. Elderly patients: age. |
| Murphy,B. et al (2005) | 260 AMI patients or awaiting CABG's. (female only) | Acute-Interview 4-8 s after admission. Follow up at 2, 6 and 12 months | Open-ended question to assess causal attribution & questionnaire assessment of risk factors. | Family history, smoking, stress, no idea, diabetes, obesity, cholesterol, high fat diet, hypertension. |
| Rudy (1980) | 50 patients and spouses post MI. | Non-acute 48 hours after hospital discharge and 1 month later. | Open-ended questions, and questionnaire (list & rate importance). | Tension of life. (Spouses cited overwork more often) |
| Van Tiel, D. et al (1998) | 28 patients with symptoms indicating ACS | Non-acute, not specified. | Semi-structured interview. | Not reported. |
| Weinman,J.et al (2000) | 143 first time MI patients (& 84 spouses) | Acute –during hospital stay. Follow up at 6 months & spouses at 12 weeks | Cued list - questionnaires Quantitative | Stress, high cholesterol, eating fatty food, lack of exercise, heredity, smoking, work, being overweight, over work. |
| Zerwic,J.J. et al (1997) | 105 AMI patients or newly diagnosed CAD. | Non-acute – during hospital admission. | Interview Open ended questions Quantitative | Diet, smoking. |

| Authors | Subjects | Acute/non-acute phase | Method of assessment | Most common attributions |
|---|---|---|---|---|
| **Reviews** | | | | |
| French,D,P. et al (2001) | Review of 47 studies | Varied | Open ended, rating scales, focus groups | Chronic stress, lifestyle factors. |
| Hall,S; et al (2003) | Review of 65 studies | Varied | Not specified. | |

As well as differences in methodology between studies, the time interval between the cardiac event and data collection has also varied from the acute phase (within 5 days of the event) to the post hospital discharge period (up to two months). Despite this, most studies have found a similar pattern of attributions. As shown in Table 4.1, patients most commonly believed that their heart problems were caused by stress, personal lifestyle behaviours and heredity factors despite the use of different methods of data collection (interview, questionnaire, taped narrative) and design (open ended questions, cued or spontaneous responses).

It is likely that individuals develop causal explanations which allow them to assess their own risk of heart disease both before and after the event and to develop retrospective explanations of their own illness using knowledge and lore they have received from the wider society in which they live rather than by inventing completely fresh explanations. In a society where the media of mass communication carries such an enormous volume of up-to-date, processed, professional / scientific information, and the availability of personal reports of illness from friends, family, colleagues and celebrities, there are a wide number of sources upon which patients may base their own personalised

modifications of their health beliefs (Davison et al, 1991). These beliefs may therefore be largely socially constructed. It has also become clear that patients' causal attributions often differ from aetiological beliefs held by health care professionals. There was wide disagreement in several studies reviewed here between patients' beliefs about the causes of their heart problems and their personal risk profiles as assessed by health care professionals. Patients' perceptions of their personal health risks and general beliefs about the causes of heart disease may influence their causal attributions, but these attributions may be inaccurate if their perceptions are incorrect. Effective communication may be impeded if patients have different models of cause from clinicians. This may result in patients making inaccurate attributions or neglecting to make attributions to particular risk factors that affect them personally, and thus affect their response firstly to their symptoms at onset, and secondly to secondary prevention and making lifestyle changes.

One of the early studies to explore causal attributions in patients who had suffered an MI was by Cowie (1976) who interviewed 27 first time MI patients 3 weeks following hospital admission and a few days prior to discharge. Patients were interviewed using open ended questions such as "Why are you in hospital?" in order to investigate how patients used causal explanations about their MI to understand their illness. Findings showed that most patients did not regard their MI as a sudden, unanticipated event but rather as the result of particular, pre-existing conditions which had causal antecedents including strain, tension and overwork.

A qualitative study investigating the experiences of 30 male MI patients later found that there were differences in causal beliefs depending on age (Meyer, 1983). When interviewed, younger patients were more likely to attribute their illness to family

history, genetics and being 'pre-destined to illness', middle aged patients made attributions to life, stress, work, family problems and personal overload, while elderly patients felt that their age was the main cause of their MI.

Fielding (1987) then investigated perceived causal attributions, perceived causal potency and perceived controllability of causal attributions in 148 first time male MI patients. Patients were asked to list factors they felt had caused their MI in order of perceived pathogenicity and then rate them on a scale from 1 to 10 (least to most important). Finally, each factor was rated on a 4 point scale where 0 was 'totally uncontrollable' and 4 was 'totally controllable'. Patients cited 321 causes, which were reduced to 33 different causal categories by the researchers. 'Overwork' was the most frequently cited causal factor, followed by smoking and worry. Hypertension was rated as having the highest causal potency, but over 76% of the total potency ratings were given to 5 categories; smoking, overwork, worry, lack of exercise, and stress. Behavioural factors such as smoking, lack of exercise, lifestyle, overweight and diet were rated as significantly more controllable than overwork, worry, stress, other illness, atheroma/cholesterol, frustration/anger and family history. Family history, situational factors, age and hypertension were perceived as totally uncontrollable. Overwork, worry and stress were all rated amongst the most important causes and were also considered to be less controllable than other factors. These results indicate that patients perceived the causes of their MI as being largely psychosocial, which conflicts with the strongly biological medical understanding of causes of heart disease.

An 8 year longitudinal study by Affleck et al (1987) examined the relationship between causal attributions, perceived benefits and health outcomes in a sample of 287 men following their first MI. Causal attributions were assessed 7 weeks and 8 years post MI,

using a list of 13 causes of MI which patients rated on a 3 point scale. Patients were also asked an open ended question about what, if any, gains or benefits they saw from having an MI. Findings showed that patients made greater attributions to stress and personal behaviours, and fewer attributions to luck and other people at both baseline and 8 years. Patients who perceived benefits from a first MI were less likely to have a subsequent MI and suffered lower morbidity after 8 years. Patients' attributions 7 weeks after having had an MI predicted health outcomes. Blaming others was related to a higher incidence of re-infarction and making attributions to stress also was also predictive of greater morbidity. Patients who were interviewed at the 8 year follow up and had survived a second MI were more likely to report benefits and made a greater number of attributions than those who had not suffered another MI.

Affleck et al (1987) suggested that patients who believed stress to be a cause of their MI might perceive stress as less controllable, leading to a sense of helplessness in making adaptive life changes which might then become less likely. Equally, it is possible that these patients were exposed to more stressors or experienced more adverse reactions and that this accounted for their deteriorating health. A recent study by Rosengren et al (2004a) showed that patients suffering from ACS had been exposed to greater levels of stress. Stress is quite a complicated construct which is not easy to measure and hard to dispute. It can be treated as either an external, uncontrollable cause which may help to reduce feelings of self blame (Rudy, 1980) or alternatively it can be regarded as an internal controllable factor, which allows patients to modify their behaviour.

The studies above, however, pre-date the widespread use of thrombolytic therapy when patients were treated more passively following an MI. Survival rates have improved dramatically following the advent of thrombolytic therapy, and secondary prevention now receives a much greater emphasis in public health education campaigns which tend

to focus heavily on personal modifiable risk behaviours such as smoking, diet and exercise. This may have helped to increase general knowledge about heart disease and its risk factors, and shifted the emphasis to behavioural attributions.

### 4.5.2: Causal attributions and lifestyle change

A study by De Valle & Norman (1992) examined the relationships between causal attributions, health locus of control and reported lifestyle changes in 81 men at home awaiting coronary artery bypass graft surgery. Patients were sent a questionnaire which was divided into 3 sections. They were first given a list of 21 possible causes of coronary heart disease and, using a 3 point scale (1 = no, 2 = might have, 3 = yes) they were asked to indicate which ones they thought caused their illness. They could also add causes they considered relevant that were not on the list. Items were classified to create a scale of behavioural self blame consisting of 8 causes: smoking, drinking excessive amounts of alcohol, lack of exercise, being overweight, poor diet, eating fatty food, overwork, and over exertion or sudden exercise. Patients were then asked what they thought was the main cause of their illness. Next, patients were asked an open ended question about whether they had changed their lifestyle since their diagnosis and if so, how. Lastly, patients were asked to complete a multidimensional 'health locus of control scale' developed by Wallston et al (1978; 1991) which measured the extent to which patients believed that their health was influenced by 'internal factors', 'powerful others' and 'chance'.

The most common causal attributions were to stress, work, eating fatty foods, high levels of cholesterol, smoking, and hereditary factors. Patients thought the main cause of their MI was stress or worry, followed by smoking, heredity, and eating fatty foods. They believed their health was influenced by both internal factors and powerful others,

but were less likely to believe that chance was a causal factor. There was a positive relationship between internal health locus of control beliefs and behavioural self blame, i.e. patients who believed their health was under their own control were more likely to attribute the MI to behavioural self blame. Life style changes were reported by 83% of patients, most often related to diet, stress management and smoking. Behavioural self blame was strongly associated with the number of lifestyle changes. Patients' general health locus of control beliefs however, were unrelated to reported behavioural changes. This study suggests that patients' attributions may be important in creating healthy lifestyle changes, particularly when they include modifiable behavioural factors.

Weinman et al (2000) also reported that MI patients' causal beliefs measured by questionnaire soon after hospital admission were associated with behaviour changes 6 months after discharge. Patients were asked to rate whether they agreed or disagreed that each of 24 items was a cause of their MI on a 5 point Likert scale (from strongly disagree to strongly agree). Dietary changes were associated with patients' belief that fatty foods, high cholesterol, poor diet, and lack of exercise were causes of their MI. Increased levels of strenuous exercise were associated with stronger beliefs that lack of exercise, high cholesterol and being overweight were causes of their MI. Reduction in alcohol consumption was also associated with a stronger belief that drinking too much alcohol was a causal factor. It seems plausible that patients' causal attributions affect behaviour change during their recovery.

### 4.5.3: Causal attributions and objective risk factors

Zerwic et al (1997) investigated patients' perceptions of the causes of their coronary artery disease (CAD) in a study of 105 newly diagnosed patients. Patients had either been admitted with MI but had no previous history of CAD (N = 65) or were waiting for

coronary angioplasty because their symptoms suggested CAD (N = 40). Patients with MI were interviewed in hospital during the acute phase of their illness if they had been pain free for more than 24 hours, had been transferred to the cardiac step-down unit, or were in the non-acute phase following hospital admission and awaiting angiography. Open-ended questions were used to ascertain their beliefs about the causes of CAD. Findings showed that the most frequently cited causes for both groups were diet (including attribution to high cholesterol levels) and smoking. Zerwic et al found quite a high degree of discordance, however, between patients' attributions and their personal risk profile. Although most patients who were smokers (64%) recognised smoking as a personal cause of their heart problem, almost one third did not, some of whom did not mention smoking at all or discounted it as a personal cause. Only 15% of hypertensive patients recognised hypertension as a causal factor. None of the MI patients who were diabetic identified this as a causal factor, and only 21% of diabetic patients awaiting angiography attributed diabetes as a causal factor.

In another study of 140 patients with a diagnosis of coronary heart disease (CHD) attending a clinic prior to elective percutaneous transluminal coronary angioplasty, Astin & Jones (2004) compared patients' perceived causal attributions for CHD with their own coronary risk factor profile as documented by their attending physician. Patients were interviewed at home after the acute phase of their illness and asked open ended questions about their understanding of their heart disease. All patients had been diagnosed with CHD following angiography and 58 patients had previously suffered an MI. Findings showed a significant gender difference in the most commonly cited cause of their CHD. Women (n = 32) commonly cited stress followed by family history, cholesterol and smoking, while men (n = 108) cited poor diet followed by smoking, stress, and family history. Women were also more likely to attribute their heart disease

to biological risk factors such as aging, diabetes, family history (uncontrollable factors) while men were more likely to attribute behavioural risk factors (controllable factors) as causes.

Astin & Jones (2004) also found quite a marked degree of discordance between patients' attributions and personal risk factor profile. Although 82% of men and 88% of women in this study had a history of high serum cholesterol levels, only 14% of men and 28% of women attributed this as a cause of their heart disease. The same was true for hypertension, while 56% of men and 50% of women had a history of hypertension, only 3% of men and 6% attributed this as a cause of their heart problem. Patients also under reported family history, smoking, and being over weight. Family history was recognised as a causal factor by 28% of the 42% of men with a positive family history of heart disease. Only 38% of the 60% of male smokers and 22% of the 44% of female smokers thought this was a cause of their heart disease, and being overweight was recognised as a causal factor by 6% of the 16% of men who were overweight and by only 3% of the 69% of women who were overweight.

A study by Murphy et al (2005) investigated causal attributions for CHD in 260 women admitted to hospital following an MI or for coronary artery bypass grafts (CABGs). This study also reported discordance between patients' causal beliefs and their personal risk profile. Patients were interviewed during the acute phase of their illness within 4 to 8 days of admission and in the non-acute phase 2, 4, and 12 months post-discharge. The perceived causal attributions of the women were compared with their risk factor profile and changes in causal attributions over the 12 month period post cardiac event.

The most common general attributions were family history, smoking, stress, no idea, diabetes, obesity, cholesterol, high fat diet, and hypertension. Attributions did not change significantly over time (12 months). Results, however, again showed a high level of discordance between patients' beliefs about the causes of their heart problems and their personal risk profiles. Of 180 hypertensive women, only 5% cited this as a cause of their CHD, and only 14% of 125 women with high cholesterol levels cited this as a cause, only 22% of diabetic women cited this as a cause. Smoking was cited as a cause by 22% of smokers and 40% of women with a positive family history cited this as a cause.

Cameron et al (2005) investigated causal attributions in a study of 65 first MI patients. Patients completed a questionnaire containing the psychological measures during the acute phase of their illness, within 2 days of admission. Results showed that 75% of patients believed that stress or worry caused their heart problems. This also received the highest mean rating and may also have been related to fatigue and overwork. This was followed by high cholesterol, heredity, fatty foods and high blood pressure, with depression and bad luck receiving the lowest causal attribution ratings. Unlike previous studies, risk factors correlated moderately well with associated causal attributions. Of patients with a previous history of hypertension, 85% believed that high blood pressure was a causal factor compared with 24% of non-hypertensives, 54% of patients with a family history agreed that heredity was a causal factor compared with 27% of patients with no family history, and 67% of patients with high cholesterol levels cited this as a causal factor compared with 23% of patients without high cholesterol levels. Gudmundsdottir et al (2001) also found that of the 62% of patients who were smokers in their study, the majority (59%) attributed smoking as a causal factor of their MI to smoking. Several reasons may explain the discrepancy in concordance between this

study and those discussed above. Studies using open-ended approaches require patients to make a definite statement of belief which they may feel reluctant to do, whereas studies using a list of cued attributions allow them to express weaker beliefs and remind them of possible causes. The quality and amount of information given to patients will also vary between hospitals, so that patients who receive educational advice with a heavy emphasis on behavioural risk factors will be more likely to make these attributions.

Martin et al (2005) investigated causal attributions in 157 patients post MI focussing particularly on gender differences. Participants were telephoned after hospital discharge and asked to provide a tape recorded narrative to 3 questions concerning their beliefs about the causes of their heart problems. Most common attribution was to stress (38%), followed by comorbid conditions (32%), diet (29%), smoking (19%), heredity (17%), lack of exercise (15%) and prior cardiac history (7%). Accuracy of patients' attributions in relation to their personal risk factors showed that only 29% of patients with a history of hyperlipidaemia and 35% of those who were obese attributed their MI to diet. Similarly, only 31% of patients judged to sedentary mentioned lack of exercise and 45% of current smokers cited smoking as a cause and 24% of patients with a family history of heart disease attributed heredity. Only 16% of patients who had suffered a previous MI believed that this had contributed to their current MI. Women were significantly less likely than men to attribute their MI to dietary factors and lack of exercise, and marginally less likely to attribute smoking behaviour. Martin et al hypothesised that women adopt self schemas different to men that produce a reduced perception of their vulnerability to MI which means that women would be less likely to attribute their MI to personal risk behaviours such as poor diet, lack of exercise, and smoking.

### *4.5.4: Qualitative approaches*

One criticism of the type of studies above investigating attributions is that they are reductionist, as they reduce the information given by participants to categories (Antaki, 1988; Antaki, 1994; Hewstone, 1989). Antaki (1988) argued that two potentially important sources of information are lost by this reductionism; firstly, patients' reasoning and justification behind the attributions are ignored; secondly, the context in which they are made and inferences about what is occurring may be misleading. A qualitative study by French et al (2005a) investigated the beliefs of 12 first time MI patients about the causes of their heart problem within one week of onset. The aim was also to explore the reasoning involved in the development of causal attributions, and the possible purposes solved by such causal attributions using interpretative phenomenological analysis. The most common single factor patients attributed to causing their heart problem however remained either smoking or stress. Despite of being aware of the chronic nature heart disease, some patients still attributed single causes as a trigger rather than the underlying dispositions. Patients tried to avoid blame by normalising unhealthy behaviour, using altruistic excuses or by emphasising the unpredictability of an MI. Patients were also concerned to assert control over a future MI by describing causal factors that could be avoided in the future, such as various kinds of psychological stresses (financial stress, over work, time pressures), high blood pressure or cholesterol levels which could be treated and controlled, increased physical exercise.

This study revealed a number of interesting points. Firstly, that although participants often made several causal attributions, they tended to settle on one factor as being the necessary cause. Secondly, some participants interpreted "cause" as being an ongoing disposition and others as an acute trigger. Thirdly, participants sought to avoid blaming

themselves or others in their search for a cause whilst simultaneously seeking to establish personal control over future reoccurrence. The authors suggest that stress may have been cited as a causal factor because it is a fairly flexible concept and serves as an uncontrollable demand with a controllable response. As French et al (2001) commented in an earlier review (described in section 4.4) researchers and patients may be trying to answer different questions. Patients may have been more concerned with why they had an MI at that time, while researchers tended to focus on risk factors that distinguish those who have an MI from those who do not.

## 4.6: Attribution studies in patient and non-patient samples

Studies including non-patient samples as well as patients with heart disease have provided some useful insights into lay attributions and research methods used. In their review, French et al (2001) found that there was an association between causal attributions and whether attributions were made by patients responding to questions about their own heart disease or by non-patients (spouses or unspecified others) responding to questions about other peoples' heart disease. Patients with heart disease were more likely to attribute their own heart problems to stressors and fate or luck and less likely to attribute hypertension, while non-patients gave higher attribution ratings than patients to being overweight and hypertensive. The authors suggest that this may be because bad luck or fate and stressors are viewed as being less controllable and allow patients to avoid taking responsibility for their heart disease and to avoid making negative evaluations of themselves. Attributions to hypertension were more likely in studies where respondents were asked about the causes of heart disease in unspecified others rather than their own heart problems. Attributions to being overweight and hypertension also ranked higher in studies where respondents were asked about the causes of heart problems affecting unspecified others. This may be because these

factors are seen as being subject to behavioural control, so although cardiac patients are aware that these are modifiable risk factors they may try to avoid responsibility for their own illness.

French et al (2002) later investigated how a sample of 107 adults with no history of heart disease perceived 8 putative causes of MI (hypertension, eating fatty food, high cholesterol levels, genes, lack of exercise, smoking, stress or worry, the type of work a person does) as causally relating to each other using network analysis. The type of work a person did was generally perceived as being a distal cause operating through stress and/or high blood pressure. High blood pressure was perceived as a key mediator, being causally influenced by many other putative causes (stress, smoking, high cholesterol levels, eating fatty foods, type of work a person does), and thus directly influencing heart attacks. A person's genes were not found to exert a causal influence on any other causal element. High blood pressure was much more often seen as being influenced by other putative causes (such as eating fatty foods, high levels of cholesterol, stress or worry and type of work a person does) than exerting a causal influence on these causes. This is interesting because it highlights a lack of understanding in the general population of the influence causal factors may have on each other. It also indicates that lay people may understand hypertension to be largely a psychosocial condition rather than a physiological one.

## 4.7: Attribution studies involving spouses of patients with heart disease

Spouses of patients with heart disease may play an important role in helping the patient interpret symptoms, adjust to the prognosis and to adopt behavioural changes. An early study compared patients and spouses causal explanations in 50 first MI patients at two time periods; within 48 hours of hospital discharge and one month later (Rudy, 1980).

Open ended questions and a questionnaire listing a list of causal items were used which participants were asked to rate in terms of importance. The most frequent causal explanation of patients and spouses was 'tension in life', related to the work or home situation. Factors rated as most important were worry, nerves, feeling tense, smoking, heredity and tension at work. Spouses were more likely to cite overwork as a cause. Many patients reported changing their smoking, diet and exercise behaviour indicating that although these behaviours were not named, they were considered as causal factors. The author suggested that tension is named most often as the cause because it is difficult to measure, allows the cause to be externalised and thus avoids issues related to self blame.

Weinman et al (2000) reported that the most commonly endorsed attributions for both MI patients and their spouses were stress, high cholesterol and various risk factors such a lack of exercise and high blood pressure. Patients (N = 143) completed questionnaires assessing their causal attributions and health related behaviour during their hospital stay and 6 months later. Most of the spouses (N = 84) were female and completed the questionnaire 12 weeks post MI. Patients who believed that their MI was caused by poor health habits were likely to have made dietary changes by 6 months. Spouses' attributions to poor health habits were associated with improvements in patients exercise levels at 6 months.

Arefjord et al (2002) also investigated the causal attributions of 37 wives of MI patients during the acute phase while their husbands were in hospital, 3 months and 10 years post-MI. Stress was seen as the main cause of the MI by wives, both in the acute phase and at the 10 year follow up. Common biomedical risk factors were mentioned as important during the acute phase although attributions to lifestyle factors increased over

the follow up period, and wives appeared to evaluate the MI within a mainly psychological and social framework. This may indicate a lack of knowledge of the role medical and lifestyle factors play. Causal attributions of the patients themselves were not reported, however this study supports earlier findings that stress is widely attributed as a main causal factor of MI by lay people, and that psychosocial rather than biomedical factors are more commonly perceived as causes of heart disease.

## 4.8: Attributions and pre-hospital delay

Several studies have examined patients' symptom attributions in relation to pre-hospital delay, as noted in Chapter 2 (section 2.3.7.2). One of the most common factors predicting shorter pre-hospital delays was patients' attribution of their symptoms to a heart problem. Results from an investigation of pre-hospital delay using focus groups revealed that patients' expectations of heart attack are that it is a sudden, severe episode of chest pain causing collapse, as often portrayed in the movies, and that they underestimated their personal risks (Finnegan, Jr. et al, 2000). Most studies have found that patients who recognised that their symptoms were cardiac rather than attributing them to another cause such as indigestion, had shorter pre-hospital delays (Carney et al, 2002; O'Carroll et al, 2001). The majority of patients, however, attribute their symptoms to other causes, particularly indigestion (Carney et al, 2002) and as few as 17% of MI patients recognise their symptoms as being those of a heart attack (O'Carroll et al, 2001). Horne et al (2000) found that patients experience longer delays when there is a mis-match between symptoms patients actually experience and those they expect would indicate a heart attack. Evidence from other studies has supported this (Perry et al, 2001; Zerwic, 1998). It is possible that some patients lacked knowledge of the symptoms of a heart attack, or that they simply denied the more serious implications of their symptoms and preferred to make attributions with less serious consequences (as

discussed in chapter 2). Contradictory findings, however, were reported in a study by Walsh et al (2004), who found that symptom identity was not predictive of patient delay. In a review by French et al (2001) chronic stress and lifestyle factors were found to be the most common causal attributions for heart disease but this was not investigated specifically in relation to pre-hospital delay.

Few studies, however, have investigated causal attributions about heart disease specifically in relation to pre-hospital delay in patients suffering symptoms of ACS. Two qualitative studies have examined patients' decision making process and pre-hospital delay. A study by Pattenden et al (2002) identified six themes that influence patients' decision making processes during onset of acute cardiac symptoms, including appraisal of symptoms, perceived risk, previous experience, psychological and emotional factors, use of the NHS, and the context of the event such as time, place and presence of a bystander. Ruston et al (1998) found that knowledge of a wider range of symptoms, recognition of personal risk and vulnerability to heart attack and correct attribution of symptoms to a cardiac cause predicted shorter pre-hospital delay. There is, however, a lack of research investigating the association between causal attributions and pre-hospital delay.

## 4.9: Summary

The studies reviewed above confirm the importance of causal attributions in patients' response to heart disease, however there little previous research which has investigated the association between causal beliefs and pre-hospital delay. The most common causal attributions of heart problems made by patients and lay people in the studies reviewed above, regardless of differing methodologies, are psychosocial, and include stress,

lifestyle factors or personal behaviour, and heredity. This could be seen to contradict medical opinion which tends to emphasize biological rather than psychosocial causes.

The accuracy with which patients attribute symptoms may have an important influence on their response to the symptoms and recovery. Patients interpret their symptom experiences depending on their understanding and beliefs about heart attack and the symptoms they associate with it (Baumann et al, 1989). In particular, patients' beliefs about illness identity and cause may be important in determining their response following the onset of symptoms of ACS, and may influence the time it takes to them to decide to seek medical help (Horne et al, 2000). Causal attributions are core components of patients' mental representations of their illnesses and have been shown to predict recovery behaviour, such as dietary change and exercise, return to work and reoccurrence of angina symptoms post AMI (Petrie et al, 2002; Weinman et al, 2000). These beliefs may be inaccurate or incorrect. It has also become apparent from the studies above (Astin & Jones, 2004; Cameron et al, 2005; Murphy et al, 2005; Zerwic et al, 1997) that patients' causal beliefs may be different to the beliefs and perceptions about aetiology held by health care professionals, and conflicting models of illness may impede effective communication.

There is little previous research which has investigated the association between causal beliefs about heart disease and pre-hospital delay. This thesis hypothesizes that longer pre-hospital delay and decision time in seeking help will be associated with patients' beliefs about the causes of their heart problem, independently of clinical severity. To date previous literature does not lead to specific predictions about the precise association between causal beliefs and delay.

# Chapter 5: Associations between patients' causal attributions of their heart problem and their decision to seek help

## 5.0: Introduction

Patients' beliefs about the causes of their heart problem may influence their help seeking behaviour following the onset of acute cardiac symptoms. Differences in causal beliefs may help to explain the variations in pre-hospital delay between patients who have short pre-hospital delays and short decision delays, and those who delay for longer.

## 5.1: Aims

The second aim of this thesis is to investigate the associations between patients' beliefs about the causes of their heart problem and their decision to seek help following the onset of symptoms of ACS.

## 5.2: Hypothesis

The second hypothesis of this thesis is that longer pre-hospital delay and decision time in seeking help is associated with patients' beliefs about the causes of their heart problem, independently of clinical severity. Previous literature does not lead to specific predictions about the precise association between causal beliefs and delay.

## 5.3: Methodology

### 5.3.1: Participants & procedure

The study population consisted of 269 patients, recruited as described earlier. Following a structured interview (described in Chapter 3.2.4), all participants were asked to complete a questionnaire pack in private containing the psychosocial measures. Of these, 171 participants returned their baseline questionnaires. Six participants answered fewer than 10 items and were excluded, leaving 165 participants who provided adequate data for this stage of the analysis (see Appendix 1).

## 5.4: Measures

### 5.4.1: Patients' beliefs about the causes of their heart problem

Patients' beliefs concerning the causes of their heart problem and heart disease symptoms were measured using a questionnaire based on the major categories of causal attribution described by French et al (2001) and Gudmundsdottir et al (2001) and on the causal belief items from the Illness Perception Questionnaire (Weinman et al, 1996). It consisted of 16 items such as "My illness is hereditary – it runs in my family", "Being overweight caused my illness", "A germ or virus caused my illness" etc. Answers were scored as yes (2), maybe (1) and no (0) (Appendix 7). Scores could range from 0 – 32. This questionnaire was delivered at the baseline assessment and repeated at again after 3 months and 13 months.

### 5.4.2: Measurement of life stress.

During their interview patients were asked if they felt they had experienced stress in the 4 weeks and/or 6 months caused by their partner, family, work, or other illnesses prior to the onset of their symptoms of ACS (Appendix 4, question 41 and 42). An example of one of these questions would be "In the past 4 weeks has your relationship with your partner been stressful?". Patients could answer 'yes' or 'no'. If the answer was 'yes', they were asked to rate the amount of stress they had been feeling for each of these 4 possible sources of stress separately on a scale ranging from 1 (low stress) to 4 (high stress). Patients were also asked if their had felt more tired or fatigued than usual over the previous 4 weeks and 6 months, possible answers were no (0) or yes (1).

## 5.5: Statistical analyses

Data were collected on 16 possible causes of heart problems. The frequency with which each item was endorsed is summarised in Table 5.1. The two items with the lowest scores: "My illness was caused by poor medical care in the past" and "A germ or virus caused my illness" were excluded since they were endorsed by only a small number of participants. The two columns indicate the proportion of patients who said that the item was definitely important and the mean rating on the 0-2 scale.

**Table 5.1: Summary of responses to causal attributions of heart problem**

| Items | Definitely Yes (%) | Mean rating of score* |
|---|---|---|
| Smoking | 30.3 | 0.86 |
| Stress | 25.2 | 0.90 |
| Bad luck | 21.2 | 0.70 |
| High blood pressure | 20.9 | 0.75 |
| Heredity | 20.1 | 0.69 |
| Over exertion | 11.5 | 0.50 |
| Poor diet | 10.4 | 0.57 |
| State of mind | 10.4 | 0.50 |
| Genetic factors | 9.8 | 0.52 |
| Lack of exercise | 9.2 | 0.52 |
| Other medical problems | 9.1 | 0.47 |
| Tiredness | 8.5 | 0.52 |
| Working too hard | 8.5 | 0.46 |
| Over weight | 6.7 | 0.46 |
| Poor medical care in past | 1.9 | 0.15 |
| Virus or germ | 0.6 | 0.10 |

*Possible answers were yes / maybe / no (scored 2 – 0)

In order to discover whether these possible causes fell into meaningful groups, I carried out factor analysis on the results.

### 5.5.1: Five factor solution

A factor analysis with varimax rotation yielded 5 factors with an eigenvalue of >1, (Figure 5.1). This was the initial factor solution that was explored.

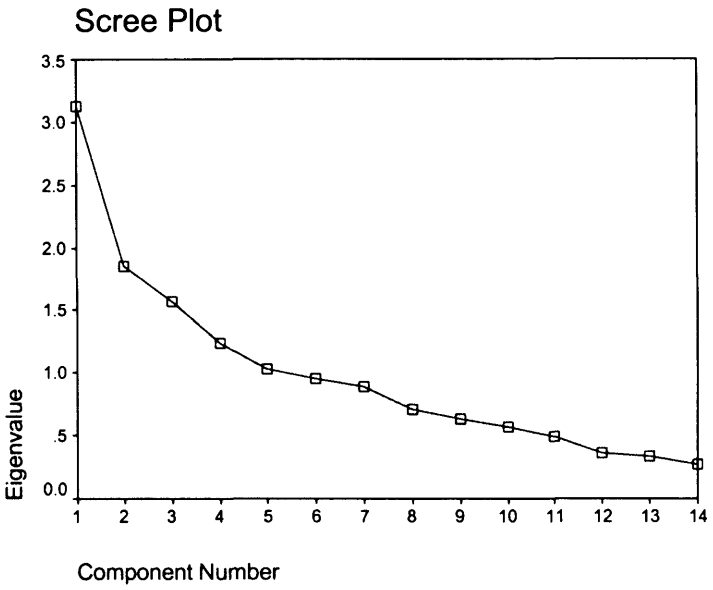


**Figure 5.1: Scree plot showing eigenvalues for the 5 factor analysis of causal attributions.**

These five factors together accounted for 62.94% of the variance as shown in Table 5.2.

**Table 5.2: Variance according to 5 factor solution**

| Factor | % Variance | Cumulative % of variance |
|---|---|---|
| 1 | 16.70 | 16.70 |
| 2 | 14.95 | 31.65 |
| 3 | 12.01 | 43.66 |
| 4 | 10.20 | 53.87 |
| 5 | 9.08 | 62.94 |

The items loading onto the 5 different factors in the rotated matrix are shown in Table 5.3.

**Table 5.3: Items loading onto the five factor solution**

| Item | Component | | | | |
|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 |
| Stress | 0.854 | | | | |
| State of mind | 0.752 | | | | |
| Over exertion | 0.630 | | | | |
| Overweight | | 0.838 | | | |
| Poor diet | | 0.653 | | | |
| Lack of exercise | | 0.632 | | | |
| Bad luck | -0.428 | -0.526 | | | |
| Genetic factors | | | 0.894 | | |
| Heredity | | | 0.857 | | |
| Smoking | | | | 0.751 | |
| Tiredness | 0.462 | | | 0.622 | |
| Working too hard | 0.421 | | | 0.621 | |
| Other medical problems | | | | | 0.853 |
| High blood pressure | | | | | 0.474 |

Rotated component matrix. Extraction method: Principal component analysis. Rotation method: Varimax with Kaiser Normalization. Rotation converged in 6 iterations.

Items loading onto factor 1 were stress, state of mind, over exertion, bad luck, tiredness and working too hard. This factor appears to relate mostly to mental state and feelings of exhaustion. The item referring to bad luck is slightly odd and is reversed meaning that participants who endorsed the other items thought that bad luck was not a cause of their heart problem. Items loading onto factor 2 were overweight, diet, lack of exercise and low levels of bad luck. This factor appears to relate to relate to physical condition, but it is a mixed factor with bad luck appearing both in factors 1 and 2. Items loading onto factor 3 were genetic causes and heredity, and clearly relates to the belief that heart disease is a condition which is inherited from one's family. Items loading onto factor 4 were smoking, tiredness and working too hard which may relate to over work or work stress but is rather confusing. This is a mixed factor with working too hard and tiredness both appearing both in factor 1 and factor 4. Items loading onto factor 5 were

other medical problems and high blood pressure. This appears to relate to general medical condition but seems rather vague. This factor solution is unsatisfactory because it contains two mixed factors with some items loading highly onto more than one factor. Only items loading at 0.30 or greater are listed.

### 5.5.2: Four factor solution

Next, a four factor solution was examined. These four factors together accounted for 55.61% of the variance as shown in Table 5.4.

**Table 5.4: Variance according to 4 factor solution**

| Factor | Cumulative % of variance |
|---|---|
| 1 | 18.98 |
| 2 | 34.30 |
| 3 | 46.23 |
| 4 | 55.61 |

This four factor solution is also unsatisfactory because there are three mixed factors and the factors are not generally coherent (see Table 5.5). The item relating to state of mind, for example, now loads onto both factor 1 and factor 4. While factor 1 seems to relate largely to mental state, factor 4 is more concerned other medical problems, and mental state does not fit well into this factor. Over exertion loads onto factors 1 and 2, relating both to mental state and behavioural risk factors. Bad luck is also loads onto factor 2 so people who think that their lifestyle is important do not think their heart problem was caused by luck . High blood pressure loads onto factors 2 and 4, relating to behavioural risk factors in factor 2 and other medical problems in factor 4.

**Table 5.5: Items loading onto four factor solution**

| Item | Component | | | |
|---|---|---|---|---|
| | 1 | 2 | 3 | 4 |
| Stress | 0.763 | | | |
| State of mind | 0.713 | | | 0.333 |
| Tiredness | 0.711 | | | |
| Working too hard | 0.694 | | | |
| Over exertion | 0.599 | 0.302 | | |
| Overweight | | 0.846 | | |
| Poor diet | | 0.633 | | |
| Lack of exercise | | 0.628 | | |
| Bad luck | | -0.563 | | |
| Smoking | | 0.314 | | |
| Genetic factors | | | 0.893 | |
| Heredity | | | 0.856 | |
| Other medical problems | | | | 0.830 |
| High blood pressure | | 0.316 | | 0.522 |

Rotated component matrix. Extraction method: Principal component analysis. Rotation method: Varimax with Kaiser Normalization. Rotation converged in 5 iterations.

### 5.5.3 Three factor solution

A three factor solution was then examined. These three factors together accounted for 46.81 % of the variance as shown in Table 5.6.

**Table 5.6: Variance according to the three factor solution**

| Factor | Cumulative % of variance |
|---|---|
| 1 | 19.12 |
| 2 | 34.89 |
| 3 | 46.81 |

Table 5.7 shows the items that loaded onto the three factor solution at 0.3 or greater following varimax rotation. Items loading onto factor 1 were stress, state of mind, tiredness, working too hard and over exertion. All the items in this factor seem to relate to aspects of mental state. Participants believed that their heart problem was caused by a negative mental state involving stress and tiredness and possibly brought on by

working too hard and over exertion. The internal reliability of this 'mental state' factor was good (Cronbach alpha 0.77).

Items loading onto factor 2 were overweight, diet, lack of exercise, bad luck (reversed) and high blood pressure. This seems to suggest that participants believed certain risk factors associated with lifestyle choices may have caused their heart problem. People who endorsed these personal behaviour factors were also less likely to believe that bad luck was relevant. The internal reliability was moderate (Cronbach alpha 0 .59). Factor 3 contained two items reflecting the belief that heart problems are inherited, genetic factors and heredity. The internal reliability (Cronbach alpha) was 0 .76.

**Table 5.7: Items loading onto three factors at ≥0.3.**

| Item | Component | | |
|---|---|---|---|
| | 1 | 2 | 3 |
| Stress | 0.770 | | |
| State of mind | 0.726 | | |
| Tiredness | 0.710 | | |
| Working too hard | 0.686 | | |
| Over exertion | 0.599 | | |
| Overweight | | 0.797 | |
| Poor diet | | 0.687 | |
| Lack of exercise | | 0.647 | |
| Bad luck | | -0.480 | |
| High blood pressure | | 0.450 | |
| Smoking | | | |
| Heredity | | | 0.867 |
| Genetic factors | | | 0.855 |
| Other medical problems | | | |

Rotated component matrix. Extraction method: Principal component analysis. Rotation method: Varimax with Kaiser Normalization. Rotation converged in 4 iterations.

The three factor solution appeared to be the most appropriate solution to investigate causal attributions in relation to pre-hospital delay, decision time and home to hospital delay. The item groupings are more coherent and sensible than other solutions, and reflect 3 different aspects of causal beliefs; "mental state" (factor 1), "personal

behaviour" (factor 2) and "heredity" (factor 3). Factor scores were therefore constructed for these three factors by summing ratings on the individual contributing items; the item 'bad luck' was reverse scored for factor 2. Totals could range from 0 – 10 (factors 1 and 2) and from 0 – 4 (factor 3). To ensure comparability, the three factors were all scaled to a range of 0 – 10, with high ratings indicating very strong beliefs in the relevance of this factor.

T-tests were used to test the association between the categories of pre-hospital delay (e.g. less than or greater than 60 minutes, or less than or greater then 120 minutes), decision time (less than or greater than 30 minutes, or less than or greater than 60 minutes), and home to hospital delay (less than or greater than 120 minutes) and each of the three factors. When effects were significant, I ran a logistic regression on the delay variables with age and gender as covariates. The reference category was the shorter delay category (less than 60 minutes or less than 120 minutes). The odds of a short delay with 95% confidence interval are presented.

## 5.6: Results

### 5.6.1: Population characteristics of patients with data on causal attributions.

Comparison between patients who had completed at least 10 causal attribution items on their questionnaires (N = 165) with those who had not (N = 104) in the complete study population (N = 269) revealed no significant differences between the 2 groups for any of the major clinical, demographic and psychological variables. Only 3 variables showed significant differences between the two groups. These included season, number of symptoms except chest pain, and number of non-pain symptoms. The presence of diabetes also approached significance ($p = 0.06$). Logistic regressions were carried out to determine the strength and direction of these variables (see Table 5.8).

**Table 5.8 Comparison between characteristics of patients who completed ≥10 causal attributions and patients who completed < 10 causal attributions**

| Variable | | Odds ratio ( 95% C.I.) adjusted for age and gender of ≥10 causal attributions. | | Adjusted p-value* |
|---|---|---|---|---|
| **Season:** | Jan - Mar | 1 | | |
| | Apr – June | 0.41 | (0.21 – 0.81) | **0.010** |
| | July - Sept | 0.44 | (0.22 – 0.88) | **0.019** |
| | Oct - Dec | 3.23 | (1.23 – 8.20) | **0.014** |
| Diabetes: | No | 1 | | |
| | Yes | 0.51 | (0.25 – 1.03) | 0.061 |
| **Number of non-chest pain symptoms:** | | | | |
| | none | 1 | | |
| | 1 – 3 | 0.42 | (0.22 – 0.80) | **0.008** |
| | 4 – 8 | 0.29 | (0.15 – 0.58) | **<0.001** |
| **Number of non-pain symptoms:** | | | | |
| | none | 1 | | |
| | 1 – 2 | 0.27 | (0.13 – 0.55) | **<0.001** |
| | 3 - 6 | 0.15 | (0.07 – 0.33) | **<0.001** |

* adjusted for age and gender

Compared with patients admitted from January to March, patients admitted to hospital from April to June, and July to September were less likely to complete at least 10 causal attributions, while patients were more likely to complete 10 or more causal attributions if they admitted between October and December (see Table 5.8). The reason for this is unclear and could be due to chance, but during the autumn/winter months patients may have had fewer visitors and spent more time completing the questionnaire, or perhaps have felt more reflective as end of the year drew closer. Patients who suffered a greater number of symptoms other than chest pain (with or without pain elsewhere) were less likely to make at least 10 causal attributions than patients who suffered no symptoms except chest pain possibly because they had a more confused picture of their illness and found it more difficult to make clear attributions (see Table 5.8). This may also explain why diabetic patients were less likely to make at least 10 attributions than patients who were not diabetic.

### 5.6.2: Overall analysis of causal beliefs:

#### *5.6.2.1: Most common attributions.*

It can be seen in Table 5.1 that smoking, stress, bad luck, high blood pressure and heredity were all endorsed by more than 20% of participants. There was then a gap, since the next highest item (over exertion) was endorsed by 11.5%. The frequency distribution of individual scores for each of the 3 factors is shown in Figures 5.2 to 5.4.

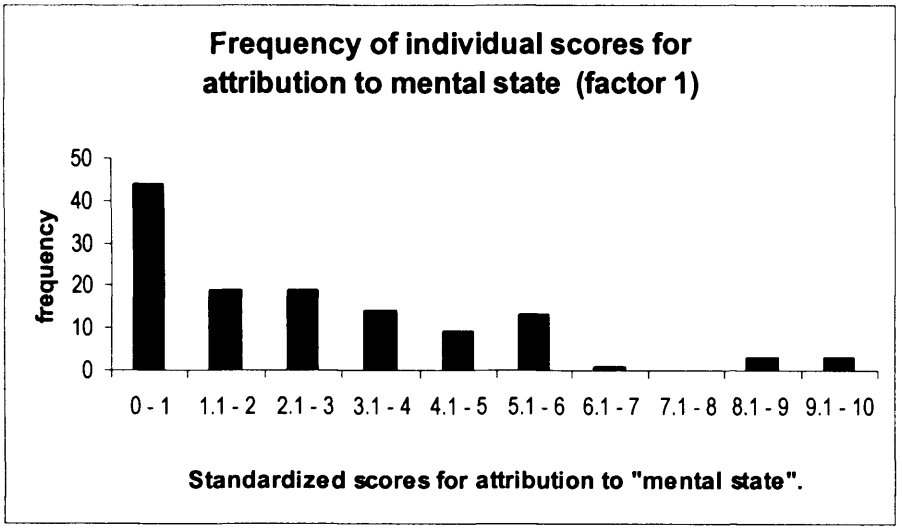


**Figure 5.2: Frequency distribution of individual scores for attribution to mental state**

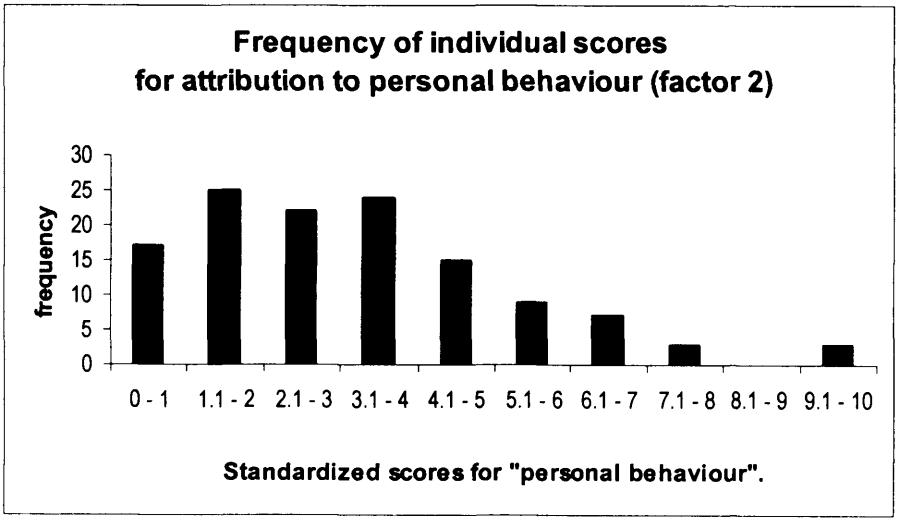


**Figure 5.3 Frequency distribution of individual scores for attribution to personal behaviour.**

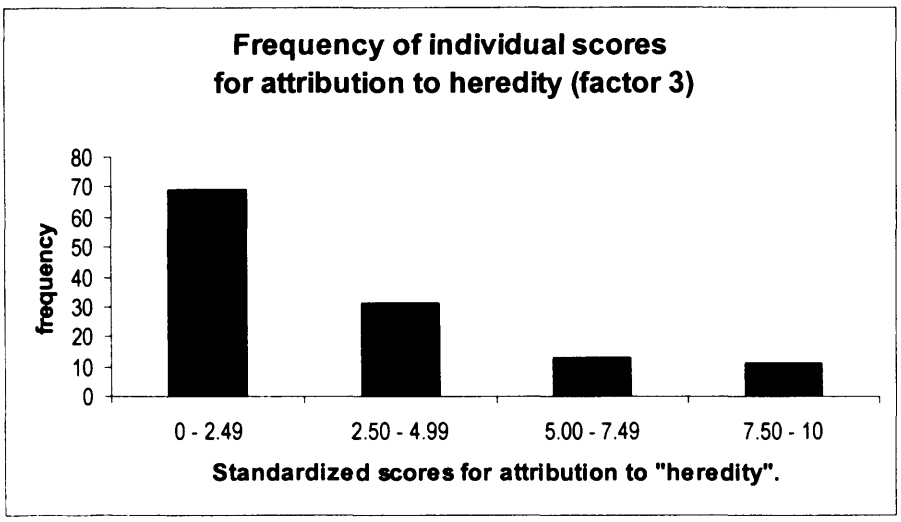


**Figure 5.4 Frequency distribution of individual scores for attribution to heredity.**

### *5.6.2.2: Overall comparison of the mean scores of the individual factors.*

The mean scores for each of the three factors were compared using a repeated measures analysis of variance. The mean score for patients' belief that mental state caused their heart problem (factor 1) was 2.79 (SD 2.39), the mean score for patients' belief that personal behaviour caused their heart problem (factor 2) was 3.65 (SD 2.14) and the mean score for the belief that heredity was a cause (factor 3) was 3.27 (SD 3.36). There was a significant difference between the 3 factors ($F = 3.87$, $p = 0.022$). The highest mean score was for the personal behaviour factor meaning that patients had stronger beliefs that personal lifestyle behaviours (such as poor diet, lack of exercise, hypertension and being overweight) caused their heart problem than the other two factors, mental state or heredity. The lowest overall mean score was for patients' belief that their mental state caused their heart problem.

### *5.6.2.3: Were attributions to the individual factors influenced by gender or age ?*

When beliefs in each of the three factors were examined for gender differences using t-tests there were no significant differences between men and women for belief that either mental state ($F = 0.27$, $p = 0.61$) or heredity ($F = 3.14$, $p = 0.08$) were causal

factors. There was a significant difference in means between men and women however, for the belief that personal behaviour caused their heart problem. The mean score for men was 3.84, SD 0.21, and for women it was 2.70, SD 2.23 ($p = 0.019$) indicating that men had a stronger belief than women that personal behaviour caused their heart problem.

Comparison of age categories (<50 years, 50 to 60 years, 60-70 years, and >70 years) showed no significant association between age and the belief that mental state was a cause of their heart problem ($F = 1.84$, $p = 0.143$). Table 5.9 shows that younger patients believed more strongly that personal behaviour caused the heart problem ($F= 3.97$, $p = 0.01$). This may also reflect the fact that although there are fewer women this study, they tended to be older than men and, as shown above, were less likely to believe strongly that personal behaviour caused their heart problem. The belief that heredity caused their heart problem was not significantly related to age group ($F = 0.78$, $p = 0.51$).

**Table 5.9: Mean scores for patients' beliefs that their heart problem was caused by the personal behaviour factor by age group**

| Age Group (in years) | N (125) | Mean (SD) | F value | P value |
|---|---|---|---|---|
| <50 | 30 | 4.03 (±2.76) | 3.97 | **0.01** |
| 50 – 60 | 43 | 4.14 (±1.96) | | |
| 60 – 70 | 27 | 3.41 (±1.47) | | |
| >70 | 25 | 2.46 (±1.89) | | |

### *5.6.2.4: Accuracy of patients' causal attributions and their personal risk profile*

The causal belief that was most strongly endorsed by patients in this study was that smoking caused their heart problem. A total of 30.3% of participants in the sample

strongly endorsed smoking as a causal attribution (see Table 5.1). It is possible, however, that this attribution was strongly endorsed only by patients who were current smokers. Analysis of data using a Chi squared test for association and t-tests revealed that indeed 53.4% of smokers strongly endorsed the statement that smoking was a causal attribution compared with only 12.0% of non-smokers ($p < 0.01$). A comparison of means using a t-test revealed a significant difference between non-smokers (mean score of 0.40, SD 0.70) and smokers (mean score 1.44, SD 0.67) showing a positive association between patients who were current smokers and a strong belief that smoking caused the heart problem ($p < 0.001$) compared with non-smokers.

In this sample, a total of 20.9% of all participants believed that high blood pressure caused their heart problem (see Table 5.1). Analysis of data using t-tests showed that patients who had previously been diagnosed as hypertensive had higher mean scores (1.32, SD 0.64, $p < 0.001$) for beliefs that hypertension was a cause of their heart problem than patients who had no previous diagnosis of hypertension (mean score 0.30, SD 0.075). Among previously diagnosed hypertensive patients, analysis using chi squared tests showed that 41.1% believed that hypertension was a causal factor compared with 4.4% of patients with no such previous diagnosis.

As shown in Table 5.1, 20.1% of patients in this sample endorsed the item concerning their belief their heart disease was hereditary (item 1 in the causal beliefs questionnaire: 'My illness is hereditary – it runs in my family', Appendix 7). Analysis using chi squared tests showed that 33.0% of patients had a relative who had suffered with heart disease and believed that heredity was a cause of their own heart problem compared with 1.5% of patients who had no such family history who believed that heredity caused their heart problem. These patients also had higher mean scores for the belief that

hereditary factors caused their ACS (mean 1.05, SD 0.78) compared with patients who did not have relatives who had suffered with heart disease (mean 0.15, SD 0.40) ($p < 0.001$). Similarly, Table 5.1 shows that overall, 9.8% of patients strongly endorsed the attribution referring to genetic factors (item 13 in the causal attributions questionnaire: 'Genetic factors caused my illness', Appendix 7). Chi squared tests showed that 14.3% of patients with a family history of heart disease had strong beliefs that genetic factors caused their illness while only 3.1% of patients without a positive family history shared this belief. Patients who had a relative with heart disease also had higher mean scores for the belief in genetic factors as a cause of their heart problem (mean 0.79, SD .68) compared with patients who did not have relatives who had suffered with heart disease (mean 0.23, SD 0.49)($p = 0.001$).

In this study, 9.2% of all patients endorsed the statement that lack of exercise was a cause of their heart problem (see Table 5.1). Results of Chi Squared test showed that twice as many participants who did no exercise (10.6%) strongly believed that lack of exercise was a cause of their heart problem compared with participants who did some regular exercise (5.2%) ($p = 0.24$), although this was not significant. Patients who did no exercise at all however, had higher mean scores (0.60, SD 0.68) in their belief that lack of exercise caused their heart problem than patients who did some regular exercise (0.33, SD 0.58) ($p = 0.028$).

### 5.6.3: Was pre-hospital delay associated with patients' causal beliefs ?

When the three causal factors were analysed, only the mental state factor showed a significant association with pre-hospital delay. Logistic regression indicated that the odds ratio for having shorter than average total pre-hospital delay for mental state factor was 0.85 (95% C.I. 0.73 to 0.99, $p = 0.036$) (see Table 5.10). This means that for every

point increase in the mental state factor score, the odds of having a less than average total pre-hospital delay decreased by 15%. Thus, stronger beliefs that mental state was a cause of the heart problem were associated with longer delays.

**Table 5.10 Association between the 3 factors and pre-hospital delay <120 mins**

| Factor | Odds ratio (95% Confidence interval) of total pre-hospital delay < 120 mins | p-value |
|---|---|---|
| **Factor 1: Mental state** | | |
| Age | 0.75 (0.52 – 1.10) | .138 |
| Gender | 1.58 (0.60 – 4.14) | .356 |
| **Mental state** | **0.85 (0.73 - .99)** | **.036** |
| **Factor 2: Personal behaviour factors** | | |
| Age | 0.78 (0.54 – 1.13) | .186 |
| Gender | 1.60 (0.61 – 4.20) | .340 |
| Personal behaviour | 0.95 (0.80 – 1.13) | .559 |
| **Factor 3: Heredity** | | |
| Age | 0.83 (0.57 – 1.19) | .300 |
| Gender | 1.55 (0.59 – 4.09) | .374 |
| Heredity | 1.02 (0.92 – 1.14) | .667 |

There was no significant association however, between patient decision time and their attribution to any of the causal factors, including mental state (see Table 5.11).

**Table 5.11: Association between the 3 factors and patient decision time <60 mins**

| Factor | Odds ratio (95% Confidence interval) of decision delay < 60 mins | p-value |
|---|---|---|
| **Factor 1: Mental state** | | |
| Age | 0.76 (.45 – 1.29) | .315 |
| Gender | 1.99 (.57 – 6.96) | .280 |
| Mental state | 1.05 (.86 – 1.27) | .649 |
| **Factor 2: Personal behaviour** | | |
| Age | 0.76 (0.45 – 1.30) | .319 |
| Gender | 2.19 (0.62 – 0.67) | .222 |
| Risk factors | 1.04 (0.83 – 1.31) | .735 |
| **Factor 3: Heredity** | | |
| Age | 0.77 (0.46 – 1.31) | .342 |
| Gender | 1.94 (0.55 – 6.85) | .306 |
| Heredity | 1.06 (0.92 – 1.22) | .449 |

Rather, there was a significant association between patients' belief that their mental state caused their heart problem and home to hospital delay. Patients who attributed their symptoms to their mental state had an odds ratio of 0.84 (95% C.I. 0.71 to 1.00, p = 0.054) of having a home to hospital delay of less than 120 minutes compared to patients who did not attribute their symptoms to their mental state (see Table 5.12). As discussed earlier, there was also an association between age and home to hospital delay (see section 3.3.8) whereby younger patients are more likely than older patients to have a short home to hospital delay.

**Table 5.12: Association between the 3 factors and short home to hospital delay**

| Factor | Odds ratio (95% Confidence interval) of home to hospital delay < 120 mins | p-value |
|---|---|---|
| **Factor 1: Mental state** | | |
| Age | 0.571 (.36 – .90) | .016 |
| Gender | 1.98 (.60 – 6.50) | .262 |
| **Mental state** | **0.84 (.71 – 1.00)** | **.054** |
| **Factor 2: Personal behaviour** | | |
| Age | .68 (.43 – 1.05) | .080 |
| Gender | 2.21 (.63 – 7.76) | .215 |
| Heredity | 1.06 (.84 – 1.32) | .644 |
| **Factor 3: Heredity** | | |
| Age | .64 (.41 – 1.00) | .048 |
| Gender | 2.23 (.63 – 7.81) | .215 |
| Heredity | 1.00 (.88 – 1.13) | .969 |

Interestingly then, the association between the belief that mental state caused their heart problem and total pre-hospital delay following symptom onset was not due to patients' decision time, but to home to hospital delays and what happened after the patient called for help and before they reached hospital.

### 5.6.4: Background characteristics associated with attributions to mental state

One-way analysis of variance was used to investigate associations between the mental state factor and previous medical history, psychological factors, and symptoms at onset

(see Table 5.13). Tukey post hoc tests were applied to variables of more than 2 levels. The presence of diabetes was the only aspect of patients' previous medical history to show a significant association with "mental state". Patients who were diabetic had a stronger belief that their heart problems were due to their mental state than patients who were not diabetic (see Table 5.13). Patients who felt that they had been under stress at work, in their family or with their partner in the 4 weeks and 6 months preceding their heart problem had a stronger belief that their mental state was a causal factor of their ACS (see Table 5.13). Patients who admitted to feeling stressed from other illnesses and more fatigued in the previous 4 weeks and 6 months also had a stronger belief that their mental state caused their heart problem than patients who did not report this. People with a large social network had a stronger belief that their mental state caused their heart problem than patients with smaller social networks, as did patients who had a previous history of depression, and those who suffered from diabetes. People who had lower mean scores on the cardiac denial of impact scale also had higher mean scores for the belief that their heart problem was caused by their mental state than patients with lower mean scores on the cardiac denial of impact scale. Patients with low levels of denial therefore had strong beliefs that their mental state caused their heart problem. Patients who suffered lower levels of pain intensity (pain score $\leq 6$) at onset had a stronger belief that their mental state caused their heart problem than patients who suffered more intense pain (pain score $>6$), and/or no symptoms other than chest pain had higher scores on the mental state factor. Other demographic, socio-economic, social and proximal factors showed no significant associations.

**Table 5.13: Mean scores for patients' belief that their heart problem was caused by mental state and their background characteristics**

| Independent variable | | Mean (SD) | | F value | p- value |
|---|---|---|---|---|---|
| Diabetes: | Yes | 4.13 | (±3.36) | 4.78 | **.031** |
| | No | 2.67 | (±2.29) | | |
| Previous MI : | Yes | 2.71 | (±2.44) | 3.45 | .066 |
| | No | 4.04 | (±2.45) | | |
| Type of MI: | UA/NSTEMI | 3.52 | (±2.73) | 3.23 | .075 |
| | STEMI | 2.61 | (±2.35) | | |
| Family stress in 4 weeks pre-ACS: | None-Mild | 2.49 | (±2.20) | 12.64 | **.001** |
| | Moderate to Very Stressful | 4.55 | (±3.10) | | |
| Family stress in 6 months pre ACS: | None-mild | 2.66 | (±2.41) | 60 | **.020** |
| | Moderate to Very Stressful | 4.12 | (±2.64) | | |
| Work stress in 4 weeks pre-ACS: | None-Mild | 2.35 | (±2.14) | 10.96 | **.001** |
| | Moderate to Very Stressful | 4.24 | (±2.91) | | |
| Work stress in 6 months pre-ACS: | None-Mild | 2.35 | (±2.23) | 13.97 | **<0.001** |
| | Moderate to Very Stressful | 4.48 | (±2.68) | | |
| Fatigue in 4 weeks pre-ACS: | None-Mild | 2.33 | (±2.17) | 2.17 | **.041** |
| | Moderate to Very Stressful | 3.24 | (±2.64) | 2.64 | |
| Fatigue in 6 months pre-ACS: | None-Mild | 2.07 | (±1.86) | 28.84 | **<0.001** |
| | Moderate to Very Stressful | 4.33 | (±2.82) | | |
| Partner stress in 4 weeks pre-ACS: | None-Mild | 2.85 | (±2.53) | 5.99 | **.016** |
| | Moderate to Very Stressful | 4.53 | (±1.92) | | |
| Partner stress in 6 months pre-ACS: | None-Mild | 2.84 | (±2.49) | 5.97 | **.016** |
| | Moderate to Very Stressful | 4.62 | (±1.98) | | |
| Stress from other illnesses in 4 weeks pre-ACS: | No | 2.62 | (±2.38) | 4.27 | **.041** |
| | Yes | 3.75 | (±2.68) | | |
| Stress from other illnesses in 6 months pre ACS: | | | | | |
| | No | 2.53 | (±2.24) | 7.02 | **.009** |
| | Yes | 3.89 | (±2.92) | | |

| Independent variable | | Mean (SD) | | F value | p- value |
|---|---|---|---|---|---|
| Social network: | Small | 1.73 | (±1.63) | 4.13 | **.018** |
| | Medium | 3.27 | (±2.84) | | |
| | Large | 3.08 | (±2.29) | | |
| History of depression: | No | 2.54 | (±2.34) | 7.65 | **.007** |
| | Yes | 4.01 | (±2.65) | | |
| Cardiac denial: | Lowest tertile | 3.37 | (±2.44) | 3.34 | .070 |
| | Higher tertiles | 2.54 | (±2.44) | | |
| Intensity of pain: | <6 | 3.65 | (±2.56) | 4.01 | **.022** |
| | 6 – 8 | 2.16 | (±1.86) | | |
| | >8 | 2.07 | (±2.08) | | |
| Number of non-pain symptoms: | None | 3.89 | (±2.76) | 3.70 | **.028** |
| | 1 - 2 | 2.74 | (±2.37) | | |
| | 3 - 6 | 2.23 | (±2.21) | | |
| Number of non-chest pain symptoms: | None | 4.00 | (±2.88) | 3.97 | **.021** |
| | 1 - 3 | 2.76 | (±2.27) | | |
| | 4 - 8 | 2.11 | (±2.38) | | |

## 5.7: Discussion

The aim of the study was to examine the associations between patients' beliefs about the causes of their heart problem and pre-hospital delay, and it was hypothesised that longer pre-hospital delay and decision time in seeking help would be associated with patients' beliefs about the causes of their heart problem, independently of clinical severity. Results of this study supported that hypothesis in that patients' belief that their heart problem was caused by their mental state significantly predicted greater pre-hospital delay. However this was in relation to the home to hospital phase of delay rather than decision delay.

### 5.7.1: The general pattern of attributions

The pattern of attributions reported in the present study generally supports findings from a recent systematic review and previous studies (French et al, 2001; Cameron et al, 2005; De Valle & Norman, 1992; Weinman et al, 2000). Patients tried to explain their illness in a variety of different ways in order to make sense of their experience. Identification of causes may give patients a sense of predictability and control over their illness and thus help in the process of coping (Roesch & Weiner, 2001). Causal attributions are clinically important for several reasons. Firstly, communication between patients and medical staff may be impeded if patients have different models to those of medical staff, they may lose confidence in the knowledge of health care professionals and fail to follow medical advice and recommendations. Secondly, causal beliefs may stimulate secondary prevention such as lifestyle changes (Weinman et al, 2000) and adherence to medication. Thirdly, some causal attributions may be mistaken or maladaptive, leading to poor psychological adjustment or invalidism (Affleck et al, 1987).

Overall, patients endorsed a range of causes for their heart problem including stress, lifestyle factors and heredity. The most frequently endorsed attributions were stress, smoking, hypertension, heredity, poor diet, lack of exercise, tiredness and overexertion. The strongest attributions were to stress, smoking, high blood pressure and heredity. Similar results have been reported in several previous studies (Affleck et al, 1987; Cameron et al, 2005; De Valle & Norman, 1992; Weinman et al, 2000).

The personal behaviour factor received the highest mean score of the three factors in this study, followed by the heredity factor and mental state factor, showing that patients

held strong beliefs that their heart problem was caused by personal behaviour and their lifestyle (such as poor diet and lack of exercise). This is supported by a previous study which also produced a similar 3 factor solution in which lifestyle factors were found to be important in predicting dietary changes (Weinman et al, 2000). This may also indicate that patients preferred to make attributions to behavioural factors (also categorised as "behavioural self-blame" in some studies) over which they felt had some control and perceived to be modifiable (De Valle & Norman, 1992; Weinman et al, 2000).

Causal attributions have been described as post hoc interpretations or redefinitions of the causes of illness (Sensky, 1997). They may therefore be the products of stereotypic lay beliefs and thus be social constructions. An example of this might be stress, which is commonly reported as a cause of diabetes, rheumatoid arthritis and breast cancer as well as heart disease (Cameron & Moss-Morris, 2004). Patients admitted to modern coronary care unit are provided with extensive information concerning their personal health status and risk factors. Detailed health information is available via the internet, and media advertising and health education campaigns promoting healthy lifestyle factors have helped to raise awareness of personal responsibility for health and fitness. This information often focuses on characteristics that may be common to patients perceived to be at high risk of heart disease, the 'coronary candidate', and usually emphasizes the importance of adopting a healthier lifestyle to reduce both individual vulnerability to cardiac events and the incidence in society as a whole (Davison et al, 1991). Stopping smoking, in particular, has received a great deal of media attention and government funding in recent years. This may make it a more immediate and visible risk factor which is hard for smokers to deny. It is interesting that the lifestyle and

personal behaviour factors that patients believe to be a causes of their heart problems have also been associated with behaviour change in cardiac patients post discharge (De Valle & Norman, 1992; Weinman et al, 2000).

High blood pressure was also a frequently endorsed attribution but this may reflect a more general lay association with stress. It is a commonly held lay assumption that high blood pressure is caused by emotional, rather than physiological stress (Taylor & Ward, 2003; Wilson et al, 2002). Patients' causal beliefs about hypertension and stress in relation to heart disease may therefore be strongly related to one another and hypertension may therefore be considered more of a lifestyle factor related to personal behaviour than a physiological factor by patients and lay people.

In this study, 21.2% of patients (Table 5.1) believed that their heart problem was caused by bad luck. An earlier study reported that 33% of patients attributed their heart problems to bad luck (Gudmundsdottir et al, 2001). The stronger effect in the earlier study may be explained by the way results were combined for patients giving any positive attribution (yes or maybe) to this item rather than only those who only expressed a definite strong belief (yes) as in this study. Patients in the present study may not have felt sufficiently confident to make such a strong statement of belief. Studies by Carney et al (2002) and O'Carroll et al (2001) also showed that attributions to health locus of control (chance) was a predictor of delayed hospital presentation, however the sample sizes in these studies was smaller (N = 62 and N = 72 respectively). The attribution to chance may be an attempt to blame an external uncontrollable factor, and may be linked to denial. It has been argued that patients who believe that their health is under their own control rather than that of powerful others (such as health care

professionals) or chance are more likely to engage in activities which promote health (Seeman & Seeman, 1983). This is supported by evidence in this study showing that "personal behaviour" gained the highest mean score in the factor analysis. The bad luck item was incorporated into this factor but scored negatively (see Table 5.7) such that patients did not believe bad luck to have caused their illness.

Heredity was strongly endorsed by 20.1% of patients in this study (Table 5.1) and this supports evidence from a number of other studies (Astin & Jones, 2004; Cameron et al, 2005; Murphy et al, 2005). Cameron et al (2005) found that 45% of participants believed that their illness was caused by hereditary factors and Murphy et al (2005) reported that family history was the most frequently cited cause among female participants. This is usually seen as being an uncontrollable, external factor which is not modifiable. Attribution to hereditary factors may allow patients to deny responsibility for their heart problem and avoid making changes to their lifestyle. Previous studies have shown that attributions to external factors are associated with poor physical and psychological outcomes (Astin & Jones, 2004; King, 2002; Roesch & Weiner, 2001). Patients therefore appear to make causal attributions to a range of common factors, including those perceived as both controllable and uncontrollable, to explain their illness.

In common with most other studies, in this study stress was frequently cited and strongly believed to be a causal factor (see Table 5.1) (Affleck et al, 1987; De Valle & Norman, 1992; Weinman et al, 2000). Stress is a flexible construct and may be classified as either an internal or external attribution. It can be hard to measure and hard to dispute, and may therefore provide a convenient attribution for individuals in their

search for a cause. It may help to motivate patients to find ways to make their lives less stressful, such as change of job, retirement or adoption of different coping strategies (De Valle & Norman, 1992). If patients believed their heart problem was caused by stress but also believe the stress in their lives is uncontrollable (external), this could lead to a sense of helplessness, making later adaptive life style changes less likely (Affleck et al, 1987). There is some evidence to suggest that patients who have had a heart attack perceive stressful life events in a more emotionally adverse and less controllable way (Affleck et al, 1987; Byrne, 1983) and may also have experienced a higher level and prevalence of various types of stress prior to their cardiac event (Rosengren et al, 2004a).

### 5.7.2: Age and patients' attribution of their heart problem to personal behaviour

In the present study younger patients aged less than 60 years had stronger beliefs that the personal behaviour caused their heart problems than older patients (Table 5.9). This may reflect the gender composition of the study since there were fewer female patients and they tended to be older than men and less likely to believe that personal behaviours and lifestyle factors caused their illness (see section 5.6.2.3). Few previous studies have reported an association between causal attributions and age. One early qualitative study by Meyer (1983) reported differences in causal attributions dependent on age category, but this was due to factors such as fate, overwork and age rather than lifestyle factors, and pre-dates more pro-active modern treatments methods and health education.

### 5.7.3: Gender differences and causal attributions

Results of this study indicated that men had a stronger belief that personal behaviour factors caused their heart problem than women ($p = 0.0019$). This is supported by

previous studies (Astin & Jones, 2004; Bennett et al, 2001; De Valle & Norman, 1992). Murphy et al (2005) found that women were more likely to attribute their heart problems to positive family history than lifestyles factors (except smoking which was the most common modifiable risk factor cited by women), which they argue illustrates that women tend to externalize the cause of their heart disease. Martin et al (2005) also reported that women were significantly less likely than men to attribute their heart attacks to personal behaviours such as dietary factors, lack of exercise, and smoking. They suggest that this may be related to gender based stereotypes, reinforced by the many studies which have used male study populations. Women may not have perceived heart disease to be a significant threat and developed a concept of heart disease as a male disease. Health behaviours may not, therefore, have been perceived to be as important to women as men. No significant association was found in this study between gender and attribution to heredity but the number of female participants was relatively small (N = 50).

### 5.7.4: How accurate are patients' attributions in relation to their personal risk profile?

The relationship between the presence of risk factors and causal attributions was generally positive in this study. For example, smoking attributions were associated with current smoking status, attributions to high blood pressure were associated with previous history of hypertension, attribution to heredity and genetics was associated with family history of heart disease, and attribution to lack of exercise was associated with a sedentary lifestyle.

There were quite large discrepancies, however, in the accuracy of many patients' causal attributions and their clinical risk factors as assessed by medical staff. While it makes sense that patients with a certain risk factor would be more likely to cite this as a causal attribution, in many cases patients did not accurately attribute risk factors that applied to them personally. For example, although 41.1% of hypertensive patients in this study strongly believed hypertension caused their heart disease, compared with 4.4% of normotensive patients, 58.9% of hypertensive patients were less convinced that hypertension was a causal factor.

The accuracy of patients' attributions in relation to their actual risk factor profile has varied widely in previous research across all risk factors. Attribution to hypertension among hypertensive patients has varied from 5% (Murphy et al, 2005) to 15% (Zerwic et al, 1997) to 85% (Cameron et al, 2005), whilst in the present study 41.1% of known hypertensives thought that hypertension was a cause of their heart problem. Also, 33% of patients who had a relative with heart disease believed that heredity was a cause of their own illness. This is compares well with other studies which vary from 24% (Martin et al, 2005) to 54% (Murphy et al, 2005). Just over half (53.4%) of current smokers in the present study strongly believed that smoking caused their heart disease. The degree of concordance regarding attribution of smoking as a causal factor among smokers has again varied widely in previous research, from only 22% (Murphy et al, 2005) to 45% (Martin et al, 2005) to as much as 64% (Zerwic et al, 1997).

It is understandable that patients whose personal risk profiles did not include particular risk factors were less likely to make these attributions. Patients' accuracy, however, in recognising their own risk factors in relation to attributions of their own heart problems,

was quite poor. These discrepancies may be due to a number of factors. There may simply be a lack of knowledge among some patients as to the causes of heart disease. Previous research has shown that over a fifth of patients said that they did not know what caused their heart problems (Martin et al, 2005; Murphy et al, 2005). The lack of concordance in some previous studies may also be a reflection of the study methodology. Studies that have reported low concordance have often used open ended questions that require the patient to make a definite statement of belief, which they may not feel confident enough to do, rather than cued methods. There may also be differences between hospitals in the quality and intensity of information given to patients. Since cardiac rehabilitation seemed to have little effect on causal beliefs in some studies, it may be the result of poor or ineffective communication between medical staff and patients (Murphy et al, 2005).

It is possible that risk factor identification and secondary prevention is pursued more vigorously in men than women, so that women are less aware of the importance of behavioural risk factors as causes of heart disease (Simpson et al, 2004). Other studies, however, have shown a fairly good level of knowledge of the causes of heart disease among the general public but coupled with a poor perception of personal risk (Eaker et al, 1999; van Tiel et al, 1998). The inaccuracy of patients' attributions and perceptions of their own risk factor profile may be due to denial and form part of a coping mechanism. Patients may therefore be knowledgeable about the causes of heart disease but be unable to utilize this information in their personal situation if it does not match their personal schema of their vulnerability to heart disease. For example, patients who have had previous coronary interventions but mistakenly believe they are no longer at

risk (Pattenden et al, 2002), and women who believe heart disease is largely a male problem (Mosca et al, 2000).

In relation specifically to hypertension, some patients may not be aware of the physiological role of hypertension plays as a risk factor for heart disease and associate it with more psychosocial problems such as emotional stress as discussed earlier (Wilson et al, 2002). They may also assume that if their hypertension is controlled by medication then it is no longer a risk factor. French et al (2002) found that people viewed high blood pressure more often as being influenced by other putative causes (such as eating fatty foods, high levels of cholesterol, stress or worry and type of work a person does) rather than exerting a causal influence on these causes. This belief may also apply to other comorbid conditions such as diabetes.

### 5.7.5: Pre-hospital delay and patients' belief that their mental state caused their heart problem.

Results of analyses presented in this thesis are consistent with the hypothesis that patients' beliefs about the cause of their symptoms predict pre-hospital delay. Results show that patients who had strong beliefs that their mental state caused their heart problem had longer delays, so were potentially at greater risk than others (see Table 5.10). The effect is very large – 15% odds change for every point on the 10 point scale. This was not due to an association between their beliefs that mental state caused their heart problem and decision time, but rather to an association that mental state caused their heart problem and home to hospital delay (see Table 5.12). The situation therefore is not that patients were attributing their symptoms to stress and not bothering to seek

help, but rather that their beliefs that their mental state caused their heart problem are associated with what happens to them after they seek help.

There may be several explanations for this. Perhaps emergency staff took patients less seriously if they thought the symptoms were due to their mental state or over exertion. This seems unlikely since almost 95% patients who initially contacted emergency ambulance services for help had a short home to hospital delay. Patients who preferred to call a family member or friend initially may have had prolonged home to hospital delays because this probably entailed some discussion over the best course of action to take. The friend/relative may not have taken their symptoms so seriously if they thought they were due to stress or over tiredness, sought to deny them or recommended inappropriate lay remedies (Dracup et al, 1995; Dracup & Moser, 1997; McKinley et al, 2000).

Patients may who were highly anxious or distressed may not have communicated their problems clearly so that those they asked for help may have misjudged the severity of their symptoms. Patients who contact their GP first may have difficulty getting quick access to their GP and are rarely able to speak to their GP directly. They are often prioritised by non-medical untrained staff, such as receptionists or call centre staff, who act as the gatekeepers to the GP services. This may increase their frustration in seeking help and lead to aggressive or hostile communications, or engender a sense of helplessness, which may produce a negative reaction in the helpers.

Analysis of variance showed that patients who experienced a greater number of different types of symptoms (non-chest pain symptoms such as jaw pain or shoulder pain, or non-pain symptoms such as nausea or shortness of breath) and greater level of

pain had significantly lower scores on believing that their mental state caused their heart problem. Patients who suffered a greater number of symptoms were significantly more likely to have shorter than average pre-hospital delay (Table 3.13) and a short home to hospital delay (Table 3.16). Patients who suffered severe pain and a greater number of symptoms, and who did not report a lot of stress in their lives prior to this may have come to the conclusion that they were having a heart attack more rapidly and responded accordingly. Afterwards, they may have realized that they had survived a major medical crisis and believed that their mental state had little to do with it. The greater number of symptoms may also have made the illness seem more urgent and diagnosis quicker for medical staff. On the other hand, patients who experienced chest pain but no other symptoms, and suffered less intense pain, had higher mean scores in believing that their mental state was a cause of their illness (Table 5.13). These patients may have tried to play down or normalize symptoms such as chest pain by attributing it to anxiety, or panic, or even over tiredness to a family member, friend or GP they have contacted for help, particularly if their pain is of low intensity. This may reduce the level of perceived urgency, make diagnosis more difficult and prolong their home to hospital delay period. It may also encourage a 'wait and see' approach, particularly in relatives/friends/work colleagues or even the GP.

Patients who reported that they experienced at least moderate stress due to illness, fatigue or depression also had significantly stronger beliefs that their heart problem was caused by mental state (Table 5.13) but this was not associated with home to hospital delay. Previous studies have found that patients who have suffered an MI experienced more stress in their lives than other people (Rosengren et al, 2004a) and they may be more aware and more sensitive to their mental state. There is growing evidence to

suggest that negative emotional states (such as depression, anxiety, anger and mental stress) and social isolation are risk factors or triggers for cardiac events (Berkman et al, 1992; Carney et al, 2001; Frasure-Smith et al, 1995b; Strike et al, submitted a). This may help to explain why patients who believed that their mental state caused their symptoms were more likely to have longer total pre-hospital delays since high levels of background stress and anxiety may have acted as a distraction from recognising symptoms and seeking help. There is also some evidence to suggest that patients who are depressed or anxious are more likely than other patients to endorse negative emotions as causes of their heart disease (Day et al, 2005) and reporting bias due to negative affect and depression could influence the ratings of perceived causes (Watson & Pennebaker, 1989). This may have increased patients' ratings of pain and distress, although it is unlikely to have affected clinical assessment of their symptoms.

### 5.7.6: Factors associated with mental state

Symptoms which start as non-specific fatigue, illness or feelings of stress and gradually increase may also be more difficult for patients to acknowledge and lead to a normalizing of the symptoms until they suddenly change or become severe. It is interesting that patients with lower mean scores on the cardiac denial of impact scale also had stronger belief that their mental state caused their heart problem, although this was not associated with home to hospital delay. Patients with lower levels of denial may perhaps be more aware of their mental state and more likely to acknowledge the impact it might have in causing their ACS. Previous research by O'Carroll et al (2001) also showed that shorter pre-hospital delay was predicted by lower scores on denial, although in another study no significant association was found between denial and delay (Carney et al, 2002).

Patients with a larger social network of 4 or more social contacts also had a stronger belief that their mental state was a cause of their symptoms (see Table 5.13). This contrasts somewhat with previous research which has shown larger social networks and greater social support to be protective against negative mental states such as depression (Barefoot et al, 2000; Frasure-Smith et al, 2000). Social relationships can be complicated, however, and these results may indicate that the larger the social network the more potential for conflict or stress in within the relationships. The social network index indicates the number of social contacts a patient might have but not the quality of these contacts, so that a large number social contacts which included stressful relationships may actually produce a negative effect on mental or emotional well-being (Ell, 1996). This may prolong home to hospital delay if a family member or friend is the initial contact after symptom onset for reasons outlined above. Alternatively, patients with a large social network may also have greater awareness of their own emotional state than socially isolated individual. Stress is, in general, rated highly as a cause of heart problems and chest pain among lay people, and patients with a large social network may have more exposure to these lay beliefs with the result that they give stress a stronger emphasis on their causal beliefs.

### 5.7.7 Did patients misinterpret the question about causal attributions ?

Previous studies have found some evidence that participants interpret research questions in different ways and this may affect responses (French et al, 2001; Murphy et al, 2005). It is possible that patients in the present study may have misinterpreted the question about causal attributions. The question as stated in the questionnaire (Appendix 7) was "what do you think caused your heart problem ?", and the intention was to discern which factors patients believed caused their heart problem from a list of 16 possible

causes. Some patients may have interpreted the wording in a more general way about what caused their heart disease, while others may have answered in terms of acute triggers of their cardiac event. This may have affected patients' responses and may reflect patients' long term beliefs about heart problems in general rather than their beliefs about the immediate cause of their heart problem. It can be seen from Table 5.1 that many of the perceived causes are indeed long-term determinants like smoking, heredity and high blood pressure. Other factors are more ambiguous (stress and over exertion) and could be operating either in the long-term or acutely. There is no reason to suppose that general beliefs in long-term lifestyle influences or hereditary factors would predict delays in seeking medical help following symptom onset before admission or patient decision times. Nevertheless, the association between the mental state factor and delay is interesting. Mental states such as anger and depression can operate as a trigger of acute cardiac events (Strike et al, submitted a; Strike et al, submitted b), so this relationship is potentially important.

## 5.8: Limitations to the investigation of causal attributions

### 5.8.1: Timing

Patients causal attributions were assessed within the first few days following admission and may thus be strongly influenced by information given to patients by nursing and medical staff. Patients are often given a lot of information whilst on the coronary care unit which will include information about the causes of ACS. Efforts were made to interview patients early in their treatment in order to elicit their own causal beliefs but it is possible that they had already been influenced by information given during the initial stages of their hospital admission.

### 5.8.2: Measures

#### *5.8.2.1: Type of question*

There may have been some limitations associated with the method of scoring used. A cued questionnaire was used which offered a list of possible causes. This may have produced a higher rate of responses than would have been the case if open ended questions had been used (Gudmundsdottir et al, 2001). Attributions have been found to be less likely to be associated with poorer outcomes if they were elicited using open-ended questions than if other methods were used such as rating checklists (French et al, 2001). The causal beliefs questionnaire in this analyses offered 3 possible answers to each item ("no", "maybe" or "yes") but only definite endorsements (i.e. "yes") were used to generate positive attribution scores. Since some patients may not have been confident enough about their views to make a definite statement about cause, the results reported here may be weaker than results of analyses which combined both possible and definite endorsements (i.e. "maybe" and "yes").

#### *5.8.2.2: Focus of the question*

Patients may have misinterpreted the question about causal attributions by interpreting the question to be about heart disease in general or about possible triggers of chest pain/heart disease, rather than to be about the cause of their own ACS.

### 5.8.3: Individual bias

There is mixed evidence as to the accuracy of self report measures of health behaviours (Cappuccio et al, 2003; McKeown et al, 2001; Rennie & Wareham, 1998). Patients may be subject to interviewer bias whereby compliance is over estimated in order to please the researcher.

# Chapter 6: Literature review of adherence, psychological adjustment and quality of life following ACS in relation to causal beliefs and emotional state

## 6.0: Introduction

The second clinical problem that this thesis will investigate is the problem of poor adherence to medical recommendations, poor psychological adjustment and quality of life following discharge from hospital, and the role played by patients' causal beliefs. In this chapter I will discuss the importance of adherence and risk factor management, and review previous published literature which has investigated cardiac rehabilitation attendance and adherence to medication in relation to patients' causal attributions. I will also review literature which has investigated psychosocial factors such as the importance of social support and the impact of depression and anxiety on adherence. Lastly, I will discuss factors which predict quality of life and associations with patients' causal attributions.

## 6.1: Aims of secondary prevention

There is considerable evidence to show that secondary prevention programmes to reduce cardiovascular disease risk factors have a favourable impact on morbidity and mortality (Wood et al, 1998). Findings from the Framingham Study showed that a previous medical history of cardiovascular disease increases the relative risk of subsequent premature cardiovascular morbidity and mortality by 5 to 7 times (Kannel et al, 1979). Among survivors of a first AMI, the rate of subsequent AMI is increased 3 to

6 times, and the risk of any cardiovascular disease event may be as high as 80% (Schlant et al, 1982).

The aim of secondary prevention for patients with coronary heart disease is to reduce their risks of having further cardiovascular events and increase chances of survival, and to improve their quality of life. A number of recommendations have been put forward defining goals with regard to lifestyle, risk factors and therapy. These include quitting smoking, making healthy food choices and taking regular physical exercise; maintaining a body mass index of less than 25 kg/m2 , blood pressure of less than 140/90, total cholesterol level of under 5.0 mmol/L, and appropriate use of prophylactic drugs such as aspirin, beta-blockers and angiotensin converting enzyme (ACE) inhibitors, and anticoagulants (Wood et al, 1998). Risk factor management, effective medication and cardiac rehabilitation have also been highlighted as health care priorities by the UK government and all of these recommendations were included in the National Health Service Framework for Heart Disease (Department of Health, 2004).

## 6.2: Definition of adherence

Adherence to medical advice may be defined as taking medications as prescribed, attending cardiac rehabilitation classes if recommended and modifying lifestyle to incorporate healthy behaviours (quitting smoking, regular physical activity, control of body weight and eating a healthy diet etc). When patients are non-compliant, they do not take their medications correctly, forget or refuse to follow a diet, do not engage in prescribed exercise, cancel or do not attend appointments, and persist in lifestyles that endanger their health (DiMatteo et al, 2000). Patients may be non-adherent for a variety of reasons such as misunderstanding or complexity of advice given, purposefully

ignoring advice or being given inappropriate advice, or forgetting. Clearly, the extent to which patients are in concordance with medical advice has an important effect on how effective their treatment is. Since patients are at increased risk of further cardiac events following an ACS, secondary prevention is an important part of their treatment. The issue of adherence is therefore a particularly salient issue among these patients.

## 6.3: Risk factor management

Modifiable risk factors such as smoking, body weight, blood cholesterol level and blood pressure are often poorly controlled among survivors of ACS. A large study by Qureshi et al (2001) examined secondary prevention among 1252 patients who survived MI, stroke or both in the USA. They reported that hypertension was adequately controlled in only 38 % of known hypertensives, and despite having been seen by a health care professional within the past 6 months, an additional 11% were only diagnosed during the study. Only 40 % of diabetic participants had adequately controlled blood glucose control, just under half (49%) of patients with known hypercholesterolaemia were considered to be adequately controlled. They also reported that 43% of patients were overweight and 18% of survivors continued to smoke.

A study by Wood (2001) compared the results of 2 large surveys of modifiable risk factors among patients from 9 European countries with CHD (Euroaspire I and II). The study gave a mixed picture of the implementation of secondary preventions from the time of the first study in 1995-6 to the time of the second in 1999-2000. The prevalence of smoking remained unchanged at 19.4% vs 20.8%. Obesity increased substantially from 25.3% to 32.8%, and the prevalence of diabetes also increased. The proportion of patients with hypertension remained unchanged at 55.4% vs 53.9%. While the

prevalence of hypercholesterolemia had decreased substantially from 86.2% to 58.8%, over half of these cardiac patients had serum cholesterol levels above current recommendations. Aspirin and other anti-platelet therapy was widely used in both surveys (83.9%), and use of beta-blockers, ACE- inhibitors and lipid lowering drugs increased although there were large variations between countries.

These results revealed a continuing high prevalence of modifiable risk factors among patients with CHD. A review of 12 randomised trials of multi-disciplinary disease management programmes for patients with CHD reported a positive impact on risk factor profiles and prescription of recommended drugs (McAlister et al, 2001). Their findings showed a significant reduction in admissions to hospital and a trend towards improved symptom scores, exercise tolerance, and quality of life. The Government had set comprehensive guidelines and targets in the National Service Framework for Heart Disease (National Health Service, 2000) for an effective multi-disciplinary approach to the management of secondary prevention in CHD patients in an attempt to improve morbidity and survival.

## 6.4: Cardiac rehabilitation programmes and predictors of attendance

There is strong evidence to support the beneficial effects for patients of attending a cardiac rehabilitation programme (Dinnes et al, 1999). Meta-analyses have suggested a reduction in total mortality of at least 20% among those who attend rehabilitation programmes (Thompson & Lewin, 2000). Cardiac rehabilitation programmes can bring about substantial improvements in exercise tolerance, symptoms, blood lipid levels, psychological well being and stress, reduction in smoking (Dinnes et al, 1999).

Only a small proportion of patients post AMI, are offered or take up cardiac rehabilitation and this predominately tends to be middle-aged men with a diagnosis of uncomplicated AMI. Lane et al (2001b) reported that only 41% of survivors of AMI attended rehabilitation classes. Non-attenders were more likely to live in more deprived areas, were less likely to be in paid employment, lived alone and were more likely to be female. They registered more symptoms of depression and anxiety and did less exercise. They were also more likely to have had a more severe AMI, have a previous history of AMI, suffer from angina pectoris and were less likely to have received thrombolytic therapy. Thus, ironically, patients who were at increased risk of further cardiac events were both less likely to be invited to participate in rehabilitation programmes and less likely to attend.

When questioned, patients gave various reasons for non-attendance including not wishing to attend, the presence of co-morbid health problems, returning to work, being the main carer for a significant other, and living too far away (Lane et al, 2001b). Cooper et al (1999) also reported that non-attenders were likely to be older, had a lower level of income and greater level of deprivation, less likely to be employed and to deny the severity of their illness. Melville et al (1999) also reported that greater social deprivation, previous history of AMI or revascularisation and not being given an outpatient appointment predicted non-attendance, whilst factors which predicted attendance included younger age, male gender, prescription of diuretics, admission to a coronary care unit, diagnosis of AMI and receiving thrombolysis. Other studies also support evidence that older patients are less likely to participate in rehabilitation (Sotile & Miller, 1998) and women are more likely to drop out of cardiac rehabilitation programmes than men (McGee & Horgan, 1992).

Social support has been reported as a predictor of adherence to medical advice and attendance at rehabilitation programmes (Krantz & McCeney, 2002). A Canadian study of patients following AMI or coronary artery bypass graft surgery (CABG's) reported that women had significantly less social support than male counterparts, and elderly patients had significantly less social support than younger patients at 6 months follow up (King et al, 2001). Patients who attended a rehabilitation programme had significantly higher scores for health maintenance self efficacy and behaviour performance and significantly greater improvement in health maintenance over the follow-up period than non-attenders. Factors such as low level of education, deprivation, living in a low socio-economic neighbourhood increases the probability that an individual will encounter stressors without having support systems sufficient to enable them to cope effectively (Krantz & McCeney, 2002). These patients are therefore at increased risk of failure to adhere to treatment programmes.

## 6.5: Patients' beliefs and attendance at cardiac rehabilitation

Studies which have investigated associations between illness perceptions and adherence to health behaviours have shown inconsistent findings. The components of illness perceptions that predict adherence have varied between studies (described below) and some studies have found no associations at all between illness beliefs and adherence behaviours, such as attendance at cardiac rehabilitation programmes, lifestyle changes or adherence to prescribed medication. Five studies reviewed below have examined the associations between illness perceptions and attendance at cardiac rehabilitation programmes, two investigated more general aspects of healthy lifestyle behaviours and two focussed on adherence to medication.

Petrie et al (1996) investigated the role of patients illness perceptions in a prospective study based on the self regulation model of attendance at a cardiac rehabilitation programme, return to work, disability and sexual dysfunction in 143 patients who had suffered their first MI in New Zealand. Patients completed a research questionnaire while they were in hospital, which focussed on 4 illness perceptions (identity, timeline, consequences, and cure/control). Causal beliefs were not specifically investigated in this study. Attendance at cardiac rehabilitation was found to be significantly related to a stronger belief during admission that the illness could be cured or controlled. Non-attenders showed a trend to believe that their myocardial infarction held less serious consequences for them in the future and to be less distressed by their infarction. A strong illness identity was associated with greater sexual dysfunction both 3 months and 6 months later. There was also a non-significant trend for non-attenders to be less generally distressed than attenders. This study showed that illness perceptions were important factors involved in various aspects of recovery.

Cooper et al (1999) also investigated the role of illness beliefs held by 152 patients who had suffered MI or CABG's during hospitalisation in a prospective study and found that only 40% of participants attended cardiac rehabilitation classes. Participants completed a questionnaire containing the measures based on the self regulation model just before hospital discharge. Patients who believed that their heart problems could be cured or controlled, and patients who attributed the cause of their heart problems to lifestyle factors were significantly more likely to attend rehabilitation.

A cross sectional study by Whitmarsh et al (2003) examined the influence of illness beliefs on attendance at a cardiac rehabilitation programmes among 93 patients invited to attend. Participants were asked to complete a range of self report measures (the Illness Perception Questionnaire, the Hospital and Anxiety and Depression Scale, and the Coping Orientation to Problems Experienced questionnaire) several weeks after hospital discharge, shortly before the start date of the programme. Results showed that attenders perceived a greater number of symptoms and consequences than poor/non-attenders. Patients with a stronger illness identity, and stronger beliefs that the illness was controllable or curable were therefore more likely to attend the rehabilitation programme. Attenders also experienced significantly greater distress than non-attenders, evidenced by higher mean scores for both anxiety and depression, and tended to use problem focussed and emotion focussed coping more frequently. Patients who continued to experience a greater degree of distress several weeks after discharge and prior to commencing rehabilitation were more likely to attend. Attenders did not differ from non-attenders regarding causal beliefs (including stress) with the exception of causal attribution to a germ/virus. Non-attenders were significantly more likely to believe that their illness was caused by a germ or virus than attenders.

A prospective study by French et al (2005b) of 194 MI patients investigated the extent to which illness perceptions predict attendance at cardiac rehabilitation and quality of life following MI. They found no significant associations between illness perceptions (including causal attributions) and cardiac rehabilitation attendance, or with depression or anxiety. Illness perceptions were, however, predictive of quality of life after 6 months, with beliefs about the consequences of the illness being most strongly related to

emotional, physical and social quality of life, even after controlling for anxiety and depression.

A qualitative study using an interpretative phenomenological approach by Cooper et al (2005) found that patients' beliefs may act as a barrier to attendance. Thirteen MI patients were interviewed after discharge from hospital but before attendance at a cardiac rehabilitation programme. Five key themes were identified which may influence attendance at cardiac rehabilitation including content of the course (particularly concerns about the exercise content and role of physical activity in recovery), perceived benefits, explicit barriers to attendance (such as transport, taking time off work, social interaction), cardiac knowledge (particularly causal attributions for ACS), and the nature of CHD. Patients commonly attributed stress and worry as causes of their heart problem, and those who found it difficult to perceive a causal explanation were less likely to see how attendance at cardiac rehabilitation would benefit them.

A later randomized controlled trial by Petrie et al (2002) used an intervention aimed at changing illness perceptions in MI patients. The intervention consisted of 3 sessions delivered by a psychologist whilst the patient was in hospital. The first session explored patients beliefs about the cause of the AMI, focussing on addressing the common misconception that stress is the only cause of MI and broadening the patients' causal model to include lifestyle factors. The second session focussed on developing a plan for minimizing future risks and increasing patients' beliefs about controllability. The third session reviewed this plan and discussed symptoms of recovery. Although there was no difference in attendance at rehabilitation classes between the two groups, patients in the intervention group felt better prepared to leave hospital and returned to work faster than

the control group, they also reported a significantly lower rate of angina symptoms after 3 months than the control group. This study showed that patients' illness perceptions can be modified and that modification of patients' beliefs about the cause of their MI may have an impact on their quality of life.

Weinman et al (2000) investigated the effects of causal attributions and subsequent lifestyles changes in prospective study of 155 first time MI patients and their spouses. They reported that patients who believed that their MI was caused by unhealthy lifestyle behaviours (assessed during hospital admission) were more likely to have made dietary changes at 6 months follow up, and spouse attributions to poor health habits (assessed 12 weeks and 6 months following their partner's MI) were associated with improvements in patients level of exercise. They reported that patients' and their spouse's beliefs about the causes of their MI affected their adherence to healthy lifestyle changes. A recent re-analyses of these data, however, found that once pre-MI behaviour had been controlled for, there was no consistent relationship between causal attributions and subsequent behaviour change, but rather causal attributions were associated with pre-MI behaviour (French et al, 2005a). French et al argued that there is little published evidence that patient or spouse attributions influence behaviour change, although different causal attributions play an important role in adherence and may predict outcome. They suggested that attributions may be associated with outcomes either because blaming attributions yields negative mood or attributions to lifestyle protect against negative mood.

A cross sectional study by Byrne et al (2005), based on the self regulatory model, found a weak association between illness perceptions and longterm CHD patients' secondary

preventive behaviour, and a small to medium relationship between medication beliefs and adherence to medication. The most commonly endorsed illness attributions were stress, and heredity, followed by lifestyle. A stronger belief that personal behaviour caused the heart problem was related to a higher alcohol intake, but not to other health behaviours such as smoking, diet and exercise. There were also conflicting findings concerning emotional representations of health related behaviour reflecting the emotional impact of the illness however; lower levels of emotional representations were related to more frequent exercising but higher alcohol consumption. Patients who viewed their illness in a more catastrophic and highly emotional way which emphasized the negative aspects such as the seriousness of the illness and a strong illness identity, were more likely to have a reduced functional capacity and poor psychological adjustment. A stronger belief in the necessity of medication and fewer concerns about medication was predictive of higher adherence to medication. In this study, the interval between the cardiac event and measurement of illness perceptions was quite long (average of 7 years), participants also reported a low level of illness identity. The authors suggest that since patients' perceptions of the symptoms and health threat was low, it was not surprising that the relationship between illness beliefs and behaviour was weak.

Beliefs about causal attributions have not been specifically investigated in relation to medication adherence, and studies investigating adherence to medication have used a slightly different model to assess patients' beliefs. A cross sectional study by Horne and Weinman (2002) investigated asthma patients' beliefs about medicines and their role in adherence, and found that non-adherent behaviours were associated with patients' doubts about the necessity of medication, and concerns about its potential

adverse effects and with more negative perceived consequences of illness. Illness perceptions and treatment beliefs were both substantial independent predictors of adherence in this study. A negative correlation was found between perceived consequences of illness and adherence to medication. Patients who perceived more negative consequences of their illness, had stronger beliefs in the necessity of their medications. This supported evidence from a previous study by Horne and Weinman (1999) of patients from 4 different illness groups (including cardiac, asthma, renal and oncology) that showed that many patients engage in an implicit cost-benefit analysis in relation to medication adherence where beliefs about the necessity of medication are weighed against concerns about its potential adverse effects.

## 6.6: Patients' beliefs and adherence to medication

Adherence to prescribed medication is very important for patients diagnosed with ACS since evidence from a number of large drug trials have shown medications such as beta-blockers, cholesterol lowering medication, aspirin and ACE inhibitors to significantly reduce mortality and morbidity rates (Antithrombotic Trialists Collaboration, 2002; Chalmers, 2004; Sacks et al, 1996; Scandinavian Simvastatin Survival Study, 1994). Clearly, patients cannot receive the full benefits of medication if they do not adhere to prescribed therapies.

Adherence to medication has been measured in a variety of different ways. Direct measurements of concentrations of a drug, its metabolite or a biologic marker in blood or urine is expensive, susceptible to distortion and impractical in many situations, although it is commonly used in particular conditions such as assessment of adherence to antiepileptic drugs. There are problems with all methods of measurement of

medication adherence. For example, performing pill counts (counting the number of pills remaining in the bottle) does not take into account patients discarding of pills or switching bottles to give the impression of compliance; assessing clinical response may be confounded by many other factors other than medication adherence; ascertaining rates of refilling prescriptions can be used to corroborate patients self report but does not prove the medication has been used correctly; using electronic medication monitors can only measure whether a container has been opened or activated not whether the medication has actually been ingested; self report measures such as patient questioning or the use of questionnaires may be subject to patient report bias whereby the patient wants to please the clinician/researcher and over-estimates compliance. As suggested by Stone (1979) however, the patient knows best what s/he has been doing and if the atmosphere created is supportive and non-punitive, will usually tell the truth about the problems they experience with adhering to treatment regimes. Each method has advantages and disadvantages and there is therefore currently no gold standard.

Adherence to a short-term use of medication is generally estimated to be quite poor, approximately 20% to 30% of patients are non-adherent to prescriptions for a 10 day course of antibiotics (DiMatteo et al, 1992). For long-term medication, when there is no end in sight, it is estimated that about 50% of patients fail to follow the prescribed regime (Benner et al, 2002). Six general patterns of taking medication have been reported among patients with chronic illness who continue to take their medications (Osterberg & Blaschke, 2005). Approximately one sixth come close to perfect adherence; one sixth take nearly all doses but with some timing irregularity; one sixth miss an occasional single day's dose; one sixth take drugs holidays three to four times a year, with occasional omission of doses; one sixth have a drugs holiday monthly or

more often, with frequent omissions of doses; and one sixth take few or no doses while giving the impression of good adherence.

Predictors of poor adherence include a number of different factors, such as the presence of psychological problems (particularly depression) or cognitive impairment, treatment of an asymptomatic disease, inadequate follow up or discharge planning, drug side effects, patients' lack of belief in the benefit of medication or lack of insight into the disease, a poor patient-doctor relationship, complexity of the drug regime, and cost of medication (if applicable) (Osterberg & Blaschke, 2005).

In a review of 21 studies investigating the impact of medication adherence on morbidity and mortality among patients with or at risk for coronary artery disease and congestive heart failure, greater adherence to medication was found to have a significant impact on lower hospital readmission rates and improved outcomes (McDermott et al, 1997). The improvement in outcomes, however, was not confined only to participants who were taking the prescribed medication but were also reported in participants who were taking placebo medications in placebo-controlled trials. This suggests that the adherent behaviour itself may be a marker of better prognosis or that is somehow confers a protective effect on patients with coronary heart disease. Similar results have been found in other studies. The Coronary Drug Project Research Group (1980) showed that 5 year mortality was lower in participants with a minimum rate of adherence of 80% rate regardless of whether they took the cholesterol lowering medication or the placebo. Likewise, the Beta-blocker Heart Attack trial investigated adherence among 2176 post AMI patients and found that overall, poor adherers (who took 75% or less of prescribed medication) were 2.6 times more likely than good adherers to die within a year of

follow-up (Horwitz et al, 1990) The authors suggest that conscientious adherence to a medical regime may be one manifestation of a favourable psychological profile, reflecting an individual's ability to make lifestyle adjustments that would limit disease progression.

A recent study investigated the effect of self reported cultural background on beliefs about medicines among 500 UK undergraduate students who identified themselves as being from either an Asian or European background (Horne et al, 2004). Findings showed an association between cultural background and beliefs about the benefits and dangers of medicines. Participants who had an Asian cultural background were significantly more likely to perceive medicines as being intrinsically harmful, addictive substances that should be avoided. This clearly has a potential impact on adherence to medication, and emphasizes the need for clear communication between patients and health care practitioners which takes into account possible cultural differences in the perception of medicines and their importance in treatment regimes.

## 6.7: The association between social support and adherence

Social isolation and social support may be important factors in successful recovery from a cardiac event and adherence to treatment recommendations. The effect of social support on health may be to modify patients' appraisal of the stressfulness of a situation and their perception of stress (Aalto et al, 2005). Studies have found that life stress and social isolation along with depression are related to morbidity and mortality following diagnosis of ACS (Barefoot et al, 2000). Patients classified as being socially isolated and having high life stress had 4 times the risk of death than patients with lower levels of stress and isolation (Horwitz et al, 1990). Social isolation and withdrawal from those

who would otherwise provide emotional support often accompanies depression which then increases the likelihood of non-adherence.

A number of studies have highlighted the importance of family support and the social network in patient's attempts to comply with treatments. A study by Brummett et al (2005) investigating perceived social support as a predictor of mortality in coronary patients found that patients who were active at baseline and follow up had consistently higher social support scores than patients who were sedentary at baseline. Positive support may therefore help patients to maintain an exercise programme, or conversely discordant relationships may deter adherence to exercise regimes. Individuals with positive perceptions of their social support were less likely to be sedentary, and smoking was also negatively associated with social support. Social support has also been found to be positively associated with weight loss in post coronary patients (Conn et al, 1992) and behaviours such as quitting smoking, having a blood pressure and cholesterol check, physical activity and eating fruit and vegetables (Ford et al, 2000). Evidence from some other studies that have examined the association between social support and smoking however have produced negative or opposite findings (Conn et al, 1992; Ford et al, 2000).

## 6.8: The impact of depression and anxiety on adherence

Although not all clinical studies agree, symptoms of depressed mood in the days following admission for acute coronary syndromes (ACS) have been found to predict future morbidity and mortality (Lesperance et al, 2000; Rosengren et al, 2004a; Rumsfeld & Ho, 2005). One study reported that depression was associated with a 3-4 fold increase in cardiac mortality over the first 18 months following an MI (Frasure-

Smith et al, 1995a). Approximately 17% of patients with AMI experience a major depressive disorder and almost half of patients (42%) with minor depressive symptoms go on to develop major depression (Hance et al, 1996). Studies have also found evidence to suggest a dose-response relationship between in-hospital depression and post AMI mortality beginning below the cut off point of $\geq 10$ suggested by Beck and Steer (1993) for defining even mild symptoms (Bush et al, 2001; Lesperance et al, 2002). Not all studies support these findings however, and results from the first National Health and Nutrition Examination Survey reported mixed findings (Ferketich et al, 2000). Whilst depression was associated with an increased risk of CHD incidence among men and women, it was associated with CHD mortality in men only. Findings from an intervention study also showed that although a cognitive intervention was effective in reducing depression and improved social support, it had no affect on mortality and morbidity after 6 months (ENRICHD Investigators, 2003).

Although the evidence is not entirely consistent, depression has been linked to non-adherence to medical treatment recommendations in a number of studies. In comparison with non-depressed patients, DiMatteo et al (2000) reported that the odds were 3 times greater that depressed patients will be non-adherent to treatment. Patients with major depression were found to have significantly lower adherence in taking medication as prescribed. Carney et al (1995) found that elderly patients suffering from depression and coronary artery disease were less likely to adhere to prescribed aspirin therapy when compared with non-depressed patients. Ziegelstein et al (2000) reported that patients who had symptoms of mild to moderate depression in the initial 5 day period post AMI had significantly lower adherence in following a low-fat and low cholesterol diet, exercising regularly, reducing stress and were less able to increase their

social support at 4 month follow up. Guiry et al (1987) found that depression, poor motivation and poor body image measured in hospital among patients admitted for ACS were inversely related to smoking cessation and increasing exercise activity one year later.

According to DiMatteo et al (2000) positive expectations and beliefs in the benefits and efficacy of treatment are essential to patient adherence. Since depression often incorporates an appreciable degree of hopelessness, adherence might prove difficult for a patient who has little optimism that any action is worthwhile. DiMatteo et al suggested that a feedback loop may operate whereby depression causes non-compliance with medical regime and non-compliance then exacerbates depression. It may also be possible that a third variable, such as poor health status, is responsible for both. Depression may also be associated with reductions in cognitive functioning which are essential to remembering and complying with treatment recommendations (Wing et al, 2002). It is possible that depression may provide a potentially useful marker for non-adherence and vice- versa. As both factors carry increased mortality and morbidity rates among patients suffering from ACS early screening and treatment for depression could potentially improve these outcomes.

Conversely, a study investigating cardiac rehabilitation attendance (described above) by Whitmarsh et al (2003) reported that attenders had higher scores on both anxiety and depression than non-attenders. Petrie et al (1996) also reported a trend for non-attenders to be less generally distressed than attenders, although this was not significant. On the other hand, French et al (2005b) found no relationship between anxiety and depression and cardiac rehabilitation attendance in a study among 194 MI patients. The

relationship between emotional distress and adherence to medical recommendations is therefore not clear.

Nevertheless, recognition and treatment of depression may be important not only because of its possible impact on mortality and morbidity, but also because of its negative impact on quality of life. Anxiety and depression have been associated with diminished health status and substantially lower health related quality of life persisting over time (Sherbourne et al, 1996). Depression has been associated with failure to return to work, and poor rehabilitation (Lewin, 1999).

In contrast to depression, the association between anxiety and adherence has been reported as minimal. In a meta-analysis of 13 studies, the difference in risk of non-compliance between anxious and non-anxious patients was only 4% (DiMatteo et al, 2000). The relationship between anxiety and depression is unclear. Anxiety can vary from panic, which may have no direct effect on adherence to obsessive compulsive disorder and generalized anxiety about health which may even improve adherence.

Anxiety is exceptionally common in patients with ACS, with a hospital incidence of about 50% among coronary care patients (Januzzi, Jr. et al, 2000; Moser & Dracup, 1996). Martin and Thompson (2000) reported high levels of both anxiety and depression within 24 hours of admission to the coronary care unit among patients following acute MI. A number studies have suggested that anxiety influences both acute and chronic outcomes following ACS. Studies have reported an increase in ischemic complications following AMI resulting from anxiety from 2.5 – 5 times that of non-anxious patients (Frasure-Smith et al, 1997; Moser & Dracup, 1996). Reviews of

several clinical and experimental studies have shown that mental stress (acute, sub-acute or chronic) increases the risk of ischemia, MI or death in patients with established ischemic heart disease (Januzzi, Jr. et al, 2000; Kubzansky & Kawachi, 2000).

## 6.9: Health related quality of life following ACS

Health related quality of life represents the effect of an illness and its treatment as perceived by the patient. There are few studies which have investigated the association between causal attributions and health related quality of life specifically, but it is important to measure quality of life because it has been shown to influence mortality and morbidity in cardiac patients. A study of 2480 patients undergoing coronary artery bypass graft surgery (CABG's) by Rumsfeld et al (1999) reported that patients with poorer self perceived physical health measured by the physical component of the SF36 (relating to physical health such as being able to dress, walking, and activity level) had greater 6 month mortality.

### 6.9.1: Predictors of quality of life following ACS

Measurement of self perceived health status can be useful in assessing the broad impact of a disease on patients and the effectiveness of interventions. A qualitative study of 31 patients interviewed at home following an MI by Roebuck et al (2001) reported that the area of life patients were most concerned about was their physical symptoms and their effect on every day life. Symptoms such as breathlessness, particularly at night, were reported as more distressing than chest pain. Patients also reported that their inability to perform basic activities of daily living such as shopping, or gardening were as distressing as the symptoms themselves. The association between causal attributions and quality of life was not investigated in this study but other issues were identified as

having a major impact on health related quality of life and well being. These included fear of both making symptoms worse and of having another heart attack which undermined the patients self confidence and often resulted in strained family and social relations. Fear of resuming activities of daily life resulted in reduced life satisfaction and well-being (Roebuck et al, 2001). Over protection by friends, family and work colleagues can result in the patient becoming a 'cardiac invalid' which can lead to a decline in fitness (Petrie & Weinman, 1997) and worsening angina and lead to further decline in quality of life (Lewin, 1997).

Brown et al (1999) investigated the impact of MI on quality of life in 476 patients 4 years after their MI compared with a normal population using the SF-36. This study investigated the overall impact of MI on quality of life rather than the association of causal attributions specifically, but this study illustrates the importance of assessing quality of life as an outcome, as well as mortality and morbidity. Quality of life was significantly poorer among participants under the age of 65 years, who had suffered an MI in all domains but particularly those with a physical element. Smaller but significant differences were also found in the domains of emotion and mental health. Older patients aged 65 – 74 years were comparable with the normative populations except for a slightly lower but significant domain score for physical functioning. Overall, quality of life was impaired for patients who were unfit for work, those with anxiety or sleep problems, the presence of coexistent lung disease and those with angina and dyspnoea.

There is extensive evidence that depression and anxiety predict poor quality of life following MI (Brown et al, 1999; Rumsfeld et al, 2003; Ruo et al, 2003). For example,

a study by Lane et al (2001a) reported that quality of life 12 months after an MI in was predicted by anxiety and depression among 288 AMI patients. Depression was measured using the Beck Depression Inventory (detailed in chapter 7) and anxiety was measured using the State-Trait Anxiety Inventory (which consists of 2 self report scales which assess both state and trait anxiety). Quality of life was measured using Dartmouth COOP chart system which consists of 9 charts that assess physical, social, and role functioning, emotional status, overall health, perceived pain, change in health, social support and perceived quality of life over the previous 4 weeks. Causal attributions were not examined in this study. Their findings showed that poorer quality of life at 12 months was predicted by greater baseline level of depression, greater severity of infarction, living alone and state anxiety. These findings are also supported by evidence from a study by Mayou et al (2000) which reported that greater baseline levels of anxiety and depression among 344 MI patients within the first 72 hours after hospital admission predicted poorer quality of life after one year. Nevertheless, there is some doubt about whether the association is independent of pre-existing illness and clinical characteristics. Quality of life following MI is associated with comorbidities such as diabetes, medical history including previous MI, and is improved by management with PTCA (Kim et al, 2005; Ruo et al, 2003). It is also strongly associated with ongoing angina and chest pain (Rumsfeld et al, 2003). Not all these factors have been well controlled in studies of anxiety and depression.

A longitudinal Swedish study by Brink et al (2002) investigated quality of life and coping strategies in 114 first MI patients after 5 months using the SF-36. Compared with normative data of people who had not had an MI, participants had poorer quality of life after 5 months measured both by the physical and mental health summary

components of the SF-36. This was particularly marked in the areas of limitations due to physical problems (problems with work and other daily activities due to physical problems) and limitations due to emotional problems (mental distress). Women reported significantly poorer quality of health than men. They experienced significantly poorer physical health and bodily pain. There were fewer women in this study however, and they were significantly older than men and this may partially explain the gender difference. This finding has also been supported by other studies (Wiklund et al, 1993). Women also reported greater problems in social functioning, and the authors suggest that women may be more concerned than men about social activities such as caring for other family members and relationships with friends. Causal attributions were not examined specifically in this study. Depression and anxiety measured during the first week in hospital (just before discharge) using the Hospital Anxiety and Depression Scale (HADS) (detailed in chapter 7) did not predict poor quality of life 5 months later. It is possible that there are differences in mental distress related particularly to hospital admission or discharge, so that the timing of the administration of this measure may affect the results.

### 6.9.2: Associations between quality of life and adherence

A cross sectional study investigating the relationship between adherence and quality of life in patients following an MI reported inconsistent results depending on the follow up period. Schron et al (1996) found that poorer physical health quality of life and better mental health quality of life were associated with improved adherence after 4 months, using a measure of quality of life developed for this study. They did not find an association between quality of life and adherence after 8 months, however, after one year they found that higher mental health quality of life predicted adherence. This was

a cross sectional study with follow up at three different time points which makes it difficult to draw conclusions about causal relationships, and causal attributions for heart disease were not investigated specifically in relation to quality of life. Analyses were not adjusted for depression, which as discussed above, may have a negative influence on adherence and quality of life. Only one measure, adherence to medication, was used to assess adherence.

A prospective study by Fogel et al (2004) also examined the relationship between quality of life and adherence, and in particular whether quality of life predicted adherence, independent of depression, after 4 months in post MI patients. Causal attributions in relation to quality of life were not examined in this study. Measures included the Medical Outcomes Short Form Questionnaire (SF-36) to assess quality of life (described fully in chapter 7), the Beck Depression Inventory (BDI), and 7 items relevant to post-MI patients from the Medical Outcomes Study Specific Adherence Scale (MOSSAS) which assesses adherence behaviours such as following a low salt, following a fat or weight loss diet, taking prescribed medication, carrying necessary medicines when going out, exercising, reducing stress, and trying to increase social contacts. Quality of life scores in the physical health summary score did not differ between baseline and 4 months but scores for the mental health summary score improved. Physical health quality of life rather than mental health, however, was found to affect adherence independently of depression. Fogel et al suggested that the physical health measure was associated with deficits in energy and function necessary to prepare proper meals, go to the pharmacy, open pill bottles, exercise etc which may be related to critical aspects of adherence.

A study by Sin et al (2004) of investigated the relationship between quality of life and adherence after 1 year in a sample of 146 cardiac patients who had completed a cardiac rehabilitation programme of 8 – 14 weeks. This was a retrospective study based on secondary analysis prospectively collected data from participants of the Cardiopulmonary Outcomes Prospective Evaluation study (COPE). Quality of life was measured using the SF-36 and adherence was measured by proxy using change scores in physical activity. Overall physical activity and quality of life scores improved over the follow up period. Adherence with exercise sessions was good, 73% of participants had 100% attendance rate. Factors found to influence poorer adherence to advice concerning physical activity and poorer quality of life were female gender, not being employed and high-risk health status. Causal attributions were not investigated. These findings however, generally support evidence from other studies (above) that cardiac rehabilitation programmes are effective at improving physical activity and quality of life outcomes. Measuring quality of life in patients with ACS could help to improve adherence by focussing attention on those who are finding it more difficult to cope.

### 6.9.3: The association between causal beliefs and quality of life

Only one published study has investigated the associations between causal beliefs and quality of life among patients following ACS. A study by French et al (2005b) investigated extent to which illness perceptions predict health related quality of life using the Illness Perceptions Questionnaire (Weinman et al, 1996) and HADS to measure illness perceptions and anxiety and depression in MI patients within 24 hours of hospital admission. They reported that illness perceptions predicted emotional, physical and social quality of life, with beliefs about consequences being most strongly related. Three causal attributions items were negatively related to quality of life,

including stress, other peoples' behaviour and state of mind. These relationships remained after controlling for anxiety and depression.

## 6.10: Hypotheses

This thesis hypothesizes that:

- Adherence to medical advice (attendance at cardiac rehabilitation, medication compliance, and life style change) will be predicted by beliefs about causes, independently of severity and clinical treatment.

- Quality of life and psychological adjustment at 3 months and 13 months following hospital discharge will be predicted by causal beliefs, independently of treatment and clinical indices.

# Chapter 7: Predictors of adherence, adjustment and quality of life 3 months after hospital admission for ACS

## 7.0: Introduction

Understanding patients' causal beliefs may be important in optimizing clinical management and improving adherence to treatment recommendations and life style changes. Causal beliefs may play an important role in identifying patients who find it particularly difficult adjusting to their heart problem and who may benefit from appropriate interventions. Quality of life following hospital discharge may also be predicted by patients' causal attributions. This chapter will present the methodology used to investigate adherence, adjustments and quality of life reported by the study population 3 months and 13 months following hospital discharge. Results of analyses carried out after 3 months will be presented in this chapter, along with a discussion of these findings, while the results of analyses carried out after 13 months will follow in Chapter 8.

## 7.1: Aims

The third aim of this thesis is to investigate the relationship between patients' causal attributions and adherence to medical advice 3 months and 13 months after hospital discharge, and to identify factors which may predict non-adherence.

The fourth and final aim of this thesis will aim to investigate the relationship between patients' causal attributions and adjustment and quality of life 3 months and 13 months after hospital discharge.

## 7.2: Methodology

### 7.2.1: Participants

All participants in the study sample were eligible to take part in the follow up phase (N = 269). Consent was gained at the initial time of recruitment onto this study in order to contact patients for follow up.

### 7.2.2: Procedure

During their initial hospital admission all patients were given advice by medical, nursing or rehabilitation staff regarding increasing their level of exercise, maintaining a healthy weight, managing/reducing their stress levels appropriately, maintaining their alcohol intake within recommended limits, following a healthy diet (for example eating at least 5 portions of fruit and vegetables daily, eating oily fish at least twice per week and following a low fat diet) and quitting smoking (if applicable). All patients were discharged with medication prescribed by their doctor aimed at treatment of the current heart problem and/or secondary prevention. All appropriate patients were invited to cardiac rehabilitation programmes.

Attempts were made to contact and follow up all participants using a telephone interview at 3 months and 13 months after admission. The telephone interview assessed self reported adherence to lifestyle changes recommended by coronary care and rehabilitation staff, attendance cardiac rehabilitation classes (if appropriate) and compliance with prescribed medication (Appendices 8 and 9). Patients were also sent a questionnaire containing the psychological and quality of life measures by post.

### 7.2.3: Measures

#### *7.2.3.1: Telephone interview follow up measures*

Both the 3 month and 13 month follow up telephone interviews was based on a similar interview format used in an earlier study (Ziegelstein et al, 2000) that assessed 10 adherence behaviours relevant for cardiac patients who had suffered an ACS (see Appendices 8 and 9). In this study patients were asked whether or not they had attended a cardiac rehabilitation programme (if appropriate) and how many sessions of the total number they had attended. They were also asked whether or not they had implemented advice given to them by medical, nursing or rehabilitation staff including increasing their level of exercise, maintaining a healthy weight, managing their stress levels appropriately, maintaining their alcohol intake within recommended limits, following a healthy diet and quitting smoking (if applicable). Patients were also asked what medication they were prescribed and whether they took their medication every day. Patients were considered to be adherent if they answered 'yes' to these questions and non-compliant if their answers were 'partial' or 'no'.

An adherence index was developed, similar to the one use by Ziegelstein et al (2000), to assess 5 adherence behaviours relevant to patients who have had an ACS: (1) taking medications as prescribed, (2) eating a healthy diet, (3) maintaining a healthy weight, (4) exercising regularly, (5) managing / reducing stress levels. Scores ranged from 0 (partial or non-adherence) to 1 (adherent). Patients could therefore score a minimum of 0 to a maximum of 5.

### *7.2.3.2: GRACE risk index*

The GRACE risk index is a composite score to define risk of 6-month post discharge death applicable to all types of ACS. It is calculated using an algorithm developed in the Global Registry of Acute Coronary Events (GRACE) study which is based on 9 criteria (age, history of congestive cardiac failure, history of MI, systolic blood pressure and heart rate on admission, ST segment depression, initial serum creatine, elevated cardiac enzymes and no in-hospital percutaneous coronary intervention) (Eagle et al, 2004). These 9 clinical prediction variables are given weighted scores based on the model's variable co-efficients. The algorithm assigns a point total to each variable allowing a total point score for each patient to be calculated. The total score can range from 1 – 263, and this is then applied to a reference plot normogram showing the corresponding risk of death. Higher scores signify greater risk of 6 month mortality in patients with ACS.

### *7.2.3.3: Patients beliefs about the causes of their heart problems*

The measure of causal beliefs (as described in chapter 5.4.1) was repeated at 3 and 13 months follow up.

### *7.2.3.4: Medical Outcome Short Form 36 (SF-36)*

Quality of life was measured using the SF-36 health status measure (see appendix 10), adapted for use in the UK (Jenkinson et al, 1996; Ware & Sherbourne, 1992). The SF-36 assesses 8 domains of health-related quality of life. There are 36 individual items which are grouped into 8 multi-item subscales representing the 8 domains. These include physical function (limitations in physical activity due to physical health), role limitations due to physical problems (problems with work and daily activities due to

physical health), bodily pain (severity), general health perception (evaluation of physical health and likelihood of improvement), vitality (energy level), social functioning (interference with social activities due to physical and emotional health problems), role limitations due to emotional problems (problems with work and daily activities due to emotional problems), and mental health (anxiety and depression). Each subscale is scored so that 0 represents the lowest (worst health) and 100 the highest possible (best health) level of function. Scores for the 8 subscales at baseline were calculated, and change in quality of life was measured by following the procedure advocated by Ware et al (1994) calculating physical and mental health status. This measure also contains 2 summary component scores; summary physical health status was calculated by averaging scores for the physical health domain subscales (physical function, role limitations due to physical problems, bodily pain and general health perception) while summary mental health status was calculated by averaging scores for the mental health domain subscales (vitality, limitations due to emotional problems, social functioning, and general mental health). The SF-36 has been used in a number of studies investigating quality of life among cardiac patients (Brown et al, 1999; Fogel et al, 2004; Rumsfeld et al, 1999). In previous published studies internal reliability statistics have exceeded the minimum standard of 0.70 recommended for measures used in group comparisons (Ware & Gandek, 1998).

#### *7.2.3.5: Beck Depression Inventory (BDI)*

Depression was measured using the second edition of Beck Depression Inventory (BDI) (Appendix 11), a 21-item instrument developed by Beck and Steer (1993). Participants were asked to rate the severity of symptoms ranging from no symptoms (0) to severe (3) on a Likert scale. The range of possible scores is from 0 to 63. The higher the score,

the greater the severity of depressive symptoms. Scores ≥10 are interpreted as indicating at least mild to moderate depression (Ziegelstein et al, 2000). The BDI has been used in a number of studies of patients with cardiac disease and is considered a valid measure of depression (Buchanan et al, 1993; Crowe et al, 1996; Frasure-Smith et al, 1997). A meta-analysis of studies (including cardiac patients) focussing on the psychometric properties and internal reliability of the BDI yielded a mean coefficient alpha of 0.81 for non-psychiatric participants (Beck et al, 1988).

#### *7.2.3.6: Hospital Anxiety Scale (HADS anxiety)*

This is one of two 7 item self report screening sub-scales taken from the Hospital Anxiety and Depression Scale (HADS)(Appendix 12). The complete measure was originally developed to detect the presence of anxiety and depression in a clinical population of medical outpatients suffering from a wide variety of illnesses (Zigmond & Snaith, 1983). HADS has been widely used in studies with patients following AMI as an index of outcome, and to assess quality of life and psychological wellbeing (Trzcieniecka-Green & Steptoe, 1996; Whitmarsh et al, 2003). In this study, only the anxiety sub-scale was used (HADS anxiety scale). This 7-item scale is scored from 1 (not at all anxious) to 3 (very often anxious), but with 5 items reverse scored. Total scores can range from 0 to 21. Higher scores reflect greater anxiety and patients were classified as being at least moderately anxious if their scores exceeded the recognized threshold of ≥8 (Zigmond & Snaith, 1983). HADS was developed for patients with physical illness and was found to be a reliable instrument for detecting severity of emotional distress in a review of validation data by Herrmann (1997). Martin & Thompsom (2000) evaluated the HADS-anxiety measure among a sample of 194 patients with confirmed MI and reported good internal reliability (Cronbach alpha 0.76).

## 7.3 Statistical analyses

The data were analysed using SPSS for windows. Firstly, the main socio-demographic, clinical and psychosocial characteristics of patients who completed the telephone interviews at 3 months and 12 months follow up were compared with those who did not using $\chi^2$ tests for association. Where significant differences between the two groups were found, logistic regression was carried out to determine the direction and strength of the relationship. Changes in mood state (depression and anxiety) over the two time periods (in hospital at baseline and at the 3 month follow up interview) were analysed using correlations and paired t-tests.

The stability of the causal attributions over the two time periods from baseline was analysed using correlations and paired t-tests. Behaviour changes over the 3 months and 13 months follow up periods were analysed using $\chi^2$ tests for association. Change in behaviour was investigated firstly for each of the individual adherence behaviours and relevant items on the causal beliefs questionnaire. For example, the belief that heart disease was caused by physical inactivity was related to self-reported increases in physical activity at 3 and 13 months. Secondly, behaviour changes were associated with the three causal attribution factors. The adherence index was analysed using linear regression. Adherence to advice to attend rehabilitation classes was analysed to investigate associations with the causal belief factors with two variables: whether patients attended a programme or not, and the number of sessions attended.

The relationship between causal attribution factors and psychological well-being at 3 and 13 months was also analysed. Product-moment correlations were computed between BDI and HAD anxiety scales and the three causal attribution factors.

Significant effects were then analysed using linear regression, with the causal attribution factor, age, gender, GRACE risk scores, and the level of the distress measure in hospital in the model. In this way, I tested whether the association between causal beliefs and later well-being was independent of well-being levels in hospital. A similar strategy was used for assessing associations between causal beliefs and 3 and 13 month quality of life, analysing the 8 specific scales and two summary measures from the SF36.

## 7.4: Results of analyses of follow up at 3 months following ACS

### 7.4.0: The study population available for follow up at 3 month post ACS

It was intended that all patients should be contacted to complete the telephone follow up interview 3 months after their hospital admission. Some patients were more difficult to contact, however, and required repeated attempts and there were some patients we were unable to contact at all. The mean interval between hospital admission and follow up telephone interview was 109 days ± 26 (15.4 weeks). The study population of patients who were available to complete the telephone interview at 3 months (N = 216) was compared with those we were unable to contact (N = 53) and this revealed some differences between the two groups (see Table 7.1).

### 7.4.1: Comparison between patients who were available to complete the 3 month follow up telephone interview and those who were not

Results of analyses using chi squared comparing the group who were available to complete the 3 month follow up telephone interview and those that were not are shown in Table 7.1. Only 6 variables showed significant differences between the 2 groups. These included type of ACS, presence of hypercholesterolemia, frequency of physical exercise, size of social network and whether or not the patient had reported a moderate

or high level of stress in their relationship with their partner in the 4 weeks prior to the onset of their symptoms.

**Table 7.1: Comparison between patients who provided interview data at 3 months follow up (N = 216) and patients who did not ( N = 53) in complete sample (N = 269)**

| | | No 3 month interview data N (%) | 3 month interview data completed N (%) | Difference p-value ($\chi^2$) |
|---|---|---|---|---|
| **Demographic factors** | | | | |
| Age: | <50 years | 12 (20.0) | 48 (80.0) | .500 |
| | 50-60 years | 15 (17.4) | 71 (82.6) | |
| | 60-70 years | 10 (17.2) | 48 (82.5) | |
| | > 70 years | 16 (24.6) | 49 (75.4) | |
| Gender: | Men | 45 (21.3) | 166 (78.7) | .202 |
| | Women | 8 (13.8) | 50 (86.2) | |
| Ethnicity: | White | 44 (20.1) | 175 (79.9) | .738 |
| | Other | 9 (18.0) | 41 (82.0) | |
| **Socio-economic factors** | | | | |
| Educational qualifications: | None | 31 (25.2) | 92 (74.8) | .094 |
| | Up to O'level | 8 (13.1) | 53 (86.9) | |
| | A'level + | 14 (16.5) | 71 (83.5) | |
| Deprivation: | Most deprived | 18 (15.3) | 100 (84.7) | .126 |
| | Moderately deprived | 13 (18.8) | 56 (81.2) | |
| | Least deprived | 22 (26.8) | 60 (73.2) | |
| Income per year: | <£20 k | 29 (25.0) | 87 (75.0) | .128 |
| | £20-£40 k | 12 (15.2) | 67 (84.8) | |
| | >£40 k | 10 (16.7) | 50 (83.3) | |
| Occupational group: | Employed | 25 (17.4) | 119 (82.6) | .429 |
| | Unemployed | 6 (30.0) | 14 (70.0) | |
| | Retired | 22 (21.2) | 82 (78.8) | |
| **Social factors** | | | | |
| Marital status: | Not married | 24 (25.3) | 71 (74.7) | .091 |
| | Married | 29 (16.7) | 145 (83.3) | |
| Social network: | Small | 13 (23.2) | 43 (76.8) | **.016** |
| | Medium | 12 (13.5) | 77 (86.5) | |
| | Large | 6 (8.1) | 68 (91.9) | |
| Partner stress within 4 weeks of ACS: | None - Mild | 24 (14.5) | 141 (85.5) | **.049** |
| | Moderate - High | 9 (29.0) | 22 (71.0) | |
| Partner stress within 6 months of ACS: | None - Mild | 26 (15.2) | 145 (84.8) | .065 |
| | Moderate - High | 34 (17.2) | 164 (82.8) | |

(Continued on next page

| | | No 3 month interview data | | 3 month interview data completed | | Difference p-value ($\chi^2$) |
|---|---|---|---|---|---|---|
| **Proximal factors** | | | | | | |
| Time of symptom onset: | | | | | | |
| | Midnight – 0600 hrs | 14 | (24.1) | 44 | (75.9) | .830 |
| | 0600 hrs – midday | 17 | (20.0) | 68 | (68.0) | |
| | Midday – 1800 hrs | 8 | (10.7) | 67 | (89.3) | |
| | 1800 hrs - midnight | 14 | (27.5) | 37 | (72.5) | |
| Day of onset: | Week day | 35 | (19.7) | 143 | (80.3) | .982 |
| | Weekend | 18 | (19.8) | 73 | (80.2) | |
| Season: | Jan - Mar | 21 | (28.0) | 54 | (72.0) | .064 |
| | Apr – June | 13 | (18.3) | 58 | (81.7) | |
| | July - Sept | 10 | (14.7) | 58 | (85.3) | |
| | Oct - Dec | 9 | (16.4) | 46 | (83.6) | |
| Presence of bystander: | Absent | 13 | (15.3) | 72 | (84.7) | .424 |
| | Present | 20 | (19.8) | 81 | (80.2) | |
| **Risk factors** | | | | | | |
| Previous MI: | No | 48 | (20.0) | 192 | (80.0) | .520 |
| | Yes | 4 | (14.8) | 23 | (85.2) | |
| Hypertension: | No | 27 | (18.2) | 121 | (81.8) | .507 |
| | Yes | 26 | (21.5) | 95 | (78.5) | |
| Hypercholesterolemia: | No | 20 | (15.0) | 113 | (85.0) | **.048** |
| | Yes | 32 | (24.8) | 97 | (75.2) | |
| Diabetes: | No | 44 | (18.9) | 189 | (81.1) | .391 |
| | Yes | 9 | (25.0) | 27 | (75.0) | |
| Smoker: | Non-smoker | 11 | (19.3) | 46 | (80.7) | .637 |
| | Ex-smoker | 18 | (17.8) | 83 | (82.2) | |
| | Smoker | 24 | (21.6) | 87 | (78.4) | |
| Alcohol intake: | Non-drinker | 21 | (21.2) | 78 | (78.8) | .567 |
| | Drinker | 31 | (18.3) | 138 | (81.7) | |
| Physical exercise: | | | | | | |
| | Inactive | 27 | (15.8) | 144 | (84.2) | **.004** |
| | Low(<2x per week) | 10 | (18.9) | 43 | (81.1) | |
| | High (>2x per week) | 16 | (37.2) | 27 | (62.8) | |
| **Clinical presentation** | | | | | | |
| Premonitory symptoms: | No | 31 | (21.2) | 115 | (78.8) | .493 |
| | Yes | 22 | (17.9) | 101 | (82.1) | |
| Type of ACS: | | | | | | |
| | UA / NSTEMI | 23 | (28.8) | 57 | (71.3) | **.015** |
| | STEMI | 30 | (15.9) | 159 | (84.1) | |
| Intensity of pain: | >6 | 2 | (4.4) | 43 | (95.6) | .199 |
| | 6-8 | 9 | (19.6) | 37 | (80.4) | |
| | 8-10 | 8 | (13.8) | 50 | (86.2) | |
| Number of non-chest pain symptoms: | none | 18 | (24.3) | 56 | (75.7) | .107 |
| | 1 – 3 | 26 | (20.3) | 102 | (79.7) | |
| | 4 - 8 | 9 | (13.4) | 58 | (86.5) | |
| Number of non-pain symptoms: | | | | | | |
| | None | 22 | (25.6) | 64 | (74.4) | .129 |
| | 1 – 2 | 19 | (17.4) | 90 | (82.6) | |
| | 3 – 6 | 12 | (16.2) | 62 | (83.8) | |
| **Psychological factors** | | | | | | |
| Attribution to heart attack: | | | | | | |
| | No | 44 | (22.0) | 156 | (78.0) | .117 |
| | Yes | 9 | (13.2) | 59 | (86.8) | |
| Cardiac denial of impact: | Low | 10 | (13.0) | 67 | (87.0) | .405 |
| ( tertiles) | Middle | 8 | (11.8) | 60 | (88.2) | |
| | High | 13 | (17.8) | 60 | (82.2) | |

Table 7.2 shows results of further analyses using logistic regression. Patients who had suffered an STEMI were more likely to be available to provide 3 month telephone interview data than patients who had suffered a NSTEMI / UA. Patients with hypercholesterolemia were less likely to provide 3 month follow up data. Patients who did physical exercise more than twice a week were less likely to provide 3 month telephone interview data. Patients who were available to provide 3 month telephone interview data were also more likely to have a larger social network than patients who were not available, and were less likely to report experiencing moderate to high levels of stress in their relationship with their partner in the 4 weeks prior to their ACS.

**Table 7.2: Results of logistic regression comparing patients who provided telephone follow up data 3 month post ACS with those who did not**

| | | **Odds ratio (95% Confidence Interval) (adjusted for age and gender)** | | **p-value** |
|---|---|---|---|---|
| Type of ACS: | NSTEMI/UA | 1 | | |
| | STEMI | 2.17 | (1.16 – 4.07) | **0.015** |
| Hypercholesterolemia: | No | 1 | | |
| | Yes | 0.52 | (0.28 – 0.97) | **0.002** |
| Physical exercise: | Sedentary | 1 | | |
| | Up to 2x per week | 0.78 | (0.35 – 1.75) | 0.55 |
| | More than 2x per week | 0.30 | (0.14 – 0.65) | **0.002** |
| Social network: | Small | 1 | | |
| | Medium | 1.93 | (0.80 – 4.67) | 0.144 |
| | Large | 3.57 | (1.24 – 10.32) | **0.019** |
| Partner stress in the 4 weeks prior to ACS: | None – Mild | 1 | | |
| | Moderate - Very | 0.39 | (0.16 – 0.97) | 0.043 |

Comparison of levels of depression and anxiety at baseline between patients who did and did not provide 3 month follow up data were carried out using t-tests and showed no significant differences between groups for either mood state (see Table 7.3)

**Table 7.3: Comparison of means for level of depression and anxiety at baseline between patients who provided 3 month follow up data and those who did not**

| | N | Mean (SD) | *p-value |
|---|---|---|---|
| **Depression (baseline total BDI score)** | | | |
| No 3 month follow up data | 30 | 9.85 (7.87) | .424 |
| 3 month follow up data provided | 179 | 8.66 (7.54) | |
| **Anxiety (baseline total HADS anxiety score)** | | | |
| No 3 month follow up data | 30 | 5.97 (3.87) | .801 |
| 3 month follow up data provided | 184 | 5.77 (3.93) | |

(* p-value from t-test of difference between means)

The 3 causal attribution factors of the group of patients who provided 3 month follow up data were also compared with the group of patients who did not provide 3 month follow up data using t-tests. Again, no significant differences were found between patients who did and did not provide 3 month follow up data for any of the 3 factors (see Table 7.4).

**Table 7.4: Comparison of means for the 3 factors at baseline between patients who did and did not provide 3 month follow up data**

| | N | Mean (SD) | *p-value |
|---|---|---|---|
| **Mental state factor** | | | |
| No 3 month follow up data | 22 | 2.82 (2.52) | .858 |
| 3 month follow up data provided | 144 | 2.92 (2.46) | |
| **Personal behaviour factor** | | | |
| No 3 month follow up data | 22 | 3.50 (2.52) | .828 |
| 3 month follow up data provided | 144 | 3.61 (2.19) | |
| **Personal behaviour factor** | | | |
| No 3 month follow up data | 22 | 2.83 (3.11) | .682 |
| 3 month follow up data provided | 144 | 3.15 (3.27) | |

(* p-value from t-test of difference between means)

In summary, patients who were available to complete the 3 months telephone interview follow up were more likely to have had an STEMI, have normal cholesterol levels, exercise less than twice per week and to have a large social network. They were also less likely to have experienced more than mild levels of stress in their relationship with their partner in the 4 weeks prior to their ACS. However, they did not differ in age, gender, ethnicity, socioeconomic position, or factors surrounding the onset of their ACS. Most importantly, the two groups did not differ in psychological state or causal attributions during hospitalisation.

### 7.4.2: Stability of causal attributions over 3 months

Of the patients who returned their baseline questionnaires, 165 completed the measure of causal beliefs. The same measure was completed by 110 patients at 3 months follow up. The stability of this measure was analysed using correlations and paired t-tests. Firstly, the mean scores were compared for each of the 3 factors (mental state, personal behaviour and heredity) at baseline and at 3 months. Correlations between the two time periods were significant for all three factors (see Table 7.5). Paired t-tests showed no significant difference between means at baseline and three months later for any of the factors. The internal reliability for each of the three factors at three months was satisfactory; mental state factor had an internal reliability (Cronbach alpha) of 0.77, personal behaviour was 0.60 and heredity was 0.76. Test-retest studies were also performed and showed average anova intraclass correlation co-efficients for each of the three factors as follows; mental state factor 0.81, personal behaviour factor 0.79, and heredity factor 0.83.

**Table 7.5: Correlations between causal attribution factors at baseline and 3 months**

| Factor | N | Mean (SD) | Baseline to 3 month correlation | p-value for correlation |
|---|---|---|---|---|
| **Mental state factor:** | | | | |
| At 3 month follow up | 110 | 2.85 (2.52) | 0.68 | **<0.001** |
| Baseline | 110 | 2.84 (2.47) | | |
| **Personal behaviour factor:** | | | | |
| At 3 month follow up | 109 | 3.66 (2.04) | 0.66 | **<0.001** |
| Baseline | 109 | 3.50 (1.91) | | |
| **Heredity factor:** | | | | |
| At 3 month follow up | 109 | 3.12 (3.19) | 0.71 | **<0.001** |
| Baseline | 109 | 3.00 (3.11) | | |

Secondly, the stability of each of the 16 items in the measure was tested using correlations. The scores at baseline and at 3 months later for all of the 16 items were highly correlated ($p < 0.005$). Paired t-tests showed no significant differences between mean scores for each of the individual items at baseline and at 3 months later. Evidence from these analyses shown in Table 7.6 therefore suggests therefore that the measures used to examine causal attributions in this study were stable over the 3 month follow up period. It can be seen that there were no significant changes in the strength of beliefs in any causal item between baseline and 3 months. Moreover, the test-retest correlations were all significant, indicating reasonable stability over this time period. The highest correlation was for smoking (0.87) and the lowest was for poor diet (0.21). The diet effect was the only one not to be signficant at $p<0.001$.

**Table 7.6: T-test and individual correlations for the causal belief individual items at baseline and 3 months follow up**

| Factor | Time point | N | Mean | SD | t | p-value of t | Pearsons' r | p-value of r |
|---|---|---|---|---|---|---|---|---|
| Heredity | Baseline | 108 | 0.65 | 0.75 | -0.69 | 0.50 | 0.73 | <0.001 |
| | 3 months | 108 | 0.69 | 0.78 | | | | |
| Smoking | Baseline | 109 | 0.80 | 0.85 | -0.65 | 0.52 | 0.87 | <0.001 |
| | 3 months | 109 | | | | | | |
| Other medical problems | Baseline | 108 | 0.47 | 0.63 | 1.35 | 0.18 | 0.40 | <0.001 |
| | 3 months | 108 | 0.39 | 0.53 | | | | |
| Stress | Baseline | 108 | 0.90 | 0.77 | -0.83 | 0.41 | 0.60 | <0.001 |
| | 3 months | 108 | 0.95 | 0.77 | | | | |
| Overweight | Baseline | 107 | 0.41 | 0.60 | -0.54 | 0.59 | 0.63 | <0.001 |
| | 3 months | 107 | 0.44 | 0.66 | | | | |
| Blood pressure | Baseline | 107 | 0.77 | 0.75 | -1.52 | 0.13 | 0.65 | <0.001 |
| | 3 months | 107 | 0.86 | 0.78 | | | | |
| Poor diet | Baseline | 108 | 0.49 | 0.62 | -0.96 | 0.34 | 0.21 | 0.03 |
| | 3 months | 108 | 0.56 | 0.66 | | | | |
| Overexertion | Baseline | 109 | 0.45 | 0.66 | -0.26 | 0.79 | 0.40 | <0.001 |
| | 3 months | 109 | 0.47 | 0.67 | | | | |
| Bad luck | Baseline | 106 | 0.71 | 0.80 | -0.23 | 0.82 | 0.43 | <0.001 |
| | 3 months | 106 | 0.73 | 0.81 | | | | |
| Poor medical care | Baseline | 106 | 0.12 | 0.36 | -1.62 | 0.12 | 0.53 | <0.001 |
| | 3 months | 106 | 0.19 | 0.48 | | | | |
| Lack of exercise | Baseline | 109 | 0.53 | 0.65 | 0.16 | 0.87 | 0.58 | <0.001 |
| | 3 months | 109 | 0.52 | 0.66 | | | | |
| Tiredness | Baseline | 108 | 0.51 | 0.65 | -1.26 | 0.21 | 0.45 | <0.001 |
| | 3 months | 108 | 0.59 | 0.66 | | | | |
| Genetic factors | Baseline | 108 | 0.56 | 0.63 | -0.15 | 0.89 | 0.47 | <0.001 |
| | 3 months | 108 | 0.56 | 0.66 | | | | |
| State of mind | Baseline | 106 | 0.46 | 0.62 | 1.38 | 0.17 | 0.62 | <0.001 |
| | 3 months | 106 | 0.39 | 0.67 | | | | |
| Working too hard | Baseline | 107 | 0.44 | 0.65 | -0.49 | 0.62 | 0.59 | <0.001 |
| | 3 months | 107 | 0.47 | 0.65 | | | | |
| Germ/virus | Baseline | 57 | 0.07 | 0.26 | -0.44 | 0.66 | 0.54 | <0.001 |
| | 3 months | 57 | 0.09 | 0.34 | | | | |

### 7.4.3: Baseline causal attributions and behaviour change at 3 months follow up

All patients had received advice from medical or nursing staff as an in-patient or via cardiac rehabilitation classes (if appropriate) to follow specific recommendations; to quit smoking (if applicable), to undertake regular exercise, to maintain a healthy body weight according to current recommendations, to manage stress more effectively, to limit their alcohol intake to within recommended limits, to eat a healthy diet (including at least 5 portions of fruit and vegetables daily, to follow a low fat diet and to eat oily fish at least twice a week) (British Heart Foundation, 2005b). Patients had also received advice about how and when to take prescribed medication. Information booklets were freely available on hospital wards or from nursing staff. Each of the above behaviours was analysed to identify changes which may be associated with relevant items included in the causal beliefs questionnaire and which were endorsed by patients at baseline.

#### *7.4.3.1: Smoking at 3 months follow up*

All patients who were smokers at baseline were advised to stop smoking, either by coronary care nurses, medical staff or cardiac rehabilitation nurses. Although 72.4% of baseline smokers said that they had quit by the 3 month telephone follow up, there was no association between stopping smoking at 3 months and beliefs held by smokers at baseline that smoking caused their heart problem (see Table 7.7).

**Table 7.7: Association between causal attribution to smoking at baseline and smoking status of baseline smokers at 3 months follow up**

| | Belief at baseline that smoking caused the heart problem | | | |
|---|---|---|---|---|
| **Smoking at 3 months follow up** | **No** N (%) | **Maybe** N (%) | **Yes** N (%) | **p-value** |
| No | 5 (11.1) | 16 (35.6) | 24 (53.3) | 0.808 |
| Yes | 1 (6.3) | 8 (50.0) | 7 (43.8) | |

### *7.4.3.2: Healthy diet at 3 months follow up*

All patients were advised to adopt a healthy diet. This includes eating more fruit and vegetables (at least 5 portions per day), oily fish such as mackerel or salmon at least twice a week, and to follow a low fat diet. They were either given a leaflet containing these recommendations or advised by cardiac rehabilitation nurses. Analyses showed that there was no association between patients' beliefs at baseline that poor diet had caused their heart problem and whether they said they had changed their diet at 3 months (see Table 7.8).

**Table 7.8: Association between causal attribution to poor diet at baseline and dietary change at 3 months follow up**

| | Belief at baseline that poor diet caused the heart problem | | | |
|---|---|---|---|---|
| **Diet change at 3 months follow up** | **No** N (%) | **Maybe** N (%) | **Yes** N (%) | **p-value** |
| No | 34 (56.7) | 22 (36.7) | 4 (6.7) | 0.417 |
| Yes | 43 (53.1) | 28 (34.6) | 10 (12.3) | |

### *7.4.3.3: Physical activity at 3 months follow up*

All patients were informed of the importance of regular exercise in maintaining their health and were advised to exercise regularly within the limits of their own individual

capabilities. Analyses showed that there was no association between patients belief that lack of exercise had caused their heart problem at baseline and whether they said that they had changed their exercise behaviour over the three month follow up period (see Table 7.9).

**Table 7.9: Association between causal attribution to lack of exercise at baseline and change in exercise behaviour at 3 months follow up**

| | Belief at baseline that lack of exercise caused the heart problem | | | |
|---|---|---|---|---|
| **Change in exercise behaviour at 3 months follow up** | **No** N (%) | **Maybe** N (%) | **Yes** N (%) | **p-value** |
| No | 25 (48.1) | 21 (40.4) | 6 (11.5) | 0.078 |
| Yes | 55 (61.8) | 29 (32.6) | 5 (5.6) | |

#### *7.4.3.4: Control of body weight at 3 months follow up*

All patients were advised to maintain their body weight within recommended limits (British Heart Foundation, 2005b). Those who were overweight were advised to try to reduce their weight. At the 3 month follow up interview, however, no association was found between patients' baseline belief that being overweight had caused their heart problem and weight change reported at the 3 month follow up interview (see Table 7.10). Neither was there any association between patients' belief that poor diet caused their heart problem at baseline and change in body weight at the 3 months, or patients' belief that lack of physical exercise caused their heart problem and change in body weight at 3 months.

**Table 7. 10: Association between causal attribution to being overweight at baseline and reported change in body weight at 3 months follow up**

| Change in body weight at 3 months follow up | Belief at baseline that being overweight caused the heart problem<br>No<br>N (%) | Maybe<br>N (%) | Yes<br>N (%) | p-value |
|---|---|---|---|---|
| No | 21 (39.6) | 25 (47.2) | 7 (13.2) | 0.640 |
| Yes | 14 (42.4) | 16 (48.5) | 3 (9.1) | |

These analyses were repeated to include only patients who were overweight or obese at baseline. Again, there was no association between belief that being overweight caused the heart problem at baseline and change in body weight at 3 months (see Table 7.11).

**Table 7. 11: Association between causal attribution to being overweight at baseline and reported change in body weight at 3 months in overweight or obese patients**

| Change in body weight at 3 months follow up | Belief at baseline that being overweight caused the heart problem<br>No<br>N (%) | Maybe<br>N (%) | Yes<br>N (%) | p-value |
|---|---|---|---|---|
| No | 24 (45.3) | 23 (43.4) | 6 (11.3) | 0.240 |
| Yes | 11 (34.4) | 15 (46.9) | 6 (18.8) | |

### *7.4.3.5: Stress at 3 months follow up*

All patients were given advice concerning the importance of stress management and relaxation, either via patient information leaflets or cardiac rehabilitation nurses. Analyses showed that there was no association between patients' belief at baseline that stress caused their heart problem and whether they had made any changes in their life to the way they responded to stress or to reduce the stress in their life (see Table 7.12).

**Table 7. 12: Association between causal attribution to stress at baseline and whether patient had tried to reduce the amount of stress in their life at 3 months**

| Change in stress at 3 months follow up | Belief at baseline that stress caused the heart problem<br>No<br>N (%) | Maybe<br>N (%) | Yes<br>N (%) | p-value |
|---|---|---|---|---|
| No | 27 (30.3) | 38 (42.7) | 24 (27.0) | 0.387 |
| Yes | 22 (41.5) | 17 (32.1) | 14 (26.4) | |

These data were then analysed to see if patients' baseline causal beliefs that over exertion caused their heart problem was associated with self reported changes in behaviour to reduce or manage stress at three months follow up. No association was found (see Table 7.13).

**Table 7.13: Association between causal attribution to over exertion at baseline and reported stress reduction/management at 3 months follow up**

| Change in stress at 3 months follow up | Belief at baseline that over exertion caused the heart problem<br>No<br>N (%) | Maybe<br>N (%) | Yes<br>N (%) | p-value |
|---|---|---|---|---|
| No | 51 (57.3) | 27 (30.3) | 11 (12.4) | 0.469 |
| Yes | 35 (64.8) | 13 (24.1) | 6 (11.1) | |

Data were also analysed to examine whether patients' baseline attribution to state of mind as a cause of their heart problem was associated with any reported change in behaviour to reduce stress after 3 months. These analyses revealed no significant associations between baseline attribution to state of mind and stress behaviour change after 3 months (see Table 7.14).

**Table 7.14: Association between causal attribution to state of mind at baseline and reduction of stress at 3 months follow up**

| Change in stress at 3 months follow up | Belief at baseline that state of mind caused the heart problem<br>No<br>N (%) | Maybe<br>N (%) | Yes<br>N (%) | p-value |
|---|---|---|---|---|
| No | 50 (56.8) | 30 (34.1) | 8 (9.1) | 0.440 |
| Yes | 35 (66.0) | 13 (24.5) | 5 (9.4) | |

In the same way, data were analysed to investigate any possible associations with the patients' belief at baseline that working too hard had caused the heart problem and behaviour changes aimed at managing stress reported at the 3 month follow up telephone interview. Again no significant association was found (see Table 7.15). This association also remained insignificant when the sample was limited to include only patients who were in employment at baseline.

**Table 7.15: Association between causal attribution to working too hard at baseline and stress reduction at 3 months follow up**

| Change in stress at 3 months follow up | Belief at baseline that working too hard caused the heart problem<br>No<br>N (%) | Maybe<br>N (%) | Yes<br>N (%) | p-value |
|---|---|---|---|---|
| No | 56 (63.6) | 23 (26.1) | 9 (10.2) | 0.483 |
| Yes | 35 (64.8) | 17 (31.5) | 2 (3.7) | |

### 7.4.4 : Relationships between the causal attribution factors and behaviour changes at 3 months follow up

Patients' adherence to medical advice concerning lifestyle changes and prescribed medication (as described above) was also examined in relation to the causal attribution factors; mental state factor, personal behaviour factor and heredity factor.

#### *7.4.4.1 Relationship between mental state factor and adherence after 3 months*

As shown in Table 7.16, no significant associations were found between patients' baseline scores on the mental state factor (reported as tertiles) and the self reported behaviours assessed 3 months later. Although all patients who were smokers at baseline were advised to stop smoking, no association was found between beliefs held by smokers at baseline that their mental state caused their heart problem and whether they had quit smoking or not 3 months later. Neither were patients' attributions to mental state factor associated with changes in behaviour regarding diet, exercise, body weight or stress management. The relationship with adherence to medication was also analysed. The large majority (83.7%) described themselves as adherent to medication, but this was not related to causal attributions to mental state.

**Table 7.16: Association between mental state factor and self-reported behaviour changes at 3 months follow up**

| Behaviour change at 3 months follow up | | Attribution to mental state factor (tertiles) Low N (%) | Medium N (%) | High N (%) | p-value |
|---|---|---|---|---|---|
| Smoking behaviour: *(among baseline smokers only)* | | | | | |
| | Continue to smoke | 7 (36.8) | 6 (31.6) | 6 (31.6) | 0.818 |
| | Quit | 15 (34.1) | 14 (31.8) | 15 (34.1) | |
| Diet change: | No | 23 (37.7) | 15 (24.6) | 23 (37.7) | 0.867 |
| | Yes | 28 (33.7) | 25 (30.1) | 30 (36.1) | |
| Exercise change: | No | 21 (38.9) | 16 (29.6) | 17 (31.5) | 0.338 |
| | Yes | 30 (33.3) | 24 (26.7) | 36 (40.0) | |
| Change in body weight: | No | 34 (34.3) | 30 (30.3) | 35 (35.4) | 0.937 |
| | Yes | 17 (37.8) | 10 (22.2) | 18 (40.0) | |
| Change in body weight: *(among patients with baseline BMI>25)* | | | | | |
| | No | 20 (37.0) | 16 (29.6) | 18 (33.3) | 0.258 |
| | Yes | 11 (33.3) | 5 (15.2) | 17 (51.5) | |
| Change in stress management: | No | 27 (30.0) | 27 (30.0) | 36 (40.0) | 0.118 |
| | Yes | 24 (44.4) | 13 (24.1) | 17 (31.5) | |
| Medication adherence at 3 months: | | | | | |
| | Non-adherent | 6 (27.3) | 6 (27.3) | 10 (45.5) | 0.340 |
| | Adherent | 43 (37.1) | 31 (26.7) | 42 (36.2) | |

I also analysed associations between causal attribution factors and the adherence index.

The index had a wide distribution of scores, as follows: 0 (completely non-adherent)

- 5.6%, 1 – 21.8%, 2 – 18.1%, 3 – 21.8%, 4 – 20.8%, and 5 (completely adherent)

-12.0%. These scores were entered into a linear regression, but no significant

association between the mental state factor and the adherence index was found

(B = 0.003, C.I. -0.10 to 0.11, p = 0.95).

### *7.4.4.2: Relationship between personal behaviour factor and adherence at 3 months follow up*

As shown in Table 7.17, no significant associations were found between patients' baseline scores on the personal behaviour factor (reported as tertiles) and the self reported behaviours assessed after 3 months.

**Table 7.17: Association between personal behaviour factor and self reported behaviour changes at 3 months follow up**

| **Behaviour change at 3 months follow up** | | **Attribution to personal behaviour factor** (tertiles) **Low** N (%) | **Medium** N (%) | **High** N (%) | **p-value** |
|---|---|---|---|---|---|
| Smoking behaviour: *(among baseline among smokers only* | | | | | |
| | Continue to smoke | 8 (42.1) | 4 (21.1) | 7 (36.8) | 0.886 |
| | Quit | 12 (26.7) | 22 (48.9) | 11 (24.4) | |
| Diet change: | No | 20 (32.3) | 27 (43.5) | 15 (24.2) | 0.678 |
| | Yes | 30 (36.6) | 24 (29.3) | 28 (34.1) | |
| Exercise change: | No | 12 (24.5) | 21 (42.9) | 16 (32.7) | 0.165 |
| | Yes | 29 (38.7) | 26 (34.7) | 20 (26.7) | |
| Change in body weight: | No | 14 (26.4) | 18 (34.0) | 21 (39.6) | 0.430 |
| | Yes | 7 (21.2) | 10 (30.3) | 16 (48.5) | |
| Change in body weight: *(among patients with baseline BMI>25)* | | | | | |
| | No | 7 (20.6) | 11 (32.4) | 16 (47.1) | 0.967 |
| | Yes | 7 (21.2) | 10 (30.3) | 16 (48.5) | |
| Change in stress management: | No | 28 (31.1) | 34 (37.8) | 28 (31.1) | 0.350 |
| | Yes | 22 (40.7) | 17 (31.5) | 15 (27.8) | |
| Medication adherence at 3 months: | | | | | |
| | Non-adherent | 5 (23.8) | 8 (38.1) | 8 (38.1) | 0.236 |
| | Adherent | 44 (37.6) | 39 (33.3) | 34 (29.1) | |

Using linear regression analyses, no significant association was found between the personal behaviour factor and the adherence index (B = -0.027, C.I. -0.15 to 0.09, p = 0.65).

### *7.4.4.3: Relationship between the heredity factor and adherence after 3 months*

As shown in Table 7.18 no significant associations were found between patients' scores on the heredity factor (reported as tertiles) at baseline and the self reported behaviours assessed at the 3 month follow up following the diagnosis of ACS.

**Table 7.18: Association between the heredity factor and self reported behaviour changes at 3 months follow up**

| Behaviour change at 3 months follow up | | Attribution to the heredity factor (tertiles) Low N (%) | Medium N (%) | High N (%) | p-value |
|---|---|---|---|---|---|
| Smoking behaviour *(among baseline smokers only):* | | | | | |
| | Continue to smoke | 11 (57.9) | 2 (10.5) | 6 (31.6) | .122 |
| | Quit | 16 (36.4) | 6 (13.6) | 22 (50.0) | |
| Diet change: | No | 25 (41.0) | 9 (14.8) | 27 (44.3) | .713 |
| | Yes | 35 (42.7) | 14 (17.1) | 33 (40.2) | |
| Exercise change: | No | 23 (42.6) | 7 (13.0) | 24 (44.4) | .851 |
| | Yes | 37 (41.6) | 16 (18.0) | 36 (40.4) | |
| Change in body weight: | No | 41 (41.8) | 17 (17.3) | 40 (40.8) | .845 |
| | Yes | 19 (42.2) | 6 (13.3) | 20 (44.4) | |
| Change in body weight *(among patients with baseline BMI>25):* | | | | | |
| | No | 19 (35.8) | 8 (15.1) | 26 (49.1) | .519 |
| | Yes | 14 (42.4) | 5 (15.2) | 14 (42.4) | |
| Change in stress management: | No | 28 (31.1) | 34 (37.8) | 28 (31.1) | .350 |
| | Yes | 22 (40.7) | 17 (31.5) | 15 (27.8) | |
| Medication adherence at 3 months: | | | | | |
| | Non-adherent | 11 (52.4) | 1 (4.8) | 9 (42.9) | .719 |
| | Adherent | 48 (41.4) | 22 (19.0) | 46 (39.7) | |

Using linear regression analyses, no significant association was found between the heredity factor and the adherence index (B = 0.01, C.I. -0.09 – 0.06, p = 0.70).

### 7.4.5: Attendance at cardiac rehabilitation programmes at 3 months follow up

Attendance at rehabilitation programmes was measured in two ways: firstly whether patients said that they attended any sessions at all or not; secondly, the number of sessions attended. Data regarding the former was available for all participants who completed the 3 month telephone interview (N = 216) except one which was missing. A total of 126 patients (58.6%) said that they attended a course. Data for the latter was available for 202 participants with 14 patients who did not specify the number of sessions attended. When attendance was calculated as a percentage of the total course, 23.2% of patients who attended a cardiac rehabilitation programme attended all sessions constituting the course and 49.1% attended half the course or less.

No significant correlations were found between either attendance at a cardiac rehabilitation programme or number of sessions attended and the following variables; gender, level of education, GRACE risk score, type of ACS (STEMI or NSTEMI/UA), or type of treatment (medication, coronary bypass grafts or angioplasty). Age, however, was negatively correlated with the number of sessions attended in that older patients were likely to attend fewer sessions ($r = -0.16$, $p = 0.023$). Patients who had suffered a previous MI were also less likely to attend a cardiac rehabilitation programme than patients who had not suffered a previous MI ($r = -0.17$, $p = 0.013$), and patients who had suffered a previous MI were also likely to attend fewer sessions than those who had not ($r = -0.17$, $p = 0.019$). No significant correlations were found between cardiac rehabilitation programme attendance or the number of sessions attended, and any of the three causal attribution factors (mental state, personal behaviour or heredity). Partial correlations were computed, controlling for GRACE risk score, previous history of MI, and age, and results are shown in Table 7.19.

**Table 7.19 Correlations between causal attribution factors and patients' attendance at a cardiac rehabilitation programme and number of sessions attended**

| Factor | Pearson correlation | p-value * |
|---|---|---|
| **Mental state factor** (tertiles) | | |
| Attendance at cardiac rehabilitation programme | 0.087 | 0.309 |
| Number of sessions attended | 0.088 | 0.325 |
| **Personal behaviour factor** (tertiles) | | |
| Attendance at cardiac rehabilitation programme | -0.034 | 0.694 |
| Number of sessions attended | -0.114 | 0.201 |
| **Heredity factor** | | |
| Attendance at cardiac rehabilitation programme | 0.001 | 0.991 |
| Number of sessions attended | 0.032 | 0.724 |

* Adjusted for GRACE risk score, previous MI, age & gender

### *7.4.5.1: Summary*

This study found no evidence to support the hypothesis that patients' baseline causal attributions were related to self-reported adherence to medical advice, specifically changes in behaviour regarding lifestyle (smoking, diet, exercise, body weight, stress management), attendance at a rehabilitation programme and adherence to medication as prescribed.

### 7.4.6: Causal attributions and psychological adjustment (depression and anxiety)

All patients followed up after 3 months were asked to complete the questionnaire pack containing measures assessing mood state, the Beck Depression Inventory (BDI ) (appendix 11) and the anxiety scale of the Hospital Anxiety and Depression Scale (HADS anxiety) (appendix 12). Data was then examined to determine whether there was a relationship between mood state and the causal attribution factors.

#### *7.4.6.1: Description of sample at 3 months follow up*

Of the 216 participants who were followed up by telephone interview 3 months after their ACS, 111 patients returned the 3 month follow up questionnaire pack having completed the BDI, and 109 patients completed the HADS anxiety scale. A total of 39.6% of patients who responded reported a high level of depression (BDI score ≥10 threshold) while 29.4% of patients reported high levels of anxiety (HADS anxiety score ≥8 threshold).

#### *7.4.6.2: Relationship between the 3 causal factors at baseline and mood state*

Mood state at baseline was measured using mean scores on the BDI and HADS anxiety. Results are shown in Table 7.19. Just over one third of patients had score on the BDI ≥ 10 indicating that they were depressed, and just under one third had scores ≥8 on the HADS anxiety indicating that they had high levels of anxiety (see Table 7.20).

**Table 7.20 Mean baseline scores for depression and anxiety**

| Mood state | N | Mean | SD | % above threshold |
|---|---|---|---|---|
| Depression | 209 | 8.83 | 7.58 | 38.6 |
| Anxiety | 214 | 5.80 | 3.91 | 30.8 |

The relationship between causal attributions and mood state at baseline was examined using correlations. Analyses showed that patients who attributed their heart problem to the mental state factor were significantly more likely to have greater levels of depression and anxiety at baseline (see Table 7.21). Patients who attributed their heart problems to personal behaviour were also significantly more likely to have higher levels of depression at baseline. There was no association between the heredity factor and baseline level of depression or anxiety.

**Table 7.21: Relationship between the 3 causal factors and baseline level of depression and anxiety**

| Causal attribution factor | BDI total score at baseline | HADS anxiety total score at baseline | Baseline BDI score >10 | Baseline HADS anxiety score >8 |
|---|---|---|---|---|
| **Mental state** | | | | |
| Pearson correlation | 0.40 | 0.16 | 0.32 | 0.12 |
| p-value | **<0.001** | **0.05** | **<0.001** | 0.20 |
| **Personal behaviour** | | | | |
| Pearson correlation | 0.17 | 0.04 | 0.16 | 0.05 |
| p-value | **0.04** | 0.59 | **0.04** | 0.52 |
| Heredity | | | | |
| Pearson correlation | 0.09 | 0.02 | 0.11 | -0.07 |
| p-value | 0.26 | 0.77 | 0.19 | 0.36 |

### *7.4.6.3: Relationship between baseline causal attributions and mood state after 3 months*

The relationship between baseline causal attributions and mood state 3 month following ACS was examined using product-moment correlations with the three causal attribution factors; the mental state factor, personal behaviour factor and heredity factor. There was a significant positive correlation between the mental state factor and self reported level

of anxiety at 3 months follow up (r = 0.27, p = 0.007). There was also a significant positive correlation between heredity factor and level of anxiety at 3 months follow up (r = 0.22, p = 0.028). There were no significant correlations between these two factors and level of depression after 3 months, and the personal behaviour factor was not significantly correlated with either level of anxiety or depression.

#### *7.4.6.4: Causal attributions to mental state and mood at 3 months*

Anxiety at 3 months is likely to be correlated with baseline anxiety. Baseline anxiety was also correlated with scores on the mental state attribution factor ($r$ = 0.17, $p$ = 0.05). It is possible therefore that the association between anxiety at 3 months and causal attributions could be secondary to a common relationship with baseline anxiety. A linear regression was therefore conducted to determine whether the association between causal attributions to mental state and 3 month anxiety was independent of baseline anxiety and other factors. Table 7.22 shows that patients' belief that mental state was a causal factor predicted levels of anxiety after 3 months (B = 1.10, C.I. = 0.091 – 2.11, p = 0.033). Patients' level of anxiety at baseline also predicted level of anxiety 3 months later (B = 0.23, C.I. = 0.014 – 0.45, p = 0.037). Age, gender and GRACE risk score were not significantly related to level of anxiety at 3 months follow up. Consequently, it appears that attributions to mental state predicted 3 month anxiety independently of baseline anxiety levels.

**Table 7.22: Mental state factor as a predictor of anxiety at 3 months follow up**

| | Unstandardized B coefficients | 95% Confidence Interval | p-value |
|---|---|---|---|
| **Mental state factor** | 1.10 | 0.09 – 2.11 | **0.033** |
| **Baseline level of anxiety** | 0.23 | 0.01 – 0.45 | **0.037** |
| Age | 0.10 | -1.23 – 1.44 | 0.879 |
| Gender | 0.47 | -1.59 – 2.54 | 0.650 |
| GRACE risk score | -0.03 | -0.09 – 0.03 | 0.307 |

### *7.4.6.5: Causal attributions to heredity and mood at 3 months*

A linear regression was conducted to determine whether causal attributions to heredity at baseline predicted mood state after 3 months independently of co-factors . Results shown in Table 7.23 indicated that patients' belief that heredity was a causal factor predicted levels of anxiety after 3 months (B = 1.04, C.I. = 0.13 – 1.95, p = 0.026).

**Table 7.23: Heredity factor as a predictor of anxiety at 3 months follow up**

| | Unstandardized B coefficients | 95% Confidence Interval | p-value |
|---|---|---|---|
| **Heredity factor** | 1.04 | 0.13 – 1.95 | **0.026** |
| Baseline level of anxiety | 1.00 | -0.12 – 2.11 | 0.078 |
| Age | 0.07 | -1.29 – 1.43 | 0.919 |
| Gender | 0.67 | -1.41 – 2.76 | 0.524 |
| GRACE risk score | -0.04 | -0.10 – 0.02 | 0.176 |

*7.4.6.6: Summary*

Patients with greater levels of anxiety at baseline were more likely to report greater levels of anxiety at the 3 months follow up. Patients who had stronger baseline beliefs that their heart problem was caused by the mental state factor and/or the heredity factor at baseline also had higher levels of anxiety at the 3 month follow up, independent of age, gender and GRACE scores. Baseline causal attributions did not significantly predict depression at the 3 months follow up.

### 7.4.7: Causal attribution factors and quality of life at 3 months

Between 105-111 patients completed the 3 month follow up measure (SF-36) assessing self reported quality of life. Mean scores for the 8 individual scales and 2 summary scales are shown in Table 7.24. Poorer self rated quality of life was particularly marked in relation to role limitations due to physical problems and vitality.

**Table 7.24: Mean scores of self-rated quality of life (SF-36) at 3 months follow up**

| Component scales of SF-36 | N | *Mean (SD) |
|---|---|---|
| Physical functioning | 108 | 61.97 (± 24.90) |
| **Activity limitations due to physical problems** | 106 | **39.62 (± 42.24)** |
| Activity limitations due to emotional problems | 105 | 63.81 (±40.06) |
| Social functioning | 105 | 73.86 (±24.12) |
| Mental health | 109 | 70.75 (±19.53) |
| **Vitality** | 108 | **53.94 (±20.30)** |
| Health perception | 110 | 60.00 (±21.47) |
| Pain | 103 | 70.66 ((±23.89) |
| Summary scale of physical health status | 111 | 57.50 (±23.89) |
| Summary scale of mental health status | 110 | 64.85 (±22.64) |

*Scores range from 0 (very poor quality of life) to 100 (excellent quality of life).

#### *7.4.7.1 Quality of life and cardiological and demographic variables at 3 mo-nths*

Data were analysed using correlations to determine whether there was an association between each of the 8 individual scales and 2 summary components of quality of life measured by the SF36 and relevant variables such as age, gender, GRACE risk score, type of ACS ( STEMI or NSTEMI / UA), previous MI or type of treatment received (coronary artery bypass grafts, angioplasty/stent or medication). Because of multiple comparisons I adopted a more stringent criterion for the significance of effects so only correlations at $p< 0.01$ are considered. Table 7.25 shows that negative correlations were found between physical functioning at the 3 month follow up and age and GRACE risk scores. This indicates that older patients were more likely to have greater problems with physical functioning (such as bathing, dressing, climbing stairs, walking and carrying out moderate or vigorous activities). Patients with greater GRACE risk scores were also more likely to have problems with physical functioning. Older age was also positively correlated with the individual mental health component showing that older patients were likely to have better mental health. Other variables including gender, type of ACS, previous history of MI, and type of treatment received showed no significant associations with the component scales of the SF-36.

**Table 7.25 Associations between component scales of SF-36 and characteristics of patients at 3 months follow up**

| Components | Correlations for patients' characteristics at 3 months follow up | | | | | |
|---|---|---|---|---|---|---|
| | Age | Gender | GRACE risk score | Type of AMI | Previous MI | Type of treatment |
| **Physical functioning:** | | | | | | |
| r value | -0.261 | -0.083 | -0.284 | -0.087 | -0.370 | -0.082 |
| p-value | **0.006** | 0.394 | **0.003** | 0.370 | 0.706 | 0.398 |
| Limitations due to physical problems: | | | | | | |
| r value | -0.031 | 0.028 | -0.099 | 0.113 | -0.037 | -0.082 |
| p-value | 0.705 | 0.772 | 0.314 | 0.250 | 0.706 | 0.398 |
| Limitations due to emotional problems: | | | | | | |
| r value | 0.057 | 0.025 | 0.005 | -0.029 | 0.051 | -0.002 |
| p-value | 0.564 | 0.799 | 0.963 | 0.770 | 0.608 | 0.986 |
| Social functioning: | | | | | | |
| r value | 0.086 | 0.138 | 0.057 | 0.063 | -0.020 | -0.150 |
| p-value | 0.385 | 0.160 | 0.563 | 0.525 | 0.842 | 0.126 |
| **Mental health:** | | | | | | |
| r value | 0.295 | -0.049 | 0.283 | 0.100 | 0.047 | -0.038 |
| p-value | **0.002** | 0.611 | 0.013 | 0.303 | 0.627 | 0.693 |
| Vitality: | | | | | | |
| r value | 0.109 | -0.102 | 0.067 | 0.016 | 0.102 | -0.093 |
| p-value | 0.262 | 0.291 | 0.494 | 0.870 | 0.292 | 0.337 |
| Health perception: | | | | | | |
| r value | 0.172 | -0.024 | 0.110 | 0.048 | 0.002 | -0.160 |
| p-value | 0.073 | 0.804 | 0.251 | 0.619 | 0.983 | 0.096 |
| Pain: | | | | | | |
| r value | 0.007 | 0.006 | -0.064 | 0.112 | 0.050 | -0.048 |
| p-value | 0.947 | 0.955 | 0.518 | 0.261 | 0.613 | 0.629 |
| Summary scale of physical health status: | | | | | | |
| r value | -0.050 | 0.039 | -0.099 | 0.058 | 0.050 | -0.095 |
| p-value | 0.603 | 0.681 | 0.301 | 0.546 | 0.603 | 0.323 |
| Summary scale of mental health status: | | | | | | |
| r value | 0.117 | 0.037 | 0.068 | 0.025 | 0.059 | -0.048 |
| p-value | 0.223 | 0.702 | 0.479 | 0.797 | 0.542 | 0.617 |

*7.4.7.2: Associations between causal attribution factors and quality of life at 3 months*

Analyses were carried out to examine whether there were any associations between the individual scales of the SF36 and the three causal attribution factors (the mental state factor, the personal behaviour factor and the heredity factor). Because of multiple comparisons I adopted a more stringent criterion for the significance of effects so only correlations at $p < 0.01$ are considered. Table 7.26 shows that significant correlations were found between patients' baseline causal attributions to the mental state factor and their quality of life at 3 month follow up, specifically mental health (individual component) and mental health status (summary component). In each case, patients with stronger beliefs in the role of mental state had poorer quality of life.

**Table 7.26: Associations between quality of life (SF-36) and the causal attribution factors at 3 months follow up**

| Component of SF-36 at 3 months follow up | Causal attribution factor (tertiles) | Pearson correlation | p-value |
|---|---|---|---|
| Physical functioning: | Mental state | 0.046 | 0.642 |
| | Personal behaviour | 0.084 | 0.401 |
| | Heredity | -0.046 | 0.645 |
| Activity limitations due to | Mental state | -0.149 | 0.140 |
| physical problems: | Personal behaviour | -0.158 | 0.123 |
| | Heredity | -0.081 | 0.425 |
| Activity limitations due to | **Mental state** | -0.206 | 0.041 |
| emotional problems: | Personal behaviour | -0.060 | 0.558 |
| | Heredity | -0.162 | 0.112 |
| Social functioning: | Mental state | -0.222 | 0.026 |
| | Personal behaviour | -0.145 | 0.152 |
| | Heredity | -0.113 | 0.267 |
| **Mental health:** | **Mental state** | -0.279 | **0.004** |
| | Personal behaviour | -0.112 | 0.259 |
| | Heredity | -0.081 | 0.417 |
| Vitality: | Mental state | -0.194 | 0.050 |
| | Personal behaviour | -0.090 | 0.367 |
| | Heredity | -0.203 | 0.041 |
| Health perception: | Mental state | -0.060 | 0.541 |
| | Personal behaviour | -0.101 | 0.308 |
| | Heredity | -0.072 | 0.465 |
| Pain: | Mental state | -0.035 | 0.731 |
| | Personal behaviour | -0.214 | 0.035 |
| | Heredity | 0.017 | 0.866 |
| Physical health status: | Mental state | -0.080 | 0.417 |
| | Personal behaviour | -0.142 | 0.152 |
| | Heredity | -0.041 | 0.683 |
| **Mental health status:** | **Mental state** | -0.247 | **0.011** |
| | Personal behaviour | -0.114 | 0.252 |
| | Heredity | -0.161 | 0.104 |

Causal attribution factors which showed a significant association with the quality of life measures were then analysed further using linear regression, controlling for age, gender, baseline anxiety and GRACE risk scores. Results showed that mental health as assessed by the individual mental health scale at 3 months follow up was predicted by patients' causal attributions to mental state factor, independent of age and baseline line level of anxiety (see Table 7.27). Patients who believed at baseline that their heart problem was caused by their mental state had poorer mental health 3 months later. Likewise, patients who reported higher levels of anxiety at baseline were more likely to have poorer mental health 3 months later. Older patients however were likely to report better mental health at the 3 months follow up.

**Table 7.27: Predictors of mental health (individual scale) at 3 months follow up**

| | **Unstandardized B coefficients** | **95% Confidence Interval** | **p-value** |
|---|---|---|---|
| **Mental state factor** | -4.41 | -8.78 - -0.04 | **0.048** |
| **Age** | 6.44 | 0.60 – 12.29 | **0.031** |
| Gender | -6.15 | -15.05 – 2.75 | 0.173 |
| **Baseline level of anxiety** | -1.04 | -1.96 - -0.11 | **0.029** |
| GRACE risk score | 0.00 | -0.25 - 0.25 | 0.999 |

The mental state factor was the only significant predictor of the summary measure of mental health status at 3 months, since other variables such as age, gender, level of baseline anxiety and GRACE risk score did not show any significant associations (see Table 7.28). Patients' attribution of their heart problem to their mental state predicted poorer mental health measured by the summary mental health component, but did not significantly predict limitations due to emotional problems or social functioning at 3

months. Regression analyses controlling for age and gender did not show a significant association between social functioning, limitations due to emotional problems, vitality or mental state factor.

**Table 7.28: Predictors of mental health status at 3 months (summary measure)**

| | **Unstandardized B coefficients** | **95% Confidence Interval** | **p-value** |
|---|---|---|---|
| **Mental state factor** | -5.18 | -10.33 - - 0.03 | **0.049** |
| Age | 4.72 | -2.16 - 11.61 | 0.177 |
| Gender | 0.09 | -10.39 - 10.58 | 0.990 |
| Baseline level of anxiety | -0.80 | -1.89 - 0.30 | 0.152 |
| GRACE risk score | -0.08 | -0.38 - 0.21 | 0.579 |

*7.4.7.3:* ***Summary***

Analysis of the individual components of the SF36 showed that patients who had greater problems with physical functioning at the 3 month follow up were older and had greater GRACE scores. Older patients were, however, more likely to have better mental health. None of the other clinical variables showed a significant association with quality of life when controlled for age and gender.

Analysis of the 3 causal factors showed that patients' belief that their mental state caused their heart problem, independent of baseline level of anxiety and age, predicted poorer mental health (individual factor) at 3 months follow up. Poorer scores on the summary SF-36 mental health status scale were also predicted solely by the mental state factor.

## 7.5: Discussion

Results from this study support evidence from previous studies that causal attributions remain stable over time (Cameron et al, 2005; Gudmundsdottir et al, 2001; Weinman et al, 2000). The analyses above indicate that patients' causal attributions were more likely to predict emotional and social adjustment and quality of life after 3 months than changes in behaviour, since no associations were found between causal beliefs or the causal attribution factors and changes in relevant behaviours.

### 7.5.1: Availability for follow up at 3 months

In comparing the group of patients who were available to complete the 3 month follow up interview with those who were not, I found that patients who had a diagnosis of STEMI rather than UA/NSTEMI were more likely to participate. These patients may have been more convinced of the value of research studies into cardiac problems than patients who perceived themselves to have a less serious diagnosis. Patients with normal levels of serum cholesterol at baseline were also more likely to have been available for follow up at 3 months than patients who were hypercholoesterolemic. It is difficult to explain this and it may have been a chance finding. Patients who led a more sedentary life were more likely to be available to complete the 3 month telephone interview than those who exercised more than twice a week, possibly because they were more likely to be at home or near to a phone and, perhaps, less busy, making them more easily contactable. Patients with a large social network were more likely to participate than patients with a small social network. Patients with a larger social network are more likely to comply with medical advice generally (Brummett et al, 2005) and they may have viewed participation in a research study as more of a social responsibility or obligation than those with smaller social networks and thus been more willing to give their time. They may also be more sociable and thus more likely to want to answer their

phone. Patients who experienced moderate or high levels of stress in their relationship with their partner in the 4 weeks prior to their ACS were less likely to participate in the 3 month follow up than patients who experienced only mild levels of stress or none at all, possibly due to distractions related to domestics pressures. These findings could also have occurred by chance, since only 6 significant differences between participants and non-participants were found out of 29 comparisons. No differences were found, however, on key variables such as age, gender, socioeconomic factors or on the majority of risk factors.

### 7.5.2: Causal attributions and behaviour change at 3 months follow up

The findings relating causal beliefs with behaviour change and adherence advice are disappointing and do not support the study hypothesis which stated that adherence to medical advice including medication compliance, and life style changes, would be predicted by beliefs about causes, independently of severity and clinical treatment. These findings also contradict two previous prospective studies which found that causal attributions were important predictors of subsequent changes in health behaviour (Martin et al, 2005; Weinman et al, 2000). Martin et al (2005) reported that participants causal attributions to diet, exercise, smoking and stress predicted change in the related behaviour at the 3 month assessment. Weinman et al (2000) also reported a positive association between participants' causal attributions and subsequent behaviour change, although the follow up interval in this study was slightly longer at 6 months following the MI, rather than 3 months. These participants therefore had longer to process their experience of having an ACS and medical advice given to them and to incorporate behaviour changes into their lifestyle. A previous retrospective study of 81 male pre-operative coronary artery bypass patients also found that patients who had adopted

healthier lifestyle changes following diagnosis their heart problem endorsed causal attributions related to these specific changes (De Valle & Norman, 1992).

There are a number of reasons that may help to explain the present findings. Firstly, it may be that there is no association between causal attributions and subsequent behaviour change and previous findings have been chance effects. Secondly, it may indicate that the measures used in this study were inadequate. The measures were based on one simple self-report item per behaviour rather than more detailed assessment. For example, no specific assessments of diet (such as fat, sugar, fruit and fibre consumption), physical exercise (type, frequency, strenuous or not), or amount of alcohol consumed were made, as in previous studies (Weinman et al, 2000). The measure used to assess compliance with medication may also have been weak as it was a general measure assessing only whether patients took their prescribed medication daily and how often they missed a dose (Appendix 8). On the other hand, however, some studies have shown associations between behaviour change and causal beliefs using simple measures (De Valle & Norman, 1992). Thirdly, presentation bias may have been a present with some patients claiming that they were more adherent than they really were. Alternatively, some patients may have neglected to report specific changes because they seemed like a small changes which did not occur to them to mention, such as eating a few more portions of fruit and vegetables, walking to work more often as a form of exercise, developing a more relaxed attitude towards work commitments etc. Fourthly, there may have been some selection bias. For example, fewer physically active patients took part in the 3 month follow up assessment and this may have biased results concerning physical activity. A fifth reason may be that some patients may not have started or completed their cardiac rehabilitation programme at this point due to delays, holidays, availability of places on courses etc, and may therefore have not have

fully understand the importance of lifestyle changes in relation to their own heart problem. This will be assessed further in analyses at longer term follow up.

### 7.5.3: Cardiac rehabilitation attendance at 3 months

Findings of this study did not support the hypothesis that attendance at cardiac rehabilitation would be predicted by beliefs about causes, independently of severity and clinical treatment. Just over half of the participants attended a cardiac rehabilitation course but no associations were found between causal beliefs and attendance. It is perhaps not surprising that older patients were less likely to attend a cardiac rehabilitation programme or attend fewer sessions. This finding is supported by evidence from previous studies (Cooper et al, 1999; Melville et al, 1999). Older patients are more likely to suffer from co-morbidities and less likely to be as physically fit as younger patients. They may have found it more difficult to travel to hospital regularly. Older patients may also have considered it less important or been less willing to make behavioural changes at their stage of life. Patients who had suffered a previous MI were more likely to have attended a previous cardiac rehabilitation course and may have considered it unnecessary to repeat this. Misconceptions about cardiac rehabilitation, particularly the exercise content, may also have acted as a barrier to some patients and led them to believe that it was not appropriate for them (Cooper et al, 2005). Variations between hospitals may have meant that some courses more lengthy, offered a more limited choice of times, involved a narrower range of health care professionals and were less attractive to patients. No associations were found between attendance at cardiac rehabilitation and causal attributions, as supported by previous research findings (French et al, 2005c).

### 7.5.4: Causal attribution factors and mood at 3 months

Patients' causal attributions to mental state predicted anxiety after 3 months independently of baseline levels of anxiety. This may reflect a general outlook held by patients that heart problems are caused by mental processes. Patients who believed that their heart problem was caused by their mental state may therefore worry that if their own anxious thoughts and feelings caused their initial ACS, that this could cause further cardiac problems. This may then lead to increased levels of anxiety. This supports the idea that both cognitive and emotional representations have an influence on mood. The cognitive attribution therefore predicted psychological distress independently of the initial emotional impact of the event.

Patients' beliefs that heredity and genetic factors caused their heart problems at baseline also predicted higher levels of anxiety after 3 months. This may reflect a general outlook held by the patient that they were predestined or 'doomed' to suffer heart disease. This may be due to patients having investigated their family cardiac history more thoroughly during this time and discovered that other members of their family had also suffered from heart disease, or due to the reactions of family members/friends in emphasising 'bad news stories' concerning heart disease. These patients may have harboured anxiety that their family history of heart disease made them more vulnerable or 'doomed' to heart problems and viewed their experience of ACS as confirmation of this. This may also have created a barrier to behaviour change since if they believed that they were 'doomed' to heart disease they may not have seen the point of adopting healthier behaviours.

### 7.5.5: Quality of life at 3 months follow up

Poorer quality of life due to problems with physical functioning and greater GRACE risk scores were associated with older age at the 3 months follow up. This may be explained by the likelihood that older patients are not as fit as younger patients and are more likely to suffer other co-morbidities. It is surprising, however, that older patients had better mental health. Perhaps this was because they were more psychologically prepared to be affected by illness which some may have considered to be part of aging. Younger patients may have found it more shocking to suffer a heart attack at their age and thus found it more difficult to make psychological adjustments to such an 'off time' event in such a relatively short time. Younger patients may have faced more anxieties related to employment, personal finances and life plans than older patients who were more likely to be retired.

### 7.5.6: Causal attribution factors and quality of life at 3 months follow up

Findings presented in this thesis support of the hypothesis that quality of life at 3 months following hospital discharge would be predicted by causal beliefs, independently of treatment and clinical indices. Patients' causal attributions to their mental state were significantly associated with poorer quality of life after 3 months in relation to their mental health and predicted poorer summary mental health status.

# Chapter 8: Predictors of adherence, adjustment and quality of life 13 months after hospital admission for ACS

## 8.0: Introduction

Attempts were made to contact all patients in order to complete a telephone follow up interview and postal questionnaire 12 months after their hospital admission. Due to difficulties in contacting some patients, however, this interval became slightly longer than originally intended. The mean interval for contact by telephone to complete the follow up interview was 395 days ± 104 (56.4 weeks). In this chapter I will therefore report data collected after an average interval of 13 months rather than 12 months.

## 8.1: Comparison between patients who were available to complete follow up telephone interview after 13 months and those who were not

The characteristics of the study population of patients available to complete the telephone interview after 13 months (N = 213) was compared with those we were unable to contact (N = 56). Analyses using chi squared showed that the 2 groups differed significantly in terms of level of deprivation, yearly household income, marital status, level of stress in relationship with partner in the 4 weeks and 6 months prior to symptom onset, time of onset, season of onset, physical exercise activity, number of symptoms other than chest pain and number of non-pain symptoms (see Table 8.1). Gender also approached significance.

**Table 8.1: Comparison of characteristics of patients with telephone interview data at 13 months follow up (N = 213) and patients without (N = 56) in complete sample (N = 269)**

| | | No 13 month interview data N (%) | 13 month interview data completed N (%) | Difference p-value ($\chi^2$) |
|---|---|---|---|---|
| **Demographic factors** | | | | |
| Age: | <50 years | 14 (23.3) | 46 (76.7) | .233 |
| | 50-60 years | 22 (25.6) | 64 (74.4) | |
| | 60-70 years | 8 (13.8) | 50 (86.2) | |
| | > 70 years | 12 (18.5) | 53 (81.5) | |
| Gender: | Men | 49 (23.2) | 162 (76.8) | .064 |
| | Women | 7 (12.1) | 51 (87.9) | |
| Ethnicity: | White | 43 (19.6) | 176 (80.4) | .318 |
| | Other | 13 (26.0) | 37 (74.0) | |
| **Socio-economic factors** | | | | |
| Educational qualifications: | None | 31 (25.2) | 92 (74.8) | .116 |
| | Up to O'level | 11 (18.0) | 50 (82.0) | |
| | A'level + | 14 (16.5) | 71 (83.5) | |
| **Deprivation:** | Most deprived | 29 (35.4) | 53 (64.6) | **< .001** |
| | Moderately deprived | 13 (18.8) | 56 (81.2) | |
| | Least deprived | 14 (11.9) | 104 (88.1) | |
| **Income per year:** | <£20 k | 30 (25.9) | 86 (74.1) | **.033** |
| | £20-£40 k | 17 (21.5) | 62 (78.5) | |
| | >£40 k | 7 (11.7) | 53 (88.3) | |
| Occupational group: | Employed | 30 (20.8) | 114 (79.2) | .677 |
| | Unemployed | 7 (35.0) | 13 (65.0) | |
| | Retired | 19 (18.3) | 85 (81.7) | |
| **Social factors** | | | | |
| **Marital status:** | Not married | 26 (27.4) | 69 (72.6) | **.051** |
| | Married | 30 (17.2) | 144 (82.8) | |
| Social network: | Small | 9 (16.1) | 47 (83.9) | .528 |
| | Medium | 12 (13.5) | 77 (86.5) | |
| | Large | 9 (12.2) | 65 (87.8) | |
| **Partner stress within 4 weeks of ACS:** | None - Mild | 26 (15.8) | 139 (84.2) | **.010** |
| | Moderate - Very | 11 (35.5) | 20 (64.5) | |
| **Partner stress within 6 months of ACS:** | None - mild | 27 (15.8) | 144 (84.2) | **.002** |
| | Moderate - very | 11 (40.7) | 16 (59.3) | |

(Continued on next page)

(Table 8.1 continued)

| | | No 13 month interview data N (%) | | 13 month interview data completed N (%) | | Difference p-value ($\chi^2$) |
|---|---|---|---|---|---|---|
| **Proximal factors** | | | | | | |
| **Time of symptom onset:** | | | | | | |
| Midnight – 0600 hrs | | 16 | (27.6) | 42 | (72.4) | **.025** |
| 0600 hrs – midday | | 16 | (28.6) | 69 | (81.2) | |
| Midday – 1800 hrs | | 9 | (18.8) | 66 | (88.0) | |
| 1800 hrs - midnight | | 15 | (12.0) | 36 | (70.6) | |
| Day of onset: | Week day | 40 | (22.5) | 138 | (77.5) | .350 |
| | Weekend | 16 | (17.6) | 75 | (82.4) | |
| **Season:** | Jan - Mar | 24 | (32.0) | 51 | (68.0) | **.039** |
| | Apr – June | 11 | (15.5) | 60 | (84.5) | |
| | July - Sept | 13 | (19.1) | 55 | (80.9) | |
| | Oct - Dec | 8 | (14.5) | 47 | (85.5) | |
| Presence of bystander: | Absent | 18 | (21.2) | 67 | (78.8) | .451 |
| | Present | 17 | (16.8) | 84 | (83.2) | |
| **Risk factors** | | | | | | |
| Previous MI: | No | 48 | (20.0) | 192 | (80.0) | .786 |
| | Yes | 6 | (22.2) | 21 | (77.8) | |
| Hypertension: | No | 32 | (21.6) | 116 | (78.4) | .720 |
| | Yes | 24 | (19.8) | 97 | (80.2) | |
| Hypercholesterolemia: | No | 24 | (18.0) | 109 | (82.0) | .372 |
| | Yes | 29 | (22.5) | 100 | (77.5) | |
| Diabetes: | No | 47 | (20.2) | 186 | (79.8) | .507 |
| | Yes | 9 | (25.0) | 27 | (75.0) | |
| Smoker: | Non-smoker | 9 | (15.8) | 48 | (84.2) | .185 |
| | Ex-smoker | 20 | (19.8) | 81 | (80.2) | |
| | Smoker | 27 | (24.3) | 84 | (75.7) | |
| Alcohol intake: | Non-drinker | 21 | (21.2) | 78 | (78.8) | .831 |
| | Drinker | 34 | (20.1) | 135 | (79.9) | |
| **Physical exercise:** | | | | | | |
| Inactive | | 29 | (17.0) | 142 | (83.0) | **.023** |
| Low(<2x per week) | | 12 | (22.6) | 41 | (77.4) | |
| High (>2x per week) | | 14 | (32.6) | 29 | (67.4) | |
| **Clinical presentation** | | | | | | |
| Premonitory symptoms: | No | 28 | (19.2) | 118 | (80.8) | .471 |
| | Yes | 28 | (22.8) | 95 | (77.2) | |
| Type of ACS: | | | | | | |
| UA / NSTEMI | | 20 | (25.0) | 60 | (75.0) | .273 |
| STEMI | | 36 | (19.0) | 153 | (81.0) | |
| Intensity of pain | >6 | 6 | (13.3) | 39 | (86.7) | .783 |
| | 6-8 | 10 | (21.7) | 36 | (78.3) | |
| | 8 -10 | 7 | (30.4) | 51 | (87.5) | |
| **Number of non-chest pain symptoms:** | none | 20 | (27.0) | 54 | (73.0) | **.009** |
| | 1 - 3 | 30 | (23.4) | 98 | (76.0) | |
| | 4 - 8 | 6 | (9.0) | 61 | (91.0) | |
| **Number of non-pain symptoms:** | | | | | | |
| | None | 26 | (30.2) | 60 | (69.8) | **.001** |
| | 1 – 2 | 24 | (22.0) | 85 | (78.0) | |
| | 3 – 6 | 6 | (8.1) | 68 | (91.9) | |
| **Psychological factors** | | | | | | |
| Attribution to heart attack: | | | | | | |
| | No | 46 | (23.0) | 154 | (77.0) | .147 |
| | Yes | 10 | (14.7) | 58 | (85.3) | |
| Cardiac denial of impact: | Low | 8 | (10.4) | 69 | (89.6) | .401 |
| ( tertiles) | Middle | 11 | (16.2) | 57 | (83.8) | |
| | High | 11 | (15.1) | 62 | (84.9) | |

Variables that showed a significant difference between the 2 groups were then analysed using logistic regression (see Table 8.2) adjusting for age and gender. Patients who were the least deprived were 4 times more likely to complete the 13 month follow up than the most deprived group. Similarly, patients with a yearly household income of greater than £40 000 were more than 4 time more likely to complete the follow up than patients with a yearly household income of less than £20 000. Patients who were married were more likely to complete the 13 month follow up than those who were not married. Patients who exercised more than twice per week were less likely to complete the follow up interview than patients who did no exercise. Patients who had reported experiencing either none or low levels of stress in their relationship with their partner in the 4 weeks and 6 months prior to symptom onset were more likely to complete the follow up than patients who had reported experiencing moderate to high levels of stress in their relationship. Whether or not patients were available to complete the follow up interview may therefore have been related to the stability of their lives, in terms of their financial and emotional situation.

There were also significant differences between the 2 groups in terms of the timing of their symptoms. Patients whose symptoms had started during the afternoon (between midday and 6pm) were more than twice as likely to complete the 13 month follow up than patients whose symptoms started during the night (midnight to 6am). Patients whose symptoms started in the months of January to March were less likely to be available for follow up than patients whose symptoms started at other times. Patients who suffered from a greater number of symptoms at onset were also more likely to complete the follow up at 13 months than patients who had experienced fewer symptoms.

**Table 8.2 Results of logistic regression comparing patients who provided telephone follow up data after 13 months with those who did not.**

| | | Odds ratio ( 95% Confidence Interval) adjusted for age and gender | | p-value |
|---|---|---|---|---|
| **Deprivation:** | Most deprived | 1 | | |
| | Moderately deprived | 2.31 | (1.08 – 4.94) | **.031** |
| | Least deprived | 4.03 | (1.97 – 8.36) | **< 0.001** |
| **Household income per year:** | <£20 k | 1 | | |
| | £20-£40 k | 1.64 | (0.80 – 3.36) | .177 |
| | >£40 k | 4.06 | (1.57 – 10.51) | **.004** |
| **Marital status:** | Not married | 1 | | |
| | Married | 2.58 | (1.03 – 6.44) | **.042** |
| **Partner stress within 4 weeks of ACS:** | | | | |
| | None - mild | 1 | | |
| | Moderate - very | 0.35 | (0.15 – 0.83) | **.017** |
| **Partner stress within 6 months of ACS:** | | | | |
| | None - Mild | 1 | | |
| | Moderate - Very | 0.37 | (0.16 – 0.86) | **.021** |
| **Time of symptom onset:** | | | | |
| | Midnight – 0600 hrs | 1 | | |
| | 0600 hrs – Midday | 1.61 | (0.72 – 3.60) | .243 |
| | **Midday – 1800 hrs** | 2.82 | (1.14 – 7.00) | **.026** |
| | 1800 hrs - Midnight | 0.90 | (0.39 – 2.08) | .798 |
| **Season:** | Jan - Mar | 1 | | |
| | **Apr – June** | 2.56 | (1.14 – 5.78) | **.023** |
| | July - Sept | 2.05 | (0.94 – 4.48) | .073 |
| | **Oct – Dec** | 2.88 | (1.17 – 7.09) | **.022** |
| **Physical exercise:** | | | | |
| | Inactive | 1 | | |
| | Low(<2x per week) | 0.71 | (0.33 – 1.53) | .383 |
| | **High (>2x per week)** | 0.44 | (0.20 – 0.93) | **.032** |
| **Number of non-chest pain symptoms:** | | | | |
| | None | 1 | | |
| | 1 – 3 | 1.15 | (0.59 – 2.23) | .680 |
| | **4 - 8** | 3.52 | (1.31 – 9.48) | **.013** |
| **Number of non-pain symptoms:** | None | 1 | | |
| | 1 – 2 | 1.45 | (0.76 – 2.80) | .262 |
| | **3 – 6** | 4.69 | (1.80 – 12.25) | **.002** |

Comparison of levels of depression and anxiety at baseline between patients who were available to complete the telephone follow up at 13 months and those who were not were carried out using t-tests (see Table 8.3). Patients who were not available to complete the follow up at 13 months had significantly higher baseline mean scores on the BDI than those who completed the follow up ($p = 0.032$). There was no significant difference between groups in their levels of anxiety at baseline, however, and whether or not they completed the 13 month follow up.

**Table 8.3: Comparison of means for level of depression and anxiety at baseline between patients who provided 13 month follow up data and those who did not**

| | N | Mean (SD) | *p-value |
|---|---|---|---|
| **Depression (baseline total BDI score)** | | | |
| No 13 month follow up data | 30 | 11.58 (7.74) | **.032** |
| 13 month follow up data provided | 179 | 8.37 (7.48) | |
| **Anxiety (baseline total HADS anxiety score)** | | | |
| No 13 month follow up data | 29 | 5.62 (4.32) | .793 |
| 13 month follow up data provided | 185 | 5.83 (3.86) | |

(* p-value from t-test of difference between means)

The 3 causal attribution factors of the group of patients who provided 13 month follow up data were also compared with the group of patients who did not provide 13 month follow up data using independent samples t-tests (see Table 8.4). There were no significant differences between the 2 groups.

**Table 8.4: Comparison of means for the 3 causal factors at baseline between patients who did and did not provide 13 month follow up data**

| | N | Mean (SD) | *p-value |
|---|---|---|---|
| **Mental state factor** | | | |
| No 13 month follow up data | 25 | 3.22 (2.44) | .491 |
| 13 month follow up data provided | 141 | 2.85 (2.47) | |
| **Personal behaviour factor** | | | |
| No 13 month follow up data | 25 | 3.76 (2.71) | .692 |
| 13 month follow up data provided | 141 | 3.57 (2.14) | |
| **Heredity factor** | | | |
| No 13 month follow up data | 25 | 2.60 (3.10) | .399 |
| 13 month follow up data provided | 140 | 3.40 (3.27) | |

(* p-value from t-test of difference between means)

In summary, patients who did not complete the 13 month telephone follow up interview were more likely to be more socio-economically deprived. They were more likely to have experienced either moderate or high levels of stress in their relationship with their partner in the 4 weeks and 6 months prior to the onset of their symptoms and were less likely to be married. They were more likely to experience the onset of their symptoms between the months on January to March, and their symptoms were more likely to start during the night or morning than in the afternoon. Patients who were not available to complete the 13 month follow up interview were also likely to have suffered fewer symptoms (other than chest pain) at onset than patients who were available to complete the follow up. Patients who were not available for follow up were also significantly more depressed at baseline than those who completed the follow up, although there was no difference in levels of anxiety. The 2 groups did not differ in age, gender, ethnicity, or causal attributions during hospitalisation. Possible explanations of these effects and their significance for the representativeness of findings are detailed in section 8.8.1.

## 8.2: Stability of causal attributions over 13 months

Of the patients who returned their baseline questionnaires, 165 completed the measure of causal beliefs. The same measure in the 13 months follow up questionnaire was completed by 140 patients. The stability of this measure was analysed using correlations and paired t-tests. Firstly, the mean scores were compared for each of the 3 factors (mental state, personal behaviour and heredity) at the two time periods. Paired t-tests showed no significant difference between means at baseline and thirteen months later for any of the factors. Correlations between the two time periods were significant for all three factors (see Table 8.5). Internal reliability (Cronbach alpha) for the three causal attribution factors were as follows; mental state 0.77, personal behaviour 0.49 and heredity 0.78. . Test-retest studies were also performed and showed average anova intraclass correlation co-efficients for each of the three factors as follows; mental state factor 0.78, personal behaviour factor 0.78, and heredity factor 0.83.

**Table 8.5: Results of t-tests and correlations showing the stability of the 3 factors over the 13 months follow up period.**

| Factor | N | Mean (SD) | p-value | Pearson's r for baseline & 13 month correlation | p-value |
|---|---|---|---|---|---|
| **Mental state factor:** | | | | | |
| At 13 month follow up | 139 | 2.47 (2.36) | .090 | 0.65 | **<0 .001** |
| Baseline | 139 | 2.75 (2.40) | | | |
| **Personal behaviour factor:** | 140 | 3.60 (1.93) | .669 | 0.64 | **< 0.001** |
| At 13 month follow up | 140 | 3.54 (2.11) | | | |
| Baseline | | | | | |
| **Heredity factor:** | | | | | |
| At 13 month follow up | 138 | 3.55 (3.44) | .159 | 0.71 | **< 0.001** |
| Baseline | 138 | 3.24 (3.29) | | | |

Secondly, the stability of each of the 16 items in the measure was tested using correlations. The scores at baseline and 13 months later for all of the 16 items were highly correlated ($p < 0.05$). Paired t-tests showed no significant differences between mean scores for each of the individual items at baseline and 13 months later for all items, excepting individual items attributing causal beliefs to smoking (mean difference between scores at 13 months and baseline of 0.09, $t = -2.00$, $p = 0.05$) as shown in Table 8.6. Patients' beliefs that smoking caused their heart problem were stronger at 13 months than at baseline. Moreover, the test-retest correlations were all significant, indicating reasonable stability over this time period. The highest correlation was for smoking (0.80) and the lowest was for poor diet (0.20). The diet effect was the only one not to be significant at $p<0.001$. Evidence from these analyses suggests therefore that the measures used to examine causal attributions in this study were stable over the 13 month follow up period. Although 1 item showed a change, the overall factor scores were stable.

**Table 8.6: T-test and individual correlations for the causal belief individual items at baseline and 13 months follow up**

| Factor | Time point | N | Mean | SD | t | p-value for t-test | Pearsons' r | p - value of r |
|---|---|---|---|---|---|---|---|---|
| Heredity | Baseline | 136 | 0.71 | 0.79 | -1.07 | 0.29 | 0.75 | <0.001 |
| | 13 months | 136 | 0.76 | 0.79 | | | | |
| Smoking | Baseline | 141 | 0.80 | 0.85 | -2.00 | 0.05 | 0.80 | <0.001 |
| | 13 months | 141 | 0.89 | 0.86 | | | | |
| Other medical problems | Baseline | 136 | 0.49 | 0.66 | 1.71 | 0.09 | 0.35 | <0.001 |
| | 13 months | 136 | 0.39 | 0.57 | | | | |
| Stress | Baseline | 138 | 0.90 | 0.78 | 0.24 | 0.81 | 0.57 | <0.001 |
| | 13 months | 138 | 0.88 | 0.76 | | | | |
| Overweight | Baseline | 138 | 0.46 | 0.61 | 0.88 | 0.38 | 0.54 | <0.001 |
| | 13 months | 138 | 0.41 | 0.60 | | | | |
| Blood pressure | Baseline | 137 | 0.77 | 0.78 | -0.93 | 0.36 | 0.65 | <0.001 |
| | 13 months | 137 | 0.82 | 0.76 | | | | |
| Poor diet | Baseline | 101 | 0.50 | 0.63 | -0.99 | 0.33 | 0.20 | 0.04 |
| | 13 months | 101 | 0.57 | 0.65 | | | | |
| Overexertion | Baseline | 102 | 0.43 | 0.64 | -0.27 | 0.79 | 0.38 | <0.001 |
| | 13 months | 102 | 0.45 | 0.67 | | | | |
| Bad luck | Baseline | 99 | 0.74 | 0.82 | 0.24 | 0.82 | 0.44 | <0.001 |
| | 13 months | 99 | 0.71 | 0.81 | | | | |
| Poor medical care | Baseline | 100 | 0.13 | 0.37 | -1.42 | 0.16 | 0.54 | <0.001 |
| | 13 months | 100 | 0.19 | 0.49 | | | | |
| Lack of exercise | Baseline | 102 | 0.53 | 0.66 | 0.16 | 0.87 | 0.56 | <0.001 |
| | 13 months | 102 | 0.52 | 0.67 | | | | |
| Tiredness | Baseline | 101 | 0.47 | 0.63 | -1.16 | 0.25 | 0.41 | <0.001 |
| | 13 months | 101 | 0.54 | 0.64 | | | | |
| Genetic factors | Baseline | 101 | 0.58 | 0.63 | <0.001 | 1.00 | 0.46 | <0.001 |
| | 13 months | 101 | 0.58 | 0.67 | | | | |
| State of mind | Baseline | 99 | 0.45 | 0.63 | 1.63 | 0.11 | 0.62 | <0.001 |
| | 13 months | 99 | 0.36 | 0.65 | | | | |
| Working too hard | Baseline | 100 | 0.42 | 0.64 | <0.001 | 1.00 | 0.58 | <0.001 |
| | 13 months | 100 | 0.42 | 0.64 | | | | |
| Germ/virus | Baseline | 55 | 0.07 | 0.26 | -0.44 | 0.66 | 0.54 | <0.001 |
| | 13 months | 55 | 0.09 | 0.35 | | | | |

## 8.3: Baseline causal attributions and behaviour change at 13 months

All patients had received advice during their hospital stay or cardiac rehabilitation classes to follow specific recommendations such as to quit smoking (if applicable), to undertake regular exercise, to maintain a healthy body weight according to current recommendations, to manage stress more effectively, to limit their alcohol intake to within recommended limits and to eat a healthy diet. All patients had received advice about how and when to take prescribed medication. Each of the above behaviours was analysed at 13 months follow up to identify changes which may be associated with relevant items included in the causal beliefs questionnaire and which were endorsed by patients at baseline. In addition, the adherence index (sum of 5 behaviours) was calculated. The mean was 2.21 ± 1.8.

### 8.3.1: Smoking at 13 months follow up

All patients who were smokers at baseline were advised to stop smoking, either by coronary care nurses, medical staff or cardiac rehabilitation nurses. Although 69.1% of baseline smokers who attributed their heart problem to smoking said that they had quit by the 13 month telephone follow up interview, there was no association between stopping smoking and baseline beliefs held by smokers that smoking caused their heart problem after 13 months (see Table 8.7).

**Table 8.7: Association between causal attribution to smoking at baseline and smoking status of baseline smokers at 13 months follow up**

| | **Belief at baseline that smoking caused the heart problem** | | | |
|---|---|---|---|---|
| **Smoking at 3 months follow up** | **No** N (%) | **Maybe** N (%) | **Yes** N (%) | **p-value** |
| No | 5 (11.9) | 16 (38.1) | 21 (50.0) | 0.456 |
| Yes | 1 (6.7) | 5 (33.3) | 9 (60.0) | |

### 8.3.2: Healthy diet at 13 months follow up

All patients were advised to adopt a healthy diet as explained in section 7.3.2. They were either given a leaflet containing these recommendations or advised by cardiac rehabilitation nurses. Analyses showed that there was no association between patients' baseline beliefs that poor diet had caused their heart problem and whether they said they had changed their diet at the 13 month telephone follow up interview (see Table 8.8).

**Table 8.8: Association between causal attribution to poor diet at baseline and dietary change at 13 months follow up**

| | Belief at baseline that poor diet caused the heart problem | | | |
|---|---|---|---|---|
| **Diet change at 13 months follow up** | **No** N (%) | **Maybe** N (%) | **Yes** N (%) | **p-value** |
| No | 32 (62.7) | 16 (31.4) | 3 (5.9) | 0.136 |
| Yes | 44 (50.0) | 35 (39.8) | 9 (10.2) | |

### 8.3.3: Physical activity at 13 months

All patients were informed of the importance of regular exercise in maintaining their health and were advised to exercise regularly within the limits of their own individual capabilities. Analyses showed that there was no association between patients' belief that lack of exercise had caused their heart problem at baseline and whether they said that they had changed their exercise behaviour after 13 months (see Table 8.9).

**Table 8.9: Association between causal attribution to lack of exercise at baseline and change in exercise behaviour at 13 months follow up**

| | Belief at baseline that lack of exercise caused the heart problem | | | |
|---|---|---|---|---|
| **Change in exercise behaviour at 13 months follow up** | **No** N (%) | **Maybe** N (%) | **Yes** N (%) | **p-value** |
| No | 29 (58.0) | 17 (34.0) | 4 (8.0) | 0.920 |
| Yes | 52 (59.1) | 29 (33.0) | 7 (8.0) | |

### 8.3.4: Control of body weight at 13 months

All patients were advised by coronary care nurses, medical staff or rehabilitation nurses to maintain their body weight within recommended limits (British Heart Foundation, 2005b). Those who were overweight were advised to reduce their weight. No association was found between weight change reported by patients at the 13 month follow up interview and the baseline belief that being overweight had caused their heart problem (see Table 8.10). Neither was there any association between patients' belief that poor diet caused their heart problem at baseline and change in body weight at the 13 month telephone follow up interview, or patients' belief that lack of physical exercise caused their heart problem and change in body weight after 13 months.

**Table 8.10: Association between causal attribution to being overweight at baseline and reported change in body weight at 13 months follow up in complete sample**

| | **Belief at baseline that being overweight caused the heart problem** | | | |
|---|---|---|---|---|
| **Change in body weight at 13 months follow up** | **No** N (%) | **Maybe** N (%) | **Yes** N (%) | **p-value** |
| No | 53 (64.6) | 24 (29.3) | 5 (6.1) | 0.332 |
| Yes | 32 (55.2) | 22 (37.9) | 4 (6.9) | |

These analyses were repeated so that only patients who were overweight or obese at baseline were included. Again, there was no association between belief that being overweight caused the heart problem at baseline and change in body weight at the 13 month follow up interview (see Table 8.11).

**Table 8.11: Association between causal attribution to being overweight at baseline and reported change in body weight after 13 months in overweight or obese patients only**

| | Belief at baseline that being overweight caused the heart problem | | | |
|---|---|---|---|---|
| **Change in body weight at 13 months follow up** | **No** N (%) | **Maybe** N (%) | **Yes** N (%) | **p-value** |
| No | 21 (44.7) | 21 (44.7) | 5 (10.6) | 0.774 |
| Yes | 16 (40.0) | 20 (50.0) | 4 (10.0) | |

### 8.3.5: Stress at 13 months

All patients were given advice concerning the importance of stress management, either via patient information leaflets or cardiac rehabilitation nurses. Analyses showed that there was no association between patients' belief at baseline that stress caused their heart problem and whether they had made any changes to the way they responded to stress or to reduce the stress in their life at the 13 months follow up (see Table 8.12).

**Table 8.12: Association between causal attribution to stress at baseline and whether patient had tried to reduce the stress in their life after 13 months**

| | Belief at baseline that being overweight caused the heart problem | | | |
|---|---|---|---|---|
| **Change in stress at 13 months follow up** | **No** N (%) | **Maybe** N (%) | **Yes** N (%) | **p-value** |
| No | 31 (36.0) | 32 (37.2) | 23 (26.7) | 0.997 |
| Yes | 19 (35.2) | 21 (38.9) | 14 (25.9) | |

These data were then analysed to see if patients' baseline causal beliefs that over exertion caused their heart problem was associated with self reported changes in behaviour to reduce the stress in their lives by the 13 months post discharge follow up. No association was found (see Table 8.13).

**Table 8.13: Association between baseline causal attribution to over exertion and whether patients had reduced the amount of stress in their lives after 13 months**

| Change in stress at 13 months follow up | Belief at baseline that over exertion caused the heart problem<br>No<br>N (%) | Maybe<br>N (%) | Yes<br>N (%) | p-value |
|---|---|---|---|---|
| No | 56 (65.9) | 18 (21.2) | 11 (12.9) | 0.539 |
| Yes | 31 (56.4) | 18 (32.7) | 6 (10.9) | |

Data were also analysed to see if patients' baseline attributions to state of mind being a cause of the heart problem was associated with any reported change in behaviour to reduce stress after 13 months. These analyses revealed no significant associations between the attribution to state of mind and stress behaviour change (see Table 8.14).

**Table 8.14: Association between causal attribution to state of mind at baseline and whether patient had tried to reduce their stress in their life after 13 months**

| Change in stress at 13 months follow up | Belief at baseline that state of mind caused the heart problem<br>No<br>N (%) | Maybe<br>N (%) | Yes<br>N (%) | p-value |
|---|---|---|---|---|
| No | 53 (62.4) | 24 (28.2) | 8 (9.4) | 0.738 |
| Yes | 31 (58.5) | 17 (32.1) | 5 (9.4) | |

In the same way, data were analysed to investigate any possible associations with the patients' belief at baseline that working too hard had caused the heart problem and behaviour changes aimed at managing stress reported at the 13 month follow up telephone interview. Again no significant association was found (see Table 8.15). This association also remained insignificant when the sample was limited to include only patients who were in employment at baseline

**Table 8.15: Association between baseline causal attribution to working too hard and whether patient had reduced the amount of stress in their life after 13 months**

| Change in stress at 13 months follow up | Belief at baseline that working too hard caused the heart problem<br>No<br>N (%) | Maybe<br>N (%) | Yes<br>N (%) | p-value |
|---|---|---|---|---|
| No | 58 (68.2) | 19 (22.4) | 8 (9.4) | 0.768 |
| Yes | 33 (61.1) | 18 (33.3) | 3 (5.6) | |

## 8.4: Relationships between causal attribution factors and behaviour changes after 13 months

Patients' adherence to medical advice concerning lifestyle changes and prescribed medication (as described above) was also examined in relation to the causal attribution factors; "mental state", "personal behaviour" and "heredity". At 13 months, 150 (71.4%) patients claimed to be adherent to medicatin, while 28.6% admitted to some problems of adherence.

### 8.4.1: Relationship between mental state factor and adherence at 13 months

As shown in Table 8.16, no significant associations were found between patients' scores on the mental state factor (reported as tertiles) and the self reported behaviours assessed using the telephone interview 13 months following the diagnosis of ACS.

As stated above, although all patients who were smokers at baseline were advised to stop smoking, no association was found between beliefs held by smokers at baseline that their mental state caused their heart problem and whether they had quit smoking or not 13 months later. Likewise patients' scores on the mental state factor as a cause of their heart problem were not associated changes in behaviour regarding diet, exercise, body weight stress management or adherence to medication.

**Table 8.16: Association between mental state factor and self reported lifestyle changes at 13 months follow up**

| Behaviour change at 13 months follow up | | Attribution to mental state factor (tertiles) Low N (%) | Medium N (%) | High N (%) | p-value |
|---|---|---|---|---|---|
| Smoking behaviour *(among baseline smokers only):* | | | | | |
| | Continue to smoke | 5 (26.3) | 7 (36.8) | 7 (36.8) | 0.375 |
| | Quit | 16 (41.0) | 11 (28.2) | 12 (30.8) | |
| Diet change: | No | 18 (35.7) | 15 (29.4) | 18 (35.3) | 0.873 |
| | Yes | 31 (37.3) | 23 (27.7) | 29 (34.9) | |
| Exercise change: | No | 19 (38.0) | 14 (28.0) | 17 (34.0) | 0.792 |
| | Yes | 30 (35.7) | 24 (28.6) | 30 (35.7) | |
| Change in body weight: | No | 30 (37.0) | 25 (30.9) | 26 (32.1) | 0.562 |
| | Yes | 19 (35.8) | 13 (24.5) | 21 (39.6) | |
| Change in body weight: *(among patients with baseline BMI>25):* | | | | | |
| | No | 17 (37.0) | 13 (28.3) | 16 (34.8) | 0.589 |
| | Yes | 13 (36.1) | 5 (19.4) | 16 (44.4) | |
| Change in stress management: | No | 30 (36.6) | 26 (31.7) | 26 (31.7) | 0.562 |
| | Yes | 19 (36.5) | 12 (23.1) | 21 (40.4) | |
| Medication adherence at 3 months: | | | | | |
| | Non-adherent | 12 (30.0) | 13 (32.5) | 15 (37.5) | 0.385 |
| | Adherent | 37 (40.7) | 23 (25.3) | 42 (34.1) | |

Data was also analysed as continuous data using linear regression based on the adherence index (dependent variable). No significant association between mental state factor and the adherence index was found (B = 0.002, C.I. = -0.32 to 0.31, p = 0.992).

### 8.4.2: Relationship between personal behaviour factor and adherence at 13 months follow up

No significant associations were found between patients' scores on the personal behaviour factor (reported as tertiles) and the self reported behaviours assessed using the telephone interview 13 months following the diagnosis of ACS (see Table 8.17).

**Table 8.17: Association between personal behaviour factor and self reported lifestyle changes at 13 months follow up**

| Behaviour change at 13 months follow up | | Attribution to personal behaviour factor (tertiles) Low N (%) | Medium N (%) | High N (%) | p-value |
|---|---|---|---|---|---|
| Smoking behaviour *(among baseline smokers only):* | | | | | |
| | Continue to smoke | 7 (35.0) | 4 (20.0) | 9 (45.0) | 0.419 |
| | Quit | 12 (29.3) | 22 (48.8) | 9 (22.0) | |
| Diet change: | No | 20 (38.5) | 18 (34.6) | 14 (36.9) | 0.510 |
| | Yes | 30 (33.7) | 31 (34.8) | 28 (31.5) | |
| Exercise change: | No | 18 (35.3) | 19 (37.3) | 14 (27.5) | 0.810 |
| | Yes | 32 (35.6) | 30 (33.3) | 28 (31.1) | |
| Change in body weight: | No | 31 (37.3) | 30 (36.1) | 22 (26.5) | 0.364 |
| | Yes | 19 (32.8) | 19 (32.8) | 20 (34.5) | |
| Change in body weight: *(among patients with baseline BMI>25):* | | | | | |
| | No | 12 (25.5) | 15 (31.9) | 20 (42.6) | 0.800 |
| | Yes | 12 (30.0) | 11 (27.5) | 17 (42.5) | |
| Change in stress management: | No | 32 (37.2) | 29 (33.7) | 25 (29.1) | 0.651 |
| | Yes | 18 (32.7) | 20 (36.4) | 17 (30.9) | |
| Medication adherence at 3 months: | | | | | |
| | Non-adherent | 12 (27.9) | 16 (37.2) | 15 (34.9) | 0.189 |
| | Adherent | 38 (40.0) | 31 (32.6) | 26 (27.4) | |

Using linear regression analyses, no significant association was found between personal behaviour factor and the adherence index (B = 0.007, C.I. = -0.14 to 0.12, p = 0.917).

### 8.4.3: Relationship between heredity factor and adherence at 13 months follow up

As shown in Table 8.18 no significant associations were found between patients' scores on the heredity factor (reported as tertiles) at baseline and the self reported behaviours assessed at the 13 month follow up, except for smoking. Further analyses using logistic regression showed that patients who were smokers at baseline and who had scores in the

highest tertile for believing that their heart problem was caused by the heredity factor were significantly more likely (OR 4.52, C.I. 1.24 – 16.40, p = 0.022) than patients with scores in the lowest tertile to have quit smoking after 13 months.

**Table 8.18: Association between heredity factor and self reported lifestyle changes at 13 months follow up.**

| | | Attribution to heredity factor (tertiles) | | | |
|---|---|---|---|---|---|
| **Behaviour change at 13 months follow up** | | **Low** N (%) | **Medium** N (%) | **High** N (%) | **p-value** |
| **Smoking behaviour** *(among baseline smokers only):* | | | | | |
| | Continue to smoke | 12 (60.0) | 3 (15.0) | 5 (25.0) | **.025** |
| | Quit | 13 (32.5) | 5 (12.5) | 22 (55.0) | |
| Diet change: | No | 24 (47.1) | 7 (13.7) | 20 (39.2) | .363 |
| | Yes | 33 (37.1) | 17 (19.1) | 39 (43.8) | |
| Exercise change: | No | 22 (43.1) | 7 (13.7) | 22 (43.1) | .889 |
| | Yes | 35 (39.3) | 17 (19.1) | 37 (41.6) | |
| Change in body weight: | No | 32 (39.0) | 11 (13.4) | 39 (47.6) | .274 |
| | Yes | 25 (43.1) | 6 (22.4) | 20 (34.5) | |
| Change in body weight *(among patients with baseline BMI>25):* | | | | | |
| | No | 16 (34.0) | 5 (10.6) | 26 (55.3) | .088 |
| | Yes | 18 (45.0) | 9 (22.5) | 14 (32.5) | |
| Change in stress management: | No | 36 (42.4) | 11 (12.9) | 38 (44.7) | .882 |
| | Yes | 21 (38.2) | 13 (23.6) | 21 (38.2) | |
| Medication adherence at 3 months: | | | | | |
| | Non-adherent | 18 (41.9) | 5 (11.6) | 20 (46.5) | .931 |
| | Adherent | 36 (38.3) | 19 (20.2) | 39 (41.5) | |

Using linear regression analyses, no significant association was found between heredity factor and the adherence index (B = -0.01, C.I. = -0.08 to 0.08, p = 0.980).

## 8.5: Attendance at cardiac rehabilitation programmes at 13 months

As explained in section 7.5, attendance at rehabilitation programmes was measured as whether patients said that they attended any sessions at all or not; and the number of sessions attended. Data regarding the former (attendance) was available for all participants who completed the 13 month telephone interview (N = 213) except 3 which were missing, and data for the latter (number of sessions) was available for 140 participants since 73 patients did not specify the number of sessions attended.

When attendance was calculated as a percentage of the total course, results had changed very little from the 3 months follow up results. Only 22.3% of patients who had attended a cardiac rehabilitation programme at 13 months follow up had attended all sessions constituting the course, and 45.5% of the total had attended half the course or less. For a few patients cardiac rehabilitation may not have been suitable and they may not have been invited due to co-morbidities, some patients did not want to participate and did not think it would be helpful to them or thought that the travelling to and from hospital would be too much for them.

No correlations were found between either attendance at a cardiac rehabilitation programme or number of sessions attended and the following variables; gender, level of education, type of ACS (STEMI or NSTEMI/UA), or type of treatment (medication, coronary bypass grafts or angioplasty) or whether the patient had previously experienced an MI. Age, however, was negatively correlated with the whether patients attended any sessions at all so that older patients were less likely to attend cardiac rehabilitation classes ($r = -0.16$, $p = 0.023$). GRACE risk score was also negatively correlated with whether or not patients attended rehabilitation classes at all ($r = -0.15$, p

= 0.033). Patients who had suffered a previous MI were also less likely to attend a cardiac rehabilitation programme than patients who had not suffered a previous MI ($r = -0.14$, $p = 0.048$). Younger patients with lower GRACE scores and no previous MI were therefore more likely to attend the rehabilitation programmes. This pattern is similar to the 3 month results detailed in Chapter 7.

No significant correlations were found between cardiac rehabilitation programme attendance or the number of sessions attended, and any of the three causal attribution factors (mental state, personal behaviour or heredity). Partial correlations were computed, controlling for GRACE risk score, previous history of MI, and age (see Table 8.19).

**Table 8.19 Correlations between causal attribution factors and patients' attendance at a cardiac rehabilitation programme and number of sessions attended at 13 months follow up.**

| Factor | Pearson correlation | p-value * |
|---|---|---|
| **Mental state factor** (tertiles) | | |
| Attendance at cardiac rehabilitation programme | 0.045 | 0.618 |
| Number of sessions attended | 0.004 | 0.968 |
| **Personal behaviour factor** (tertiles) | | |
| Attendance at cardiac rehabilitation programme | -0.065 | 0.470 |
| Number of sessions attended | -0.054 | 0.562 |
| **Heredity factor** | | |
| Attendance at cardiac rehabilitation programme | 0.117 | 0.192 |
| Number of sessions attended | 0.172 | 0.062 |

* Adjusted for GRACE risk score, previous MI, age & gender.

### 8.5.1: Summary

This study found no evidence to support the hypothesis that patients' baseline causal attributions were related to adherence to medical advice, specifically changes in behaviour regarding lifestyle (smoking, diet, exercise, body weight, stress management), adherence to medication as prescribed or attendance at a rehabilitation programme.

## 8.6: Causal attributions and psychological adjustment to ACS after 13 months

All patients who were available for follow up after 13 months were asked to complete the measures of mood state again, the BDI and the HADS anxiety (appendices 11 and 12). Data was then examined to determine whether there was a relationship between mood state and the causal attribution factors.

### 8.6.1 Description of sample

Of the 213 participants who were followed up by telephone interview after 13 months, 177 patients returned the 13 month follow up questionnaire pack having completed the BDI, and 185 patients completed the HADS anxiety scale. A total of 35.6% of patients who responded reported a high level of depression (BDI score $\geq$10) while 30.8% of patients reported high levels of anxiety (HADS anxiety score $\geq$8). This indicates that the rates of psychological morbidity remained substantial more than a year after ACS.

### 8.6.2 Relationship between baseline causal attributions and mood state at 13 months follow up

The relationship between patients' baseline causal attributions and mood state 13 month after their ACS was examined using product-moment correlations with the three causal attribution factors; the mental state factor, personal behaviour factor and heredity factor. There was a significant positive correlation between the mental state factor and self reported level of anxiety at 13 months follow up ($r = 0.29$, $p = 0.001$). There was also a significant positive correlation between mental state factor and level of depression ($r = 0.21$, $p = 0.018$). There were no significant correlations between the personal behaviour factor and heredity factors and levels of depression or anxiety after 13 months.

### 8.6.3 Causal attributions to mental state and mood at 13 months follow up

A linear regression was conducted to determine whether the association between causal attribution to mental state factor and 13 month level of anxiety was independent of cofactors. Table 8.20 shows that patients' baseline belief that mental state was a causal factor predicted levels of anxiety after 13 months ($B = 1.20$, C.I. = 0.344 – 2.05, $p = 0.006$). Independently, patients' level of anxiety at baseline also predicted level of anxiety 13 months later ($B = 0.97$, C.I. = 0.45 – 1.90, $p = 0.040$). Age, gender and GRACE risk score were not significantly related to level of anxiety after 13 months.

**Table 8.20: Mental state factor as a predictor of anxiety at 13 months follow up**

| | Unstandardized B coefficients | 95% Confidence Interval | p-value |
|---|---|---|---|
| **Mental state factor** | 1.20 | 0.34 – 2.05 | **0.006** |
| **Baseline level of anxiety** | 0.97 | 0.05 – 1.90 | **0.040** |
| Age | -0.94 | -2.04 – 0.170 | 0.096 |
| Gender | 0.98 | -0.80 – 2.76 | 0.278 |
| GRACE risk score | -0.02 | -0.03 – 0.06 | 0.534 |

A linear regression was also conducted to determine whether the patients' baseline causal attribution to mental state and level of depression after 13 months was independent of cofactors. Table 8.21 shows that patients' baseline belief that mental state was a causal factor did not predict levels of depression after 13 months, after baseline depression had been taken into account. The baseline level of depression itself was a predictor of depression 13 months later (B = 0.78, C.I. = 0.62 – 0.94, $p < 0.001$). Patients' level of depression at 13 months follow up was not related to age, gender or GRACE risk score.

**Table 8.21: Mental state factor as a predictor of depression at 13 months**

| | Unstandardized B coefficients | 95% Confidence Interval | p-value |
|---|---|---|---|
| Mental state factor | 0.12 | -1.28 – 1.50 | 0.879 |
| **Baseline level of depression** | 0.78 | 0.62 – 0.94 | **<0.001** |
| Age | -0.72 | -2.53 – 1.09 | 0.432 |
| Gender | 0.65 | -2.24 – 3.54 | 0.658 |
| GRACE risk score | -0.003 | -0.08 – 0.08 | 0.933 |

### 8.6.4: Summary

Patients with greater levels of anxiety at baseline were more likely to report greater levels of anxiety at the 13 months follow up. Patients who had stronger beliefs that their heart problem was caused by the mental state factor at baseline also had higher levels of anxiety at the 13 month follow up, independent of baseline anxiety, age, gender and GRACE scores. Levels of depression after 13 months were predicted by baseline level of depression but not by baseline causal attributions, and this was independent of age, gender and GRACE scores.

## 8.7: Causal attribution factors and quality of life at 13 months

Between 181-191 patients completed the 13 month follow up measure (SF-36) assessing self reported quality of life. Mean scores for the 8 factors and 2 summary components are shown in Table 8.22. As in the 3 month follow up, poorer self rated quality of life was particularly marked in relation to limitations in activity due to physical problems and vitality.

**Table 8.22: Mean scores of self rated quality of life (SF-36) at 13 months follow up**

| Component scales of SF-36 | N | *Mean (SD) |
|---|---|---|
| Physical functioning | 189 | 68.02 (± 27.53) |
| **Activity limitations due to physical problems** | 190 | **56.10 (± 43.06)** |
| Activity limitations due to emotional problems | 188 | 69.33 (±40.48) |
| Social functioning | 181 | 81.77 (±22.58) |
| Mental health | 190 | 72.29 (±20.83) |
| **Vitality** | 189 | **56.61 (±21.60)** |
| Health perception | 187 | 60.90 (±22.80) |
| Pain | 187 | 72.96 ((±26.57) |
| Summary scale of physical health status | 191 | 64.27 (±26.03) |
| Summary scale of mental health status | 191 | 69.30 (±23.81) |

*Scores range from 0 (very poor quality of life) to 100 (excellent quality of life).

**8.7.1: Quality of life and cardiological and demographic variables at 13 months**

As for the 3 month follow up analyses, correlations were used to determine whether there was an association between each of the 8 individual scales and 2 summary components of quality of life measured by the SF36 and other variables such as age, gender, GRACE risk score, type of ACS ( STEMI or NSTEMI / UA), previous MI or type of treatment received. Because of multiple comparisons I adopted a more stringent criterion for the significance of effects so only correlations at $p< 0.01$ are considered. Table 8.23 shows that negative correlations were found between physical functioning at the 13 month follow up and age, gender and GRACE risk score. Older patients were more likely to have greater problems with physical functioning. Male gender was associated with better physical functioning. Patients with better physical functioning had lower GRACE scores. Other variables including type of ACS, previous history of MI, and type of treatment received showed no significant associations with the summary components of the SF-36.

**Table 8.23: Associations between component scales of SF-36 and characteristics of patients at 13 months follow up**

| Components | Correlations for patients characteristics at 13 months follow up | | | | | |
|---|---|---|---|---|---|---|
| | Age | Gender | GRACE risk score | Type of AMI | Previous MI | Type of treatment |
| **Physical functioning:** | | | | | | |
| r value | -0.228 | -0.226 | -0.287 | 0.110 | -0.055 | 0.026 |
| p-value | **0.002** | **0.002** | **<0.001** | 0.131 | 0.449 | 0.722 |
| Activity limitations due to physical problems: | | | | | | |
| r value | -0.110 | -0.110 | -0.139 | 0.038 | 0.045 | 0.067 |
| p-value | 0.132 | 0.130 | 0.055 | 0.598 | 0.540 | 0.361 |
| Activity limitations due to emotional problems: | | | | | | |
| r value | -0.088 | -0.140 | -0.110 | 0.069 | 0.068 | 0.024 |
| p-value | 0.229 | 0.055 | 0.135 | 0.343 | 0.353 | 0.746 |
| Social functioning: | | | | | | |
| r value | -0.015 | -0.018 | -0.100 | -0.017 | -0.004 | 0.016 |
| p-value | 0.845 | 0.812 | 0.181 | 0.820 | 0.955 | 0.834 |
| Mental health: | | | | | | |
| r value | 0.169 | -0.022 | 0.116 | -0.005 | 0.013 | -0.057 |
| p-value | 0.020 | 0.761 | 0.110 | 0.942 | 0.862 | 0.437 |
| Vitality: | | | | | | |
| r value | 0.033 | -0.065 | -0.024 | 0.081 | -0.006 | -0.023 |
| p-value | 0.654 | 0.375 | 0.743 | 0.269 | 0.931 | 0.754 |
| General health perception: | | | | | | |
| r value | 0.170 | -0.002 | 0.062 | 0.112 | -0.070 | -0.063 |
| p-value | 0.020 | 0.973 | 0.401 | 0.128 | 0.343 | 0.394 |
| Pain: | | | | | | |
| r value | 0.068 | -0.052 | -0.103 | 0.042 | -0.002 | 0.001 |
| p-value | 0.353 | 0.476 | 0.159 | 0.566 | 0.983 | 0.990 |
| Summary component of physical health status: | | | | | | |
| r value | -0.088 | -0.112 | -0.148 | 0.072 | -0.009 | 0.029 |
| p-value | 0.227 | 0.123 | 0.041 | 0.321 | 0.899 | 0.697 |
| Summary component of mental health status: | | | | | | |
| r value | 0.003 | -0.070 | -0.047 | 0.033 | 0.047 | -0.002 |
| p-value | 0.966 | 0.338 | 0.515 | 0.652 | 0.520 | 0.982 |

### 8.7.2: Associations between causal attribution factors and quality of life

Analyses were carried out to examine whether there were any associations between the individual scales of the SF36 and the three causal attribution factors (the mental state factor, the personal behaviour factor and the heredity factor). Because of multiple comparisons I adopted a more stringent criterion for the significance of effects so only correlations at $p < 0.01$ are considered. Significant correlations were found between patients' baseline causal attributions to the mental state factor and their quality of life at 13 months follow up, specifically activity limitations due to emotional problems and mental health status, indicated by scores both on the individual item mental health scale and the summary mental health measure. In each case, patients with stronger baseline beliefs that their mental state caused their heart problem had poorer quality of life (see Table 8.24).

**Table 8.24 Associations between quality of life (SF-36) and the causal attribution factors at 13 months follow up**

| Component of SF-36 at 13 months follow up | Causal attribution factor (tertiles) | Pearson correlation | p-value |
|---|---|---|---|
| Physical functioning: | Mental state | -0.054 | 0.548 |
| | Personal behaviour | -0.021 | 0.802 |
| | Heredity | 0.024 | 0.782 |
| Limitations due to physical problems | Mental state | -0.053 | 0.552 |
| | Personal behaviour | 0.001 | 0.992 |
| | Heredity | -0.044 | 0.607 |
| **Activity limitations due to Emotional problems** | **Mental state** | -0.228 | **0.010** |
| | Personal behaviour | -0.064 | 0.460 |
| | Heredity | -0.068 | 0.431 |
| Social functioning | Mental state | -0.085 | 0.353 |
| | Personal behaviour | -0.123 | 0.157 |
| | Heredity | -0.047 | 0.587 |
| **Mental health** | **Mental state** | -0.271 | **0.002** |
| | Personal behaviour | -0.119 | 0.161 |
| | Heredity | -0.055 | 0.521 |
| Vitality | Mental state | -0.163 | 0.066 |
| | Personal behaviour | -0.049 | 0.566 |
| | Heredity | -0.087 | 0.310 |
| General health perception | Mental state | -0.138 | 0.124 |
| | Personal behaviour | -0.092 | 0.281 |
| | Heredity | -0.059 | 0.497 |
| Pain | Mental state | -0.115 | 0.201 |
| | Personal behaviour | -0.109 | 0.206 |
| | Heredity | -0.054 | 0.534 |
| Summary physical health status | Mental state | -0.094 | 0.290 |
| | Personal behaviour | -0.044 | 0.607 |
| | Heredity | -0.031 | 0.719 |
| **Summary mental health status** | **Mental state** | -0.223 | **0.011** |
| | Personal behaviour | -0.089 | 0.293 |
| | Heredity | -0.060 | 0.485 |

Causal attribution factors which showed a significant association with the components of the quality of life measures (including activity limitations due to emotional problems, the individual mental health scale and the summary component mental health status) were then analysed further using linear regression, controlling for age, gender, baseline anxiety and GRACE risk scores. Results showed that activity limitations due to emotional problems were predicted after 13 months by scores on the mental state factor at baseline independently of covariates (see Table 8.25). Other variables such as age, gender, baseline anxiety and GRACE risk score showed no significant associations.

**Table 8.25: Predictors of activity limitations due to emotional problems after 13 months**

| | **Unstandardized B coefficients** | **95% Confidence Interval** | **p-value** |
|---|---|---|---|
| **Mental state factor** | -10.48 | -19.16 - -1.79 | **0.018** |
| Age | 5.71 | -5.92 - 17.34 | 0.333 |
| Gender | -13.40 | -31.99 - 5.20 | 0.156 |
| Baseline level of anxiety | -0.79 | -2.73 - 1.15 | 0.420 |
| GRACE risk score | -0.30 | -0.81 - 0.21 | 0.240 |

Patients' causal attributions to mental state factor also predicted poorer mental health as assessed by the individual mental health factor after 13 months. Independently, older age predicted better mental health after 13 months (see Table 8.26). Other variables such as gender and GRACE risk score did not show any significant associations.

**Table 8.26: Predictors of mental health after 13 months (individual component)**

| | Unstandardized B coefficients | 95% Confidence Interval | p-value |
|---|---|---|---|
| **Mental state factor** | -5.36 | -9.62 - -1.10 | **0.014** |
| **Age** | 8.15 | 2.58 – 13.71 | **0.004** |
| Gender | -3.67 | -12.71 – 5.38 | 0.424 |
| Baseline level of anxiety | -0.93 | -1.88 - 0.02 | 0.055 |
| GRACE risk score | -0.20 | -0.44 - -0.05 | 0.114 |

Results for the association between patients' baseline attributions to mental state factor and the summary component of mental health status were similar to the single item scale (see Table 8.27). Patients' baseline belief that their mental state caused their heart problem predicted poorer mental health status after 13months, independent of age. Other variables such as gender, baseline level of anxiety and GRACE risk score did not show a significant association with the summary mental health status scale.

**Table 8.27: Predictors of mental health status at 13 months follow up (summary measure)**

| | Unstandardized B coefficients | 95% Confidence Interval | p-value |
|---|---|---|---|
| **Mental state factor** | -5.31 | -10.14 - -0.48 | **0.031** |
| **Age** | 6.77 | -0.46 – 13.08 | **0.036** |
| Gender | -6.56 | -16.82 – 3.69 | 0.208 |
| Baseline level of anxiety | -0.66 | -1.73 – 0.42 | 0.230 |
| GRACE risk score | -0.24 | -0.51 – 0.04 | 0.096 |

### 8.7.3: Summary

Analysis of the individual components of the SF36 were similar to the results at 3 months, and showed that patients who had greater problems with physical functioning at the 13 month follow up were more likely to be older, female and to have greater GRACE scores. Activity limitations due to physical problems and summary physical health status were likely to be greater in patients with greater GRACE risk scores. Older patients were more likely to have better mental health and better general health perceptions. Patients with greater activity limitations due to emotional problems were more likely to be female. None of the other variables analysed showed a significant association with quality of life when controlled for age and gender.

Analysis of the 3 causal factors showed that patients' belief that their mental state caused their heart problem, predicted greater activity limitations due to emotional problems. Patients' attributions to their mental state predicted poorer mental health both by the individual mental health scale and the summary component mental health status at 13 months follow up, independent of age. Other cofactors including gender, baseline anxiety, and clinical risk profile as indexed by GRACE scores did not show any significant associations. These results therefore supported the hypothesis that quality of life 13 months following hospital discharge would be predicted by causal beliefs, independently of treatment and clinical indices.

## 8.8: Discussion

Results from the 13 months follow up analyses were similar to the 3 month analyses. As supported by evidence from previous studies, the causal attributions remained stable over the 13 month time period (Cameron et al, 2005; Gudmundsdottir et al, 2001; Weinman et al, 2000). Once again, no associations were found between causal beliefs or the 3 causal attribution factors and changes in relevant behaviours, with the exception

of smoking. Patients who were smokers at baseline were more likely to have quit 13 months later if they had strong beliefs that their heart problem was caused by heredity. It is difficult to explain why patients might attribute the cause of their heart problems to heredity rather than personal behaviour, however, since this was the only significant effect among numerous comparisons it is possible that this was a chance finding. Associations were found with mood and quality of life. This suggests that patients' causal attributions were more likely to predict emotional and social adjustment and quality of life after 13 months than changes in behaviour.

### 8.8.1: Availability for follow up after 13 months

Comparison of patients who were available for follow up with those who were not showed that patients who were from a deprived background, who had a low yearly income, who were not married, were physically more active and who experienced higher levels of stress in their relationships were less likely to participate in follow up. The relationship between SES as indexed by deprivation and participation reflects a general problem in health research, which is that less affluent individuals are less likely to agree to take part in the first place, and are harder to retain in studies. Patients who had a sedentary lifestyle were more likely to participate in follow up than those who had a higher level of physical activity, possibly because they were at home more and easier to contact. Non-attenders seemed to have a higher level of instability in their lives which may have made contacting them more difficult.

There were significant differences between the 2 groups in terms of the timing of their symptoms. Patients whose symptoms had started during the afternoon were more likely to complete the 13 month follow up than patients whose symptoms started during the night. Patients whose symptoms started in the months of January to March were less

likely to be available for follow up than patients whose symptoms started at other times. It is difficult to explain this. Some patients may have been on holiday, or simply felt more lethargic or depressed and unwilling to participate due to the winter season, the post-Christmas period or other worries such as the payment of income taxes at the end of January. Some patients may not have wanted to be reminded of the anniversary of their heart problem and this may have had a stronger effect during these months.
It is interesting that patients who suffered from a greater number of symptoms at onset were also more likely to complete the follow up at 13 months than patients who had experienced fewer symptoms. These patients may have had more vivid memories of the event and taken their heart condition more seriously, and thus have been more convinced of the importance of research and more willing to complete the study. It is particularly important that non-completers were more depressed on average than those who completed the study. This may have reduced the chances of observing associations between factors measured in hospital and later depression, whereas associations with anxiety were found.

### 8.8.2: Causal beliefs and behaviour change at 13 months

As was the case with the 3 month data (chapter 7), the findings relating causal beliefs with behaviour change and adherence advice does not generally support evidence from previous prospective studies, with the exception of the association between causal belief in hereditary factors and smoking, which found that causal attributions were important predictors of subsequent changes in health behaviour (Martin et al, 2005; Weinman et al, 2000). Later re-analysis of data reported in one of these studies has recently shown that once pre-MI behaviour was controlled for, there was no evidence that patients' attributions were associated changes in lifestyle behaviours over 6 months (French et al, 2005a). A previous systematic review of 65 studies investigating causal attributions

following serious unexpected negative events, also reported no association between attributions and outcomes in 76% cases (Hall et al, 2003) so it may be that the pattern of results presented here is rather typical. The only significant association involved smoking behaviour, as discussed above. Evidence reported in this thesis does not therefore generally support the hypothesis that adherence to medical advice, including attendance at cardiac rehabilitation, medication compliance, and lifestyle changes, would be predicted by beliefs about causes independently of severity and clinical treatment.

Nevertheless, there are other possible explanations as to why initial causal beliefs were not related to behaviour change, similar to those outlined in Chapter 7. The measures were based on one simple self-report item per behaviour rather than more detailed assessment and may have been inadequate. More specific assessments may have revealed behavioural changes that were over looked. The measure used to assess compliance with medication may also have been too general a measure. There will almost certainly have been some presentation bias with some patients claiming that they were more adherent than they really were, and some selection bias in relation to physical exercise.

### 8.8.3: Cardiac rehabilitation attendance after 13 months

Results reported for the follow up at 13 months regarding attendance at a cardiac rehabilitation programme were consistent with the three month analysis. As reported in previous studies (Cooper et al, 1999; Melville et al, 1999; Sotile & Miller, 1998), older patients were both less likely to attend a cardiac rehabilitation programme and attended fewer sessions when they did participate. As discussed in chapter 7, there are a number of reasons that may help to explain this. Older patients are more likely to suffer from

co-morbidities and less likely to be as physically fit as younger patients. They may find it difficult to travel to hospital regularly, and may be less willing to change lifestyle habits at their stage of life. Misconceptions about cardiac rehabilitation may have acted as a barrier to some patients (Cooper et al, 2005). Those who had suffered a previous MI were also less likely to attend and may have considered it unnecessary if they had attended a previous cardiac rehabilitation course.

### 8.8.4 Mood and attribution to mental state after 13months

Patients who attributed the cause of their heart problem to their mental state at baseline were more likely to have higher levels of anxiety 13 months later, independently of their baseline level of anxiety. Patients who attributed their heart problems to their mental state rather than to other causes may be more aware their thoughts, moods and feelings. This may lead them to adopt a more negative outlook on life. Attribution to the mental state factor may therefore reflect a general outlook that ones' mental processes influence the development of CHD. Patients with strong beliefs that their mental state caused their heart problem may find it distressing to think they are responsible, and feel constantly fearful that they will precipitate new cardiac problems by their thoughts and feelings, thus leading to yet higher levels of distress and anxiety. The SF-36 scale measuring problems with work or other limitations to daily activities as a result of emotional problems may reflect this way of thinking.

Patients' attributions to mental state predicted anxiety after 13 months independently of baseline levels of anxiety. This result therefore supports the dual influence of cognitive and emotional representations, the emotional part being anxiety, and the cognitive part being the attribution. The cognitive attribution therefore predicted psychological distress independently of the initial emotional impact of the event. If this is the case,

these patients may have been less inclined to follow medical advice to change certain behaviours because they considered their mental state to be the main cause of their heart problem rather than their behaviour, and may also have found it more difficult to make psychological adjustments following their ACS.

Higher levels of family or work stress at baseline were associated with patients' baseline beliefs that mental state caused their heart problem. Some patients who attributed their heart problems to their mental state may have blamed themselves or others for their heart problem, from overworking or from being exposed to high levels of stress at work or at home. Blaming others is generally thought to be maladaptive and to affect emotional adjustment, morbidity and quality of life post ACS (Affleck et al, 1987; Tennen & Affleck, 1990) all of which may lead to increased anxiety. This is partly supported by a recent review which found that attributions of blame following serious unexpected negative events were generally associated with poorer outcomes, although results were not consistent (Hall et al, 2003).

### 8.8.5: Quality of life after 13 months

The mean scores for each of the 8 individual scales and the 2 summary components of the SF-36 were greater at 13 months follow up (shown in Table 8.21) than at 3 months (shown in Table 7.23) indicating that overall patients reported that their quality of life had improved during this period. The largest changes in quality of life were due to marked improvements in activity limitations due to physical problems, social functioning and physical functioning. This is probably explained by the gradual recovery of physical fitness over the course of 13 months, and, for some patients, return to work. This is in contrast to the lack of improvement in mental health, vitality and pain. Since greater levels of anxiety after 13 months were predicted by patients' belief

that their mental state caused their heart problem, and the causal attributions were found to be stable over time, this may have acted as an obstacle to patients' adapting to their ACS and making the necessary psychological adjustments.

Older patients were more likely to have poorer physical functioning at the 13 months follow up, and this may reflect their higher baseline GRACE scores. As older patients are less likely to be as fit as younger patients and more likely to suffer other co-morbidities this may not be surprising. Evidence from a previous study also reported a significant difference between younger and older MI patients in physical functioning after 5 months (Brink et al, 2002). A Finnish study also reported that older patients perceived fewer symptoms of CHD and expected a short duration of illness, where as, although younger patients expected their CHD to be more controllable, they also expected a long duration of illness (Aalto et al, 2005). Older patients attributed their CHD less often to stress and more often to life-course. A study by Day et al (2005) also found that patients who endorsed at least one negative emotion as a cause of their heart problem were significantly younger, more depressed and more anxious than those who did not endorse any negative emotions.

Women were also significantly more likely to have greater problems than men due to physical and emotional problems. A study by Brink et al (2002) also found the men had significantly better physical functioning, less bodily pain and better social functioning than women after 5 months. This may be related to age in that female patients are more likely to be older, to have more severe heart problems and to be widowed or to live alone. Aalto et al (2005) also reported that women perceived their CHD as less controllable and experienced more CHD related symptoms. They were also more likely to think their illness was caused by stress or heredity then male participants. Patients

with better physical functioning also had lower GRACE scores and this is probably related to the likely absence of other co-morbidites.

Patients' causal attributions to the mental state factor were significantly associated with poorer quality of life after 13 months. This was due to greater emotional problems and poorer mental health (reflected both in the single item and summary mental health status scale) after 13 months. Causal attribution of MI to stress responses (such as worry or feeling nervous) and blaming others has been shown to be predictive of greater morbidity in 8-year survivors (Affleck et al, 1987). These factors may interfere with emotional and psychological adjustment and impact on quality of life and suggest that patients' beliefs about the cause of their ACS were more likely to predict emotional and social adjustment, and quality of life after 13 months than changes in their lifestyle or behaviour. Evidence reported in this study therefore supports the hypothesis that quality of life 13 months after diagnosis of ACS would be predicted by causal beliefs, independently of treatment and clinical indices.

## 8.9: Limitations of this study

### 8.9.1: Representativeness of the present sample

There are several limitations to this study concerning the sample population. Fewer physically active patients took part in the follow up at both 3 month and 13 months and this may have biased the findings concerning adherence to advice and physical activity, and in terms of quality of life. Patients who had higher levels of depression at baseline were also less likely to complete the follow up after 13 months and this may have reduced chances of observing associations between factors measured in hospital and later depression. Patients who were more socioeconomically deprived were less likely

to complete the 13 months follow up and this may therefore reduce the generalizability of the findings to less deprived population groups.

### 8.9.2: Measures

The telephone questionnaire used to follow patients up at 3 months and 13 months was designed to be a brief measure assessing changes in behaviour and was based on a similar measure used previously by Ziegelstein et al (2000). The aim of making the telephone interview as brief as possible was so as not to over burden participants, who were also asked to complete a number measure included in the postal questionnaire. However, this interview may have been rather too brief and missed some important behaviour changes. The interview assessed only whether patients said they had implemented advice given to them regarding specific behaviours (such as eating a healthy diet, maintaining a healthy weight etc) and did not quantify these changes. Patients may have varied in their self assessment of the degree to which they had implemented advice, those who had a higher personal threshold of what represented behavioural change may have under-reported small important changes.

Although patients were asked about their level of physical exercise, smoking and alcohol intake prior to their ACS, it was not possible to quantify behavioural changes using information from the telephone follow up interview, since detailed information was not collected. More precise measures such as the Dietary Instrument of Nutrition Education (DINE) (Roe et al, 1994) allows more detailed assessment of dietary intake, the International Physical Activity Questionnaire (IPAQ) (Craig et al, 2003), or the Medication Adherence Report (Kravitz et al, 1993) may have allowed more accurate assessment but would also have added considerably to the research load place on

patients and may have reduced participant retention. It will be necessary to weigh up the benefits and costs of using more detailed measures in future research.

The content and quality of rehabilitation programmes may not have been consistent across the hospitals involved in the study. The number of sessions constituting the courses varied from one to twelve sessions and no information was collected about the quality of information provided. Due to the number of participating centres and variations in recording, it was not possible to verify attendance using rehabilitation programme attendance records and this may have been subject to over reporting bias. There may, therefore, have been variations in quality and quantity of information provided to patients that affected their understanding of treatment and subsequent adherence, however the self reported attendance rate in this study is comparable to previous research (French et al, 2005b; Lane et al, 2001).

### 8.9.3: Timing

Patients' baseline levels of anxiety and depression were measured within the first 5 days of hospital admission and it is possible that levels of distress vary over this time period in relation to hospital admission or discharge (Brink et al, 2002). Limitations due to timing will be discussed in more detail in chapter 9.6.2.

# Chapter 9: Final discussion

## 9.0: Introduction

In this thesis, I have presented a study which attempted to explore patients' understanding of heart disease, and investigated relationships with decisions to seek help following the onset of cardiac symptoms and adjustment up to 13 months following diagnosis of ACS. Two important clinical problems were examined; patients' delay in seeking treatment following the onset of cardiac symptoms; and problems with adjustment following hospital discharge including adherence to medical advice and recommended lifestyle changes, psychological adjustment and quality of life. The specific findings of the study have been discussed in detail in the relevant chapters and will not be repeated here. This final chapter will offer a broad discussion of the most important findings and whether the specific aims of the study have been met. It will discuss whether the findings support the hypotheses, and how these results fit in with previous research. It will also discuss the strengths and limitations of this study, and suggest areas where this research may be developed further.

## 9.1: Aim 1 - To investigate the socio-demographic and psychological factors which predict delay in contacting medical help following the onset of symptoms of ACS.

Whilst many studies investigating pre-hospital delay have focussed on specific factors such as socio-demographic factors or psychological variables, this study investigated a broad range of factors and their relationship with pre-hospital delay. This allowed examination of a series of factors simultaneously. Unlike many studies, I questioned patients in detail about their experience, so was able to divide total pre-hospital delay period into 2 component phases, patient decision delay and home to hospital delay. In

line with previous research, patient decision delay accounted for 60% of the overall total pre-hospital delay, and home to hospital delay accounted (40%) (GISSI, 1995; Schmidt & Borsch, 1990). This method of dividing total pre-hospital delay into two phases for analysis yielded some useful information and revealed that different factors predicted shorter delay in one phase but not the other. Short decision delay was predicted largely by social and psychological factors, such as being married, having a bystander present, attribution of symptoms to heart attack and low cardiac denial, while short home to hospital delay is influenced more by factors related to clinical presentation such as type of ACS and symptoms, and younger age. Contacting an ambulance as the initial call for help predicted short delays in both phases. Socio-economic factors, such as education and deprivation, predicted only very short total pre-hospital delay, and were not associated with decision time.

The results reported in this study largely supported the first hypothesis that shorter patients' decision time in seeking help would be associated with demographic and psychosocial variables including younger age, male gender, greater social support, higher socio-economic status, attribution of symptoms to heart attack and low cardiac denial. The social context in which the symptoms occurred was also important since time of onset on a week day and within work hours, and the presence of a bystander also predicted shorter delays. Shorter patient decision delay was not associated with socio-economic factors, however. While patients' attribution of symptoms predicted both pre-hospital delay and decision time, socioeconomic variables predicted short total pre-hospital delay but not decision time. This suggests that socioeconomic variables are not involved in the formation of patients' cognitive representation of their illness, but that they play a role in pre-hospital delay during the sequence of events that occur after the patient has made the decision to call for help. Most published literature has focussed on

decision delay and there is little previous research that has specifically investigated the home to hospital phase. It is possible that patients from a more deprived background face more barriers in trying to access emergency health care as discussed earlier (Ell et al, 1994; Sheifer et al, 2000). Patients who are more deprived are more likely to be socially isolated, may therefore be less likely to have a bystander available to make the decision to seek help on their behalf. They may also face more negative reactions from relatives, friends or co-workers than patients from higher socio economic backgrounds, perhaps a greater reluctance to involve official agencies or greater denial in those whose advice is sought due to financial implications, employment worries, implications for childcare etc. There is clearly a case for further research into this area and targeting interventions aimed at reducing barriers to accessing medical care for deprived patients.

Although pre-hospital delay is significantly shorter if patients call an ambulance, less than half of the patients in this study chose to call an ambulance in the first instance. Pattenden et al (2002) reported a concern among patients about wasting NHS time and resources, especially ambulances. They did not want to bother the doctor and felt guilty about calling for help. In my study, younger age was a predictor of short home to hospital delay, and this would imply that older patients were less likely to use the emergency ambulance services once they had decided to seek help. These patients presumably either called their GP or NHS Direct, or a relative/friend. Ruston et al (1998) found that it was a common perception that the correct action was first to phone the GP, who would then call the ambulance. Public information campaigns to call an ambulance in the event of acute cardiac symptoms have had very limited success. It seems these common public misconceptions and fear of wasting medical resources are hard to shift and require greater investment and more innovative methods to get the message across.

Low scores on the cardiac denial of impact scale also predicted both short total pre-hospital delays and short patient decision times. This supports the study by O'Carroll (2001). It seems likely that this is linked with other predictors of short decision time, including attribution of symptoms to a heart attack, having an STEMI rather than a NSTEMI or UA, having symptoms which start in the afternoon, having a bystander present at symptom onset and being married. Patients were more likely to have a short decision time if they recognised their symptoms as being those of a heart attack and had a more severe type of heart attack. The pattern of symptoms that patients with a more severe heart attacks experience may be different from those experienced by patients with a milder form of ACS. As a result, patients with more severe symptoms may become convinced that they should contact medical help more quickly, and be less likely to deny the seriousness of their symptoms.

It is probably also more likely that a bystander will be present at symptom onset if patients are married and at home (as most were), if their symptoms start in the afternoon and if they have a large social network. The bystander may help to reinforce patients' belief that their symptoms are serious and assist them in making the decision to seek medical help promptly. These patients may then feel more confident that their symptoms warrant calling an ambulance. Services such as NHS Direct may be useful for patients seeking to confirm the seriousness of their symptoms and may play a similar role to that of the bystander for patients who are alone at symptom onset.

It has been suggested that cardiac denial protects patients against negative emotions such as anxiety and depression, therefore high levels of cardiac denial following the onset of cardiac symptoms may reflect the use of a coping strategy to reduce anxiety and fear (Wielgosz et al, 1988; Wielgosz & Nolan, 1991; Sarantidis et al, 1997). Few

studies have specifically investigated the influence of denial among patients with ACS during the pre-hospital delay period and, since patients who had low levels of cardiac denial were significantly more likely to have short delays, this may be an area to explore further in future research.

Both shorter pre-hospital delays and patient decision times were predicted by attribution of symptoms to a heart attack, but although the level of risk factors for heart disease was higher in this sample of cardiac patients than in the general population, only a quarter of patients initially attributed their symptoms to a heart attack rather than some other less serious cause such as indigestion. Clearly, there is a problem with patients recognising the symptoms of a heart attack. Horne et al (2000) addressed the problem of a mismatch in patients expectations of heart attack symptoms and those actually experienced. Patients who had typical symptoms had shorter delays than patients who had atypical symptoms. Similarly patients who are aware of a wider range of symptoms were less likely to delay in seeking help (Ruston et al, 1998). This may indicate a lack of knowledge among the general public about symptoms, other than chest pain, which often accompany heart attack. Intervention studies using public education tools highlighting symptoms of heart attack have been largely unsuccessful at reducing pre-hospital delay, so it may be useful to develop new ways of communicating a broad range of possible cardiac symptoms and the appropriate action to take in further research.

It is interesting that previous history of MI was a predictor of short total pre-hospital delay but it did not predict shorter decision time. This implies that patients were either unaware of their personal risk factor profile or underestimated their personal vulnerability to heart disease. Some studies which have investigated causal attributions

of heart disease in relation to patients own risk factor profile also support this (Astin & Jones, 2004; Martin et al, 2005). This has implications for health education and communication of the importance of risk factor management in patients at risk of heart disease and those who have already suffered an ACS, involving both health care professionals, such as GPs, and patients themselves. If patients have a different model of illness to those of health care professionals they may misunderstand the implications of risk factors, such as hypertension and diabetes, and underestimate their own personal responsibility for health maintenance, in recognising serious symptoms and making accurate attributions, and taking appropriate action such as calling an ambulance.

## 9.2: Aim 2 - To investigate the relationship between patients' health beliefs and their decision to seek help following the onset of symptoms of ACS.

Findings from this study showed that causal beliefs were significantly associated with pre-hospital delay. Patients' attribution of their heart problems to their mental state significantly predicted longer home to hospital delays. Although there was a strong association with pre-hospital delay, evidence from this study does not support the second hypothesis that longer patient decision delay would be associated with causal beliefs. Previous literature does not lead to specific predictions about the precise association between causal beliefs and delay, so the relationship between causal beliefs and home to hospital delay is a new finding. The reason for this association is not clear and requires further investigation.

Information collected about home to hospital phase was too limited to allow clear conclusions to be drawn, however, mode of help sought (calling an ambulance) and presence of a bystander predicted shorter home to hospital delays. A more detailed

breakdown of factors involved in home to hospital delay would be needed for a more thorough investigation, such as the nature of the relationship between the bystander and patient, specific coping strategies instigated by the patient or bystander, psychological factors and the time frame in which these things happened. Patients in this study were not confident of their own estimations of specific time periods such as waiting for help to arrive, assessment time, transport time, so these time periods were not analysed separately in this study, but in future research it may be possible to check ambulance attendance times, time of call to GP and time of attendance etc.

The way in which patients interpreted the question concerning causal attributions in this study was unclear, ie whether they understood it to be about the cause of their acute symptoms or about the causes of heart disease in general. There may have been some confusion here. Because the causal factors remained stable over 13 months it is likely that the question was interpreted as a question about the causes of heart disease in general. The question used to assess causal beliefs may therefore have been too ambiguous to elicit the information sought after, and did not clearly distinguish between the causes of patients own specific symptoms and the causes of heart disease in general.

The overall ranking of causes in this study is similar to other studies, with smoking and stress being the most frequently endorsed factors (De Valle & Norman, 1992; Gudmundsdottir et al, 2001; Murphy et al, 2005). Although the factor patients endorsed most strongly as the cause of the heart problem was personal behaviour, this was not significantly associated with pre-hospital delay. However, there is no reason why patients' causal attributions to smoking, being overweight, poor diet etc should affect patients' help seeking response to the onset of acute cardiac symptoms. This is also true of the attribution to heredity; patients' beliefs that their heart problem was caused by

heredity or genetic factors does not present any reason for an association with pre-hospital delay.

Patients who reported greater family stress, work stress or stress due to another illness were more likely than others to attribute the cause of their heart problem to their mental state. Patients who had a previous history of depression were also significantly more likely to believe that their mental state caused their heart problem. These factors have been identified as risk factors for heart disease in previous research (Kuper et al, 2005; Rosengren et al, 2004a). Attributions to stress may therefore be credible and reflect greater chronic exposure to various forms of life stress. Negative mood states, however, may also influence causal attributions. Patients who endorse stress and other negative emotional states as causes of their heart problem tend to have high levels of anxiety and depression scores (Day et al, 2005). In this study, patients who attributed their heart problems to their mental state had significantly higher levels of depression at baseline than those who attributed other causes. Findings presented in thesis showed, therefore, that both recent stress exposure and current mood were relevant to the belief that mental state was a cause of the heart problem (Table 5.13 and Table 7.21), and patients who attributed mental state as a cause of their heart problem had significantly longer home to hospital delays than patients who attributed some other cause.

## 9.3: Aim 3 - To investigate the relationship between patients' health beliefs and adherence to medical advice 3 months and 13 months after hospital discharge, and to identify factors which may predict non-adherence.

The findings from this study did not support the third hypothesis that adherence to medical advice (attendance at cardiac rehabilitation, medication compliance, and life

style change) would be predicted by beliefs about causes, independently of severity and clinical treatment, with the exception of smoking. Patients who were smokers at baseline and had attributed the cause of their heart problem to heredity were more likely to have quit smoking 13 months after their ACS. However, this was the only significant effect in numerous comparisons, so it is possible that it was a chance finding.

These results do not support findings from an earlier studies by Weinman et al (2000) which showed that patients' attributions to lifestyle causes predicted adherence to diet changes and strenuous exercise 6 months later, although once pre-MI behaviour was controlled for the association with causal attributions was no longer significant (French et al, 2005a). Results reported in this thesis do not show a significant association between attributions to personal behaviour or mental state and cardiac rehabilitation attendance, adherence to lifestyle changes or prescribed medication, as reported in previous studies (Weinman et al, 2000: De Valle & Norman, 1992). This may have been due to inadequacies in the telephone interview measure (as discussed in chapter 8).

No associations were found in this study between attendance at cardiac rehabilitation and causal beliefs. These data were based entirely on patients' self report as it was not possible to verify attendance with the individual cardiac rehabilitation centres since there were at least 5 separate centres plus other outlying hospitals. Another recent study also found no significant associations between a cardiac rehabilitation attendance and causal beliefs (French et al, 2005b). It has been suggested that attendance at cardiac rehabilitation may be predicted by beliefs about treatment rather than beliefs about cause (Cooper et al, 2005; French et al, 2005b) and this may be a useful approach to pursue in the future on order to carry this research forward.

## 9.4: Aim 4 - To investigate the relationship between patients' health beliefs and adjustment and quality of life 3 months and 13 months after hospital discharge

Just under a third of patients reported high levels of baseline anxiety and were more likely to have poorer mental health after 3 months, according to the single item measure of the SF-36. However, this association was no longer significant after 13 months. Although just over one third of patients were depressed at baseline, depression was not independently associated with any of the quality of life measures once analyses was adjusted for age, gender, GRACE risk score and mental state factor. Fewer depressed patients, however, participated in the follow up and this may have biased these results.

Patients with stronger beliefs that their mental state caused their heart problem had greater levels of anxiety in the short term (after 3 months) and in the longer term (after 13 months), and poorer quality of life. These findings support the fourth hypothesis that quality of life at 3 months and 13 months following hospital discharge would be predicted by causal beliefs, independently of treatment and clinical indices. This supports the theory proposed by Leventhal's Self Regulation Model since pre-existing cognitive representations about the causes of heart disease predicted later anxiety, independently of the event. Previous studies have shown that health beliefs are amenable to change (Petrie et al, 2002), it may therefore be possible to modify patients' inaccurate causal beliefs during their hospitalization in order to improve both psychological adjustment and quality of life following hospital discharge.

Stronger beliefs that mental state caused the heart problem were significantly associated with poorer quality of life in both the short term (3 months) and longterm (13 months). The impact was largely on the domain of mental health at 3 months, and on the domains

of limitations due to emotional problems and mental health (both as a single measure and as a summary measure) a 13 months. Causal attributions to stress responses such as worry, overwork, tiredness, and overexertion may interfere with emotional and social adjustment. As discussed earlier in chapter 8, patients' cognitive attribution of their heart problem to their mental state rather than other causes may lead patients to blame themselves for their heart problem and produce a negative outlook. This may then lead to problems with work or other daily activities as a result of emotional problems, psychological distress, and hence social and role disabilities due to emotional problems. This supports the idea of parallel processing proposed by the self regulation model as patients' cognitive beliefs about the cause of their heart problem predicted psychological distress, independently of the initial emotional impact of the event. It is possible that an intervention focussing on changing patients' cognitive representations concerning cause, for example emphasising personal behaviour, lifestyle and modifiable risk factors, and correcting maladaptive attributions to mental state may lead to later improvements in emotional adjustment and quality of life. Cognitive behaviour therapy techniques have been shown to be effective at changing beliefs and behaviour in a variety of conditions, and might be useful in future research into the beliefs of cardiac patients.

## 9.5: Strengths of this thesis:

The research presented in this thesis has demonstrated some interesting findings. By dividing the total pre-hospital delay time into two constituent phases, it was possible to identify different factors predicting short delays specific to each phase. I was also able to examine a wide range of different variables shown in previous literature to predict pre-hospital delay simultaneously. The sample was of a reasonable size to detect significant results and had a good ethnic mix. It was an observational prospective

cohort design with two follow up time points at 3 months and 13 months following hospital admission, which allowed the robustness of findings to be tested over a reasonable duration of time.

## 9.6: Limitations of this study:

Several limitations to the specific measures and procedures used in this study have already been discussed earlier in this chapter, and in the Discussion sections of chapter 3, 5, 7 and 8. Here, I outline some broader limitations of the work.

### 9.6.1: Representativeness of the sample population

The sample population recruited for this study was affected by selection bias in several ways. The study reported in this thesis was part of a larger study, the ACCENT study, and was consequently confined by the recruitment criteria necessary for the larger study (outlined in chapter 3). Patients with inflammatory conditions or other co-morbidities such as renal failure, cancer and any illnesses that may have affected mood were excluded from the study. Although the demographic characteristics of patients were similar to those of other studies investigating pre-hospital delay, there were fewer female participants than other cardiac studies in general, and the mean overall age was younger. Female participants are usually older when they present with symptoms of ACS, often present initially with anginal symptoms (Lerner & Kannel, 1986). It is therefore likely that female patients presented with more comorbid conditions due to their older age, and were less certain about the precise time of symptom onset, and these factors would have excluded them from this study.

One other probable result of the selection criteria was that a larger proportion of patients with STEMI were recruited compared with NSTEMI/UA than has been described in

recent surveys (Rosengren et al, 2004b) and this may have influenced the pattern of pre-hospital delay observed. Patients whose symptoms started gradually and who did not experience a clear or sudden onset time were not recruited into this study due to the need to identify a clear time of onset. It has been estimated that up to one third of patients may not experience an abrupt onset of symptoms or have difficulty identifying the time of onset. These patients may report a prodrome of symptoms that wax and wane over time, perhaps disappearing altogether (Dracup et al, 1995). Only patients who presented with chest pain were recruited for this study, thus patients who were diagnosed with ACS but did not suffer from chest pain were not included. Patients with serious psychiatric illness, on-going critical ischaemia, and other medical conditions which would compromise medium to long term outlook, and influenced mood and symptom presentation were also excluded. Again, this may have led to the exclusion of more women than men, and older rather than younger patients.

Only patients who were well enough to be interviewed and survived their acute cardiac symptoms were recruited. These findings may therefore reflect patients who experienced less serious forms of ACS (less severe atherosclerosis and/or less serious cardiac arrhythmias) than those who may not have survived or were too ill to participate. Patients who were unable to read or write were excluded, although it is unlikely that many patients fell into this category. A small number of patients were excluded because they were not fluent in English. The ethnic representation within this study was quite good, according to the census data for 2001 overall the London region had a non-white ethnic population of 28.8 % (Commission for Racial Equality, 2005) and in this study 18.6% of participants described themselves as Black or Asian.

Fewer physically active patients took part in the follow up at both 3 month and 13 months and this may have biased the findings concerning adherence to advice and physical activity, and in terms of quality of life. Patients who had higher levels of depression at baseline were also less likely to complete the follow up after 13 months and this may have reduced chances of observing associations between factors measured in hospital and later depression. Those who remained in the study and returned for follow up after 3 months and 13 months probably had better general health than those who dropped out. Patients who were more socio-economically deprived were also less likely to complete the 13 months follow up and this may therefore reduce the generalizability of the findings to less deprived population groups.

### 9.6.2: Timing

There may be some inaccuracy in the timing of the delay phases since much of this relied on patients' self report. Patients were recruited only if they could be reasonably confident that they knew what time their symptoms began. This was checked using estimation by the admitting doctor when possible. Admission to hospital times were recorded in medical notes or A&E records and are reliable. Patients were usually confident that they could accurately remember what time they made the decision to seek help, and this was discussed in some detail using descriptions of their daily routine, other events that had happened earlier the same day and other prompts to aid recall. Some patients were also able to confirm the timing of this with bystanders. In spite of these precautions, there may be some inaccuracies in this data.

Patients were rather less confident in timing of the smaller constituent phases of the home to hospital phase. They could not accurately estimate how long they waited for transport to hospital (ambulance or own transport arrangements) or the assessment

period (paramedics, GP or discussions with friends/relatives) with confidence so this was not analysed separately but included in the home to hospital phase.

Patients causal attributions were assessed within the first few days following admission and may thus be strongly influenced by information given to patients by nursing and medical staff. Patients are often given a lot of information whilst on the coronary care unit which includes information about the causes of ACS. Efforts were made to interview patients early in their treatment in order to elicit their own causal beliefs but it is possible that they had already been influenced by information given during the initial stages of their hospital admission.

Although it is possibly a chance finding, the change in smoking emerged after 13 months, so it is possible that changes in other behaviours may also emerge later. It may be too soon to assess behaviour change at 3 months in patients following ACS.

### 9.6.3: Measurement of causal beliefs

As discussed in chapter 5, the question patients were asked in order to assess their causal beliefs may have been somewhat ambiguous. Patients may have misinterpreted this question as a question about the causes of heart problems in general rather than the cause of their own recent experience of ACS. In future research, care should be taken to ensure this question is phrased more clearly. A questionnaire asking about the attribution of specific symptoms rather than causes may have been helpful in relation to investigating associations between beliefs about illness identity and cause and patient decision delay. The study by Horne et al (2000) went some way to doing this but did not analyse component phases of delay.

As discussed above, patients were given the baseline causal beliefs questionnaire within 5 days of their admission to hospital, it is therefore possible that their causal beliefs reflected information given to them by the medical staff rather than their own personal beliefs, although these beliefs remained stable over 13 months. Patients may also have felt more stressed, anxious or depressed than normal during this period. This may have produced a greater tendency to make causal attributions to stress (Day et al, 2005).

There may have been some limitations associated with the method of scoring used. A cued questionnaire was used which offered a list of possible causes. This may have produced a higher rate of responses than would have been the case if open ended questions had been used (Gudmundsdottir et al, 2001).

In scoring this questionnaire for this thesis, only positive endorsements of items on the causal beliefs questionnaire were used in the analysis. Some patients may have agreed with some of the causal attributions listed but did not have the confidence to register strong agreement. The analysis presented in this thesis may therefore underestimate the relationships between causal beliefs and personal risk profile.

### 9.6.4: Measurement of behaviours

There were a number of weaknesses in the telephone interview measure used to assess adherence, as discussed in chapter 8 (section 8.9.2). The measurement of cardiac rehabilitation attendance across a number of different hospitals via self report may also have reduced the accuracy of this information.

### 9.6.5: Biases in reporting

The use of self report is the most common method used in psychological research, but it is subject to self presentational and recall biases. It has been estimated that self reports may under estimate the true extent of non-adherence by approximately 20% (Haynes et al, 1980). Self report measures were used extensively in this study. In order to encourage patients to be as honest as possible without concern that their answers might influence their treatment, patients were assured that the information they gave would not be entered into their medical notes and that their doctor would not see their responses. Even so, this study was subject to the risk of interviewer bias whereby patients seek to offer answers they believe will please the interviewer, and recall bias involving over or under estimation of behaviours, such as medication adherence or regular exercise.

Data may also have been affected by recall bias. Data was collected retrospectively and patients were interviewed between 1 and 5 days after hospital admission. The self reports of pre-hospital experiences may have been affected by patients' efforts to understand their experience. Poor recall may also present a particular problem in this study since accurate measurement of the time intervals constituting total pre-hospital delay, decision delay and home to hospital delay depended on patients' recall of the time their symptoms started. However, patients were recruited only if they were able to recall events from symptom onset with reasonable confidence, and they were recruited early in the hospital stay so that their memory recent events would be fresh in their minds.

### 9.6.6: Statistical modelling

The primary method of data analysis used in this thesis was to carry out simple bivariate tests of association ($\chi^2$ tests or analysis of variance) followed by logistic or linear regression. The regression analysed the odds of an outcome (such as short pre-hospital delay or mental health on follow up) adjusted for age and gender. These analyses take account of basic factors that might confound the associations between predictors and outcome. What was not done was to carry out more elaborate statistical modelling in order to discover the relationship between different predictors. For example, pre-hospital delays were associated with symptoms patterns, social networks, the presence of a bystander, and contacting an ambulance. These factors are probably not independent of one another, but this was not formally tested. More complex statistical approaches such as path analysis and structural equation modelling would be desirable to identify the pathways involved more precisely.

## 9.7: Implications and directions for future research

This study has highlighted the importance of patients' beliefs that their mental state caused their heart problem, both in their help seeking behaviour and in their psychological adjustment and quality of life following hospital discharge. The association between patients' belief that their mental state caused the heart problem and longer home to hospital delay reported here is interesting and warrants further investigation. The retrospective nature of this research is problematic in terms of accurate measurement of pre-hospital phases, but further investigation may yield useful information concerning the role of denial at symptom onset, beliefs about symptoms and treatment among cardiac patients. Evidence presented concerning the associations between socioeconomic factors with longer home to hospital delays has important implications for equality of access to health care. It is important to investigate whether

more deprived patients face greater barriers in accessing emergency health care, and how this might be improved.

The impact of patients' causal attributions to their mental state on their psychological adjustment and quality of life is also an important area of future research. An assessment of patients' level of anxiety and depression could be done as part of their routine care in hospital using simple questionnaires such as HADS or the BDI. This might draw attention to patients in need of particular help and allow early treatment or counselling referral in order to avoid later problems with adjustment. An early intervention which might help patients to change causal attributions to their mental state and stress attributions might help to reduce anxiety and improve later quality of life. These assessments could be introduced as a routine part of ongoing cardiac secondary prevention by being repeated at the 3 month follow up with the cardiologist or at the rehabilitation programme, and at yearly follow up appointments with the cardiologist or GP. Further research investigating patients' beliefs might focus on developing an intervention to change patients' maladaptive beliefs about the cause of their illness. Cognitive behaviour therapy has proved useful in treating a variety of conditions, including anxiety and depression, and is gaining popularity with clinicians. This might provide a useful approach helping to change patients' maladaptive beliefs about their heart disease.

This study highlighted the problem of patients not recognising their chest pain as a symptom of heart attack, and misattributing them to another less serious cause, particularly indigestion. There is clearly a lack of understanding about the range of symptoms that may indicate heart attack, and also the importance of calling an ambulance as soon as possible. There is room for new and innovative strategies to for

public information campaigns to convey the dangers of inappropriate actions and delay, as they have in conveying the dangers of smoking.

The presence of a bystander present at the time of onset was shown to be an important predictor of short pre-hospital delay, and this highlights the importance of making information about appropriate actions to take in the event of someone experiencing a heart attack widely available to the general public, with a strong emphasis on calling the emergency services promptly. It might also be useful for medical staff and cardiac rehabilitation staff to allow patients relatives to participate to a greater degree in their treatment and rehabilitation. Providing clear information to patients' relatives about appropriate actions to take should they experience another onset of symptoms, or offering to teach resuscitation skills to relatives might reduce delay and improve survival rate in the event of a further cardiac event.

Evidence highlighting the discordance between patients own personal risk factor profile and their beliefs about the causes of their ACS is disturbing and emphasizes the need for clinicians to assess the illness beliefs and treatment beliefs of their patients. It should not be assumed that patients hold the same model of their illness as health care professionals, or that they have beneficial perceptions of available treatment and medication. Patients could be asked to complete a short questionnaire to ascertain their illness and treatment beliefs during their in hospital stay to give health care staff or cardiac rehabilitation staff the opportunity of providing individual, tailored advice that addresses their misconceptions regarding the causes of their heart disease.

Using the sample population recruited for this study, it will be possible to follow up participants to assess their adherence to medical advice and lifestyle changes, and

quality of life 3 years after their hospital admission for ACS. It will be possible to modify the telephone interview and postal questionnaires to include more specific measures which allow comparison between pre-ACS behaviour measured at baseline and behaviours such as smoking, physical exercise and alcohol intake, exposure to life stress and medication adherence. It will also be possible to investigate the stability of the causal beliefs over a longer time period. Psychological adjustment (anxiety and depression) may also be observed an extended time period. Data collection is currently underway.

## 9.8: Conclusion

This thesis has investigated the impact of patients' beliefs on two important clinical problems concerning the treatment of patients diagnosed with ACS; pre-hospital delay, and psychological adjustment and quality of life up to 13 months following hospital discharge. Short total pre-hospital delay in seeking help following the onset of acute cardiac symptoms was predicted by a range of different factors, including socioeconomic, social, clinical, contextual and psychological factors. Different factors predicted short delays in each of the two component time periods, decision delay and home to hospital delay. This has important implications for health policy and access to emergency health services, and public health education aimed at reducing pre-hospital delay and indicates that interventions may need to be targeted more carefully.

Patients' beliefs about the causes of heart disease made an important contribution. Patients who believed that their heart problem was caused by their mental state had significantly longer home to hospital delays. This highlights the role of patients' cognitive representations of their illness play in their help seeking behaviour. After discharge, emotional and psychological adjustment and quality of life was also

predicted by emotional and cognitive representations of heart disease. These findings have implications for understanding the contribution of psychological factors to the experience of acute heart disease, and point to methods of more effective patient care and management.

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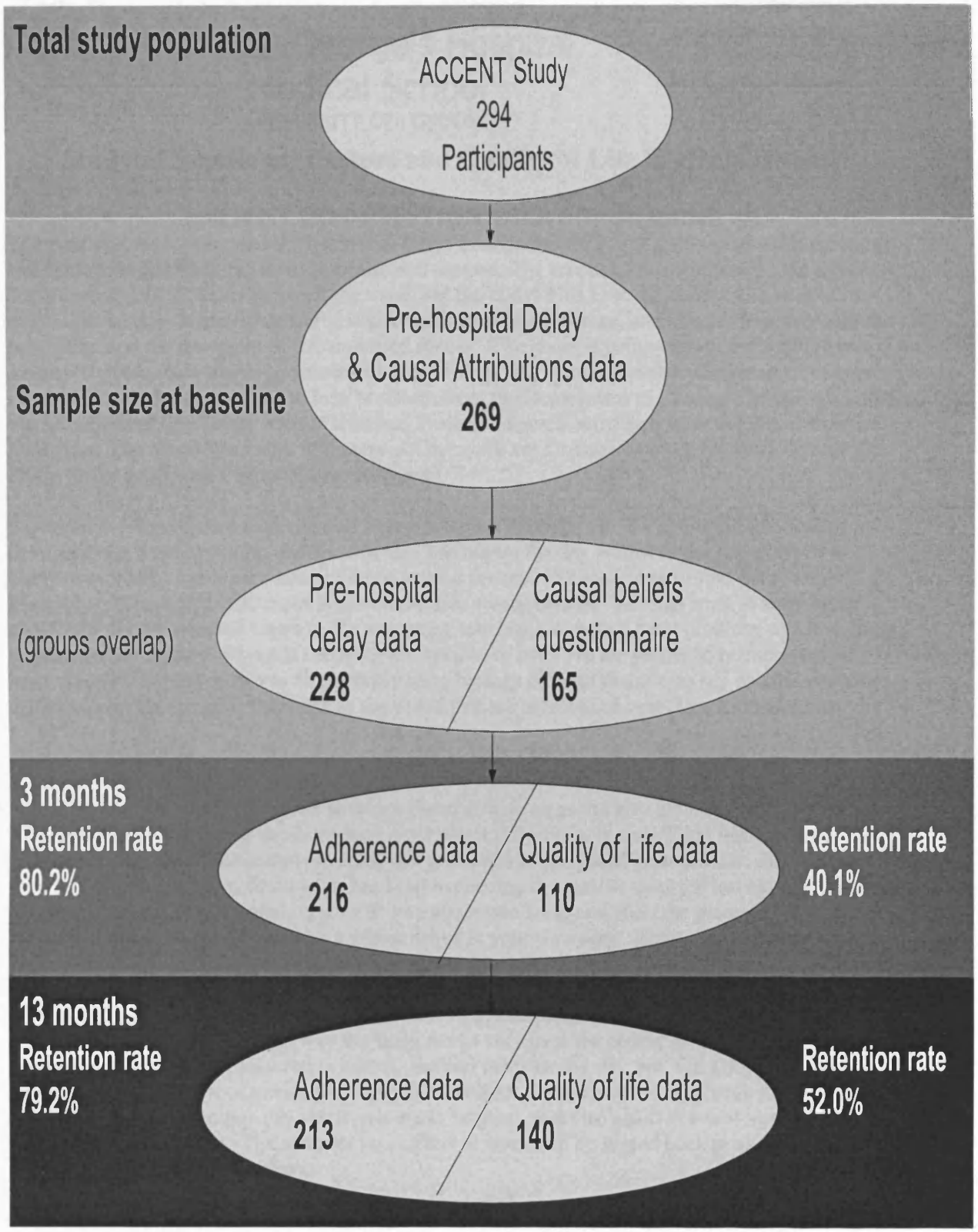
**Appendix 1: Sample size and retention rate at the different stages of study.**



**Appendix 2: Patient information sheet**

St George's Hospital Medical School
UNIVERSITY OF LONDON

**Department of Community Health Sciences**
St George's Hospital Medical School
Cranmer Terrace

# Study of Emotional Factors and Quality of Life in Heart Disease

## PATIENT INFORMATION SHEET (Confidential)

This research study is funded by the British Heart Foundation to try and explore how our emotions and behaviour influence the heart in health and disease. The results of this study will help advance our knowledge of the links between the mind and the body. This exciting and important area of medical science will contribute to the understanding of heart disease, and aims to improve both the prevention and the treatment of this common illness. The study is being carried out by Professor Andrew Steptoe from the Department of Epidemiology and Public Health at University College London, in collaboration with Dr Jean McEwan from the Department of Clinical Cardiology, and Ms Julia Sanders, Professor John Martin and Professor Steve Humphries from the Department of Medicine. The researchers who will carry out the work are Dr Lena Brydon, Dr Sue Edwards, Dr Philip Strike and Linda Porras (Research Nurse).

Exactly what triggers heart attacks and unstable angina is unknown. We still don't know why people have a heart attack on one specific day and not on the day before or the day after. It is likely to represent a complex interaction of several factors. We are trying to find out whether lifestyle and emotional state make a contribution in some patients. We also want to learn more about how people respond emotionally to coming into hospital with a heart problem, and how these responses may relate to physical recovery and quality of life. We are particularly interested in linking psychological factors with the underlying biology of heart disease, to see whether there are differences in the various chemicals in the blood that are involved in heart attacks and angina

### How You Can Help

The first thing we would like to do to take a blood sample so as to carry out biochemical analyses of substances that will help us understand more about the processes underlying heart disease. We also wish to use the blood sample to study the genes related to risk of heart disease. We would then like to interview you about what has been happening in your life over the last six months, right up until you came into hospital. This will take about one hour, and will take place on the Ward. We will also ask you to fill in some questionnaires in your own time. These concern how you are feeling about life, and how you cope with stress.

The second part of the study involves measurement of chemicals in your saliva. We know that several hormones that affect the way the body works vary over the course of the day, and fortunately these can be measured in saliva. Several times over a day, we will ask you to put a cotton dental swab in your mouth for a couple of minutes, then return it to a storage tube. We would like to do this on one day while you are in hospital, and then again in a few weeks time after you have returned home. The samples you collect at home can be posted back to us (we will provide the postage and packing).

We want to emphasise that all results obtained will be strictly confidential and will only be used for medical research purposes. You will be free to withdraw from the study at any time without giving a reason. Taking part or deciding not to take part will not affect your medical treatment in any way.Many thanks for reading this. We hope you feel able to take part in our study, which will help us understand more about the causes of heart disease and how to manage it better.

Any questions to Linda Porras (Research Nurse), Department of Epidemiology and Public Health, University College London,

**Appendix 3: Consent form**

St George's Hospital Medical School
UNIVERSITY OF LONDON

**Department of Community Health Sciences**
St George's Hospital Medical School
Cranmer Terrace
London SW17 0RE

Study Number:020151
Patient Identification Number for this trial:

## CONSENT FORM (Confidential)

Title of project: **A Study of the Emotional and Behavioural Factors in Acute Coronary Syndromes**

Name of Researcher: Professor Andrew Steptoe, Dr. Lena Brydon, Dr. Sue Edwards, Dr. Philip Strike
Any questions to Dr. Philip Strike, Department of Epidemiology and Public Health, University College London, . Telephone

**Please initial box**

1. I confirm that I have read and understood the information sheet for the above study and have had the opportunity to ask questions. ☐
2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. ☐
3. I understand that sections of any of my medical notes may be looked at by responsible individuals from (company name) or from regulatory authorities where it is relevant to my taking part in research. I give permission for these individuals to have access to my records. ☐
4. I agree to take part in the above study. ☐

| | | |
|---|---|---|
| Name of patient | Date | Signature |
| Name of Person taking consent (if different from researcher) | Date | Signature |
| Researcher | Date | Signature |

1 for Patient; 1 for Researcher; 1 to be kept with hospital notes

**Appendix 4: In-hospital semi-structured interview**

# Emotional triggers of ACS: Structured Interview

| | |
|---|---|
| Patient Study number: | Patient name: |
| Hospital no. | Date of Birth |
| Date of Admission: | Time of blood sample: |
| Date of Interview: | Interviewer: |
| Outside temperature on date of cardiac event (from Met. office): | |
| Patient's address and phone number: | |

## Clinical Details Of Acute Coronary Syndrome

| | |
|---|---|
| Admission BP | |
| Admission pulse rate | |
| ST elevation ? | |
| ST depression ? | |
| T wave inversion ? | |
| Bundle Branch Block ? | |
| Arrhythmia ? (AF/ VF/ VT) | |
| Territory (Inf/Ant/Post/Lat) | |
| Heart Failure ? | |
| Aspirin | |
| Heparin | |
| Thrombolysis | |
| Eptifibatide / IIb/IIIa | |
| Beta Blocker | |
| Nitrate | |
| Other | |
| Complications | |

**Initial Outcome**

| | |
|---|---|
| For Angiogram ? | |
| Angio result | |
| Treatment plan | |
| Revascularisation details? | |
| Final ECG | |

**Admission Blood Results**

| | |
|---|---|
| Haemoglobin | |
| Haematocrit | |
| White Cell Count | |
| Platelets | |
| Creatinine | |
| Serum cholesterol | |
| Triglycerides | |
| HDL | |
| LDL | |
| CRP (done in hospital) | |
| Troponin | |
| CK | |
| Blood glucose level | |

This interview will be divided up into several sections in which some of the questions might seem to be more relevant to your heart problem than others. Any information you provide us will be kept strictly confidential.

Are you ready to begin ?

**BACKGROUND INFORMATION**

To start with I'd like to gather some general background information about you.

1. How old are you? .....

2. Date of Birth ____ ____ ____

3. Gender: Male Female

4. Weight ..... Height ..... BMI .....

5. What is your marital status?

| | | | |
|---|---|---|---|
| Single | Married | Divorced | Widowed |
| Separated | Living as Married | Other | |

6. What category do you feel best describes your ethnic origin?

| | | |
|---|---|---|
| African | Asian | Middle Eastern |
| Oriental | White European | White non-European |
| Caribbean | Other | |

7. What educational qualifications do you have?

| | |
|---|---|
| None | |
| School Certificate | CSE's |
| GCSE's, O levels | A levels |
| Degree | Other |

8. How old were you when you left formal education?

9. With whom do you live (note how many people)?

| | | | | | |
|---|---|---|---|---|---|
| Parents | ..... | Spouse | ..... | Friends | ..... |
| Children | ..... | Other relatives | ..... | Rest/care home | ..... |

10. Can you count on anyone to give you emotional support (e.g. talking over problems to help you with a difficult decision)?

Yes No No need of help

(If Yes) How many people would give you this kind of support?

11. When you need some extra help, can you count on anyone to help with daily tasks like grocery shopping, house cleaning, cooking, telephoning, giving you a lift somewhere?

    Yes No No need of help

    (If Yes) How many people would give you this kind of support?

12. Do you rent or own your own home?

13. How many rooms are in your home (excluding bathroom and kitchen)?

14. Do you have use of a car/van? Yes / No

15. Were you employed at the time of your heart problem? If so, what was the nature of your employment?

Job title:

| | | | | | |
|---|---|---|---|---|---|
| Full time | .... | Part time | .... | Volunteer | .... |
| Disabled | .... | Unemployed | .... | Self employed | .... |

If retired, what was your last major occupation?

(If married female) What is/was your husband's occupation?

16. What is your current source of income?

17. What is your approximate personal yearly income, before tax is deducted? (If retired, any incoming money, as well as pension).

| | |
|---|---|
| Under £10,000 | .... |
| £10,000 - £20,000 | .... |
| £20,000 - £30,000 | .... |
| £30,000 - £40,000 | .... |
| Over £40,000 | .... |

18. What total income has your household received in the last 12 months? Please include your own income and that of others from any source, including wages, savings, investments, rent or property, and benefits.

| | |
|---|---|
| Under £10,000 | .... |
| £10,000 - £20,000 | .... |
| £20,000 - £30,000 | .... |
| £30,000 - £40,000 | .... |
| Over £40,000 | .... |

YOUR HEALTH

19. Do you have:

| | |
|---|---|
| Diabetes | Yes/No |
| (If Yes) Do you take insulin? | Yes/No |
| High blood pressure? | Yes/No |
| High cholesterol in your blood? | Yes/No |

20. Do you have any other health problems **at the moment** (relevant to heart problem and/or hormonal, immune, respiratory, eating disorders, etc) and medication?

21. Have you had any other health problems in the **past 5 years**?

22. When did you last have a cold or 'flu?

23. Were you taking any medicines or pills before you were admitted to hospital? Yes/No

If Yes, what type and for how long:

24. Has anyone in your family had heart disease? Yes/No

If Yes, what kind of heart disease

Did it cause the death of your relative(s)? Yes/No

If Yes, at what age did they die?

25. Do you currently have or have you had in the past any kind of mental health problem (e.g. depression, anxiety (panic attacks, severe phobia) or psychosis)? Yes/No

If Yes, what did the doctor call this?

If Yes, what, when and any medication?

26. Do you smoke cigarettes, cigars or pipes (specify)? Yes / No

If "Yes", please specify how many per day, and for how long you have smoked

If not a current smoker, did you smoke in the past? Yes / No

If "Yes", when did you quit smoking?

Are you currently taking nicotine replacement therapy? Yes / No

27. Do you drink alcohol? Yes / No

If Yes, how many units per week on average do you drink?

units per week

(1 Unit = ½ pint of beer, 1 glass of wine or 1 measure of spirit)

28. In the past 6 months have you taken any of the following drugs? If Yes, indicate average frequency.

Marijuana Yes/No
...../daily/weekly/monthly
Cocaine Yes/No
...../daily/weekly/monthly
Heroin Yes/No
...../daily/weekly/monthly
Amphetamine Yes/No
...../daily/weekly/monthly
Other Yes/No
...../daily/weekly/monthly
(details .....)

29. How many times per week do you do vigorous physical activity enough to make you out of breath?

None 1 2 3 4 5 6+

Please specify the activity
.....

30. Are you sexually active? Yes / No

If Yes, how often do you engage in sexual activity? ...../day, week, month

## EVENTS SURROUNDING YOUR HEART PROBLEM

31. What **time** of the day or night, and on what **date** did your heart problem occur?

.....
.....

(If not possible to establish time, exit/abbreviate interview here)

32. Tell me about any heart pain you experienced in the four days before you were admitted to hospital (type and duration)

.....

33. If it occurred at night were you asleep or just awakening?

.....

34. On the day your heart problem occurred, what time did you wake up?

.....

35. What time do you normally wake up? Time..... No habitual time?.....

36. Where were you when your heart problem occurred?

At home ..... Outside ..... Recreational activity .....

At work ..... In a car .....

Details ..(Was there anyone else present when symptoms began ? Who ?)

.....

37. What did you think was happening when your symptoms came on (ie did you think it was your heart or something else)?

.....

38. How long was it between the onset of your symptoms and deciding to seek medical help?

.....

What were your reasons for this delay in seeking help?

.....

39. How long did you have to wait between deciding to seek help and receiving medical attention?

.....

What were the reasons for this delay in receiving medical attention?

.....

40. Please describe what happened during the 24 hours before your heart problem

41: **During the previous 4 weeks:**

a. In the past 4 weeks has your relationship with your partner been stressful? Yes/No

(If Yes, mood rating) How stressful has it been? 1 2 3 4
*(mood ratings at back of questionnaire)*

b. In the past 4 weeks has your relationship with your family been stressful? Yes/No

(If Yes, mood rating) How stressful has it been? 1 2 3 4

c In the past 4 weeks has work been stressful? Yes/No

(If Yes, mood rating) How stressful has it been? 1 2 3 4

d Other than your heart problem, have you experienced any illnesses in the past 4
weeks that you have found stressful? Yes/No

(If Yes, mood rating) How stressful was that? 1 2 3 4

e. In the past 4 weeks have you felt more tired/fatigued than usual? Yes/No

42: . **During previous 6 months:**

a. In the past 6 months has your relationship with your partner been stressful? Yes/No

(If Yes, mood rating) How stressful has it been? 1 2 3 4

b. In the past 6 months has your relationship with your family been stressful? Yes/No

(If Yes, mood rating) How stressful has it been? 1 2 3 4

c. In the past 6 months has work been stressful? Yes/No

(If Yes, mood rating) How stressful has it been? 1 2 3 4

d. Other than your heart problem, have you experienced any illnesses in the past 6 months that you have found stressful? Yes/No

(If Yes, mood rating) How stressful was that? 1 2 3 4

e . In the past 6 months have you felt more tired/fatigued than usual?

Yes/No

**Table for mood rating**

| Level of stress | Description |
|---|---|
| 1- mild | Feeling rushed, too many problems, uneasy |
| 2 – moderately | Feeling preoccupied with problems, restless, unable to relax, short with other people |
| 3 – very | Overloaded with problems, very difficult to cope |
| 4 – extremely | Under overwhelming pressure, unable to cope, life out of control |

**Appendix 5: Social Network Questionnaire**

This section of the questionnaire is concerned with how many people you see or talk to **on a regular basis** including family, friends, workmates, neighbours, etc. Please circle your answer to each question.

1. What is your marital status at the moment?

| Single, or never married | Married, or living with your partner | Divorced, widowed or separated |
|---|---|---|

2. Do you have children?

| Yes | No |
|---|---|

If Yes, how often do you see or talk on the phone to your children?

| Never | Once a month | Once every two weeks | Once a week | Every day |
|---|---|---|---|---|

3. Are either of your parents living?

| Yes | No |
|---|---|

If your mother is living, how often do you see or talk on the phone to her?

| Never | Once a month | Once every two weeks | Once a week | Every day |
|---|---|---|---|---|

If your father is living, how often do you see or talk on the phone to him?

| Never | Once a month | Once every two weeks | Once a week | Every day |
|---|---|---|---|---|

4. If you are married or living with your partner, are either of your in-laws (spouse's parents) living?

| Yes | No |
|---|---|

If your mother-in-law is living, how often do you see or talk to her on the phone?

| Never | Once a month | Once every two weeks | Once a week | Every day |
|---|---|---|---|---|

If your father-in-law is living, how often do you see or talk on the phone to him?

| Never | Once a month | Once every two weeks | Once a week | Every day |
|---|---|---|---|---|

5. Are there other relatives who you feel close to?

| Yes | No |
|---|---|

If Yes, how often do you see or talk on the phone to these relatives?

| Never | Once a month | Once every two weeks | Once a week | Every day |
|---|---|---|---|---|

6. Do you have friends who you feel close to (i.e., people you feel at ease with, can talk to about private matters, and can call on for help)?

| Yes | No |
|---|---|

If Yes, how often do you see or talk on the phone to these friends?

| Never | Once a month | Once every two weeks | Once a week | Every day |
|---|---|---|---|---|

7. Do you belong to a church, temple, mosque or other religious group?

| Yes | No |
|---|---|

If Yes, how often do you talk to members of this religious group?

| Never | Once a month | Once every two weeks | Once a week | Every day |
|---|---|---|---|---|

8. Do you attend any classes (school, university, technical training, or adult education) on a regular basis?

| Yes | No |
|---|---|

If Yes, how often do you talk to fellow students or teachers?

| Never | Once a month | Once every two weeks | Once a week | Every day |
|---|---|---|---|---|

9. If you are currently working, how often do you talk to people (other than those you supervise) at work?

| Never | Once a month | Once every two weeks | Once a week | Every day |
|---|---|---|---|---|

10. How often do you visit or talk to your neighbours?

| Never | Once a month | Once every two weeks | Once a week | Every day |
|---|---|---|---|---|

11. Are you currently involved in any regular volunteer work?

| Yes | No |
|---|---|

If Yes, how often do you talk to people involved in this work?

| Never | Once a month | Once every two weeks | Once a week | Every day |
|---|---|---|---|---|

12. Do you belong to any non-religious groups? Examples include social clubs, recreational groups, trades unions, etc.

| Yes | No |
|---|---|

If Yes, how often do you talk to fellow group members?

| Never | Once a month | Once every two weeks | Once a week | Every day |
|---|---|---|---|---|

**Appendix 6: Cardiac denial of impact questionnaire**

These questions concern the way you feel about your heart problem. Please indicate the extent you agree with each of the following statements. Circle one answer for each statement. Please try to be as accurate and honest as you can and try not to let your answers to one question influence your answers to another question. There are no right or wrong answers.

1. I was not at all afraid when my symptoms first occurred.

| | | | |
|---|---|---|---|
| Strongly disagree | Disagree | Agree | Strongly agree |

2. I am a carefree, jovial person.

| | | | |
|---|---|---|---|
| Strongly disagree | Disagree | Agree | Strongly agree |

3. I was not at all afraid when I learned that I had had a heart problem.

| | | | |
|---|---|---|---|
| Strongly disagree | Disagree | Agree | Strongly agree |

4. I do not fear dying at all.

| | | | |
|---|---|---|---|
| Strongly disagree | Disagree | Agree | Strongly agree |

5. I very seldom take unnecessary risks.

| | | | |
|---|---|---|---|
| Strongly disagree | Disagree | Agree | Strongly agree |

6. My friends worry much more about my well-being than I do.

| | | | |
|---|---|---|---|
| Strongly disagree | Disagree | Agree | Strongly agree |

7. I seldom change the way I describe my heart problem to others, no matter who they are.

| | | | |
|---|---|---|---|
| Strongly disagree | Disagree | Agree | Strongly agree |

8 I am very calm even when faced with serious difficulties.

| | | | |
|---|---|---|---|
| Strongly disagree | Disagree | Agree | Strongly agree |

## Appendix 7: Causal beliefs questionnaire

### What do you think caused your heart problem?

Serious heart disease may be caused by many different factors. We would like to find out what factors you think were involved with your own illness. Listed below are a series of factors that patients in the past have thought helped to cause their heart disease symptoms. Please think about each item, then circle the answer that indicates how much you agree or disagree with each statement.

| **Factors that might have helped cause my illness:** | | | |
|---|---|---|---|
| My illness is hereditary – it runs in my family | **No** | **Maybe** | **Yes** |
| Smoking played a major role in causing my illness | **No** | **Maybe** | **Yes** |
| My illness was brought on by other medical problems | **No** | **Maybe** | **Yes** |
| Stress was a major factor in my illness | **No** | **Maybe** | **Yes** |
| Being overweight caused my illness | **No** | **Maybe** | **Yes** |
| High blood pressure was an important factor in my illness | **No** | **Maybe** | **Yes** |
| Diet played a major role in causing my illness | **No** | **Maybe** | **Yes** |
| I became ill because I over-exerted myself | **No** | **Maybe** | **Yes** |
| It was just by chance and bad luck that I became ill | **No** | **Maybe** | **Yes** |
| My illness was caused by poor medical care in the past | **No** | **Maybe** | **Yes** |
| Lack of exercise was a cause of my illness | **No** | **Maybe** | **Yes** |
| My illness was brought on by tiredness and exhaustion | **No** | **Maybe** | **Yes** |
| Genetic factors (genes) caused my illness | **No** | **Maybe** | **Yes** |
| My state of mind played a major part in causing my illness | **No** | **Maybe** | **Yes** |
| Working too hard caused my illness | **No** | **Maybe** | **Yes** |
| A germ or virus caused my illness | **No** | **Maybe** | **Yes** |

**Appendix 8: 3 months follow up telephone interview**

| | |
|---|---|
| Patient name | |
| EMOT no | |
| Interviewer | |
| Date of admission | |
| Date of telephone follow up | |

| | |
|---|---|
| Subsequent problems? | YES / NO - specify |
| Re – admission? | YES / NO |
| Revascularisation procedure? | YES / NO |
| Recurrence of symptoms? | YES / NO |

| | |
|---|---|
| Seen GP since discharge? | YES / NO |
| GP checked cholesterol? | YES / NO |
| GP checked BP? | YES / NO |
| GP checked blood sugar (if appropriate) | YES / NO |

| | |
|---|---|
| Attended rehab course? | YES / NO Where? |
| No. of sessions attended e.g 6/8 | |
| Found rehab course useful? | YES / NO |

Did you receive advice about the following either in hospital or subsequently on a cardiac rehab course?

| Subject | Advice given? | Advice implemented? | Comments? |
|---|---|---|---|
| Exercise | YES / NO | YES / NO / PARTIAL | |
| Weight | YES / NO | YES / NO / PARTIAL | |
| Stress | YES / NO | YES / NO / PARTIAL | |
| Alcohol | YES / NO | YES / NO / PARTIAL | |
| Diet | YES / NO | YES / NO / PARTIAL | |
| Were you a smoker before your heart problem? YES / NO | | | |
| Were you advised to stop YES / NO | | | |
| Advice implemented YES / NO / PARTIAL - specify | | | |
| Relapsed? - reason? | | | |
| How many a day do you smoke now? | | | |

What medication are you currently taking?

| | |
|---|---|
| Any problems with meds? | YES / NO - specify |
| Do you take all your tablets every day? | |
| How often do you miss a dose? | |

Thank you

**Appendix 9: 12 month telephone follow up**

| | |
|---|---|
| Patient name | |
| EMOT no | |
| Interviewer | |
| Date of admission | |
| Date of telephone follow up | |

| | |
|---|---|
| Subsequent heart problems? | YES / NO - specify |
| Severity (circle) | Mild Moderate Severe |
| Other major med probs? | |
| Re – admission? | YES / NO |
| Revascularisation procedure? | YES / NO |
| Recurrence of symptoms? | YES / NO |
| Limiting Angina? | YES / NO |

| | |
|---|---|
| Seen GP last 3 months? | YES / NO |
| GP checked cholesterol? | YES / NO |
| Cholesterol level | Mmol/l |
| GP checked BP? | YES / NO |
| GP checked blood sugar (if appropriate) | YES / NO |

| | |
|---|---|
| Attended rehab course? | YES / NO Where? |
| No. of sessions attended e.g 6/8 | |
| Found rehab course useful? | YES / NO |

Do you feel that your lifestyle has changed since your heart problem?

How?

Did you receive advice about the following either in hospital or subsequently on a cardiac rehab course?

| Subject | Advice given? | Advice implemented? | Comments? |
|---|---|---|---|
| Exercise | YES / NO | YES / NO / PARTIAL | |
| Weight | YES / NO | YES / NO / PARTIAL | |
| Stress | YES / NO | YES / NO / PARTIAL | |
| Alcohol | YES / NO | YES / NO / PARTIAL | |
| Diet | YES / NO | YES / NO / PARTIAL | |
| Were you a smoker before your heart problem? YES / NO | | | |
| Were you advised to stop YES / NO | | | |
| Advice implemented YES / NO / PARTIAL - specify | | | |
| Relapsed? - reason? | | | |
| How many a day do you smoke now? | | | |

What medication are you currently taking?

| | |
|---|---|
| Any problems with meds? | YES / NO - specify |
| Do you take all your tablets every day? | |
| How often do you miss a dose? | |

| | |
|---|---|
| Working pre heart problem? | YES / NO |
| Back to work? | YES / NO |
| When returned to work | |
| Full / part time / light duties? | |

Thank you

## Appendix 10: Medical Outcomes Survey Short Form 36 (SF36)

**The following questions are about your health and daily activities. Read each item and circle one answer for each question.**

1. In general would you say your health is:

| | | | | |
|---|---|---|---|---|
| Excellent | Very Good | Good | Fair | Poor |

2. Compared to one year ago, how would you rate your health in general now?

| | | | | |
|---|---|---|---|---|
| Much better now than one year ago | Somewhat better now than one year ago | About the same as one year ago | Somewhat worse now than one year ago | Much worse now than one year ago |

3. The following questions are about the activities you might do during a typical day.

Does **your health now limit you** in these activities? If so how much?

- Vigorous activities – such as running, lifting heavy objects, participating in a strenuous sport

| | | |
|---|---|---|
| Yes, limited a lot | Yes, limited a little | No, not limited at all |

- Moderate activities – such as moving a table, pushing a vacuum cleaner, bowling, or playing golf.

| | | |
|---|---|---|
| Yes, limited a lot | Yes, limited a little | No, not limited at all |

- Lifting or carrying groceries.

| | | |
|---|---|---|
| Yes, limited a lot | Yes, limited a little | No, not limited at all |

- Climbing **several** flights of stairs.

| | | |
|---|---|---|
| Yes, limited a lot | Yes, limited a little | No, not limited at all |

Climbing **one** flight of stairs.

| Yes, limited a lot | Yes, limited a little | No, not limited at all |
|---|---|---|

- Bending, kneeling, or stooping.

| Yes, limited a lot | Yes, limited a little | No, not limited at all |
|---|---|---|

- Walking **more than a mile**.

| Yes, limited a lot | Yes, limited a little | No, not limited at all |
|---|---|---|

- Walking **half a mile**.

| Yes, limited a lot | Yes, limited a little | No, not limited at all |
|---|---|---|

- Walking **one hundred yards**.

| Yes, limited a lot | Yes, limited a little | No, not limited at all |
|---|---|---|

- Bathing or dressing yourself.

| Yes, limited a lot | Yes, limited a little | No, not limited at all |
|---|---|---|

4. During the **past 4 weeks**, have you had any of the following problems with your
work or other regular daily activities **as a result of your physical health**?

- Cut down the **amount of time** you spent on work or other activities.

| Yes | No |
|---|---|

- **Accomplished less** than you would like.

| Yes | No |
|---|---|

- Were limited in the **kind** of work or other activities.

| Yes | No |
|---|---|

- Had **difficulty** performing the work or other activities (for example, it took extra effort).

| Yes | No |
|---|---|

5. During the **past 4 weeks**, have you had any of the following problems with your work or other regular daily activities **as a result of any emotional problems** (such as feeling depressed or anxious)?

- Cut down on the **amount of time** you spent on work or other activities:

| Yes | No |
|---|---|

- **Accomplished less** than you would like:

| Yes | No |
|---|---|

- Didn't do work or other activities as **carefully** as usual:

| Yes | No |
|---|---|

6. During the **past 4 weeks**, to what extent has your physical health or emotional problems interfered with your normal social activities with family, friends, neighbours, or groups?

| Not at all | Slightly | Moderately | Quite a bit | Extremely |
|---|---|---|---|---|

7. How much **bodily** pain have you had during the **past 4 weeks**?

| None | Very mild | Mild | Moderate | Severe | Very severe |
|---|---|---|---|---|---|

8. During the **past 4 weeks**, how much did **pain** interfere with your normal work (including both work outside the home and housework)?

| | | | | |
|---|---|---|---|---|
| Not at all | A little bit | Moderately | Quite a bit | Extremely |

9. These questions are about how you feel and how things have been with you **during the past 4 weeks**. For each question, please give the one answer that comes closest to the way you have been feeling. How much of the time during the **past 4 weeks**:

- Did you feel full of life?

| | | | | | |
|---|---|---|---|---|---|
| All of the time | Most of the time | A good bit of the time | Some of the time | A little bit of the time | None of the time |

- Have you been a very nervous person?

| | | | | | |
|---|---|---|---|---|---|
| All of the time | Most of the time | A good bit of the time | Some of the time | A little bit of the time | None of the time |

- Have you felt so down in the dumps that nothing could cheer you up?

| | | | | | |
|---|---|---|---|---|---|
| All of the time | Most of the time | A good bit of the time | Some of the time | A little bit of the time | None of the time |

- Have you felt calm and peaceful?

| | | | | | |
|---|---|---|---|---|---|
| All of the time | Most of the time | A good bit of the time | Some of the time | A little bit of the time | None of the time |

- Did you have a lot of energy?

| | | | | | |
|---|---|---|---|---|---|
| All of the time | Most of the time | A good bit of the time | Some of the time | A little bit of the time | None of the time |

- Have you felt downhearted and low?

| | | | | | |
|---|---|---|---|---|---|
| All of the time | Most of the time | A good bit of the time | Some of the time | A little bit of the time | None of the time |

- Did you feel worn out?

| All of the time | Most of the time | A good bit of the time | Some of the time | A little bit of the time | None of the time |
|---|---|---|---|---|---|

- Have you been a happy person?

| All of the time | Most of the time | A good bit of the time | Some of the time | A little bit of the time | None of the time |
|---|---|---|---|---|---|

- Did you feel tired?

| All of the time | Most of the time | A good bit of the time | Some of the time | A little bit of the time | None of the time |
|---|---|---|---|---|---|

10. During the **past 4 weeks**, how much of the time has your **physical health or emotional problems** interfered with your social activities (like visiting friends, relatives, etc.)?

| All of the time | Most of the time | A good bit of the time | Some of the time | A little bit of the time | None of the time |
|---|---|---|---|---|---|

11. How **TRUE** or **FALSE** is **each** of the following statements for you?

- I seem to get ill more easily than other people.

| Definitely True | Mostly True | Don't Know | Mostly False | Definitely False |
|---|---|---|---|---|

- I am as healthy as anybody I know.

| Definitely True | Mostly True | Don't Know | Mostly False | Definitely False |
|---|---|---|---|---|

- I expect my health to get worse.

| Definitely True | Mostly True | Don't Know | Mostly False | Definitely False |
|---|---|---|---|---|

- My health is excellent.

| Definitely True | Mostly True | Don't Know | Mostly False | Definitely False |
|---|---|---|---|---|

## Appendix 11: The Beck Depression Inventory

This part of the questionnaire consists of 21 groups of statements. After reading each group of statements carefully, circle the number (0, 1, 2, or 3) next to the one statement in each group which **best** describes the way you have been feeling **since you were admitted to hospital, including today**. If several statements within a group seem to apply equally well, circle each one. **Be sure to read all the statements in each group before making your choice.**

1. 0 I do not feel sad.
   1 I feel sad.
   2 I am sad all the time and I can't snap out of it.
   3 I am so sad or unhappy that I can't stand it.

2. 0 I am not particularly discouraged about the future.
   1 I feel discouraged about the future.
   2 I feel I have nothing to look forward to.
   3 I feel that the future is hopeless and that things cannot improve.

3. 0 I do not feel like a failure.
   1 I feel I have failed more than the average person.
   2 As I look back on my life, all I can see is a lot of failures.
   3 I feel I am a complete failure as a person.

4. 0 I get as much satisfaction out of things as I used to.
   1 I don't enjoy things the way I used to.
   2 I don't get real satisfaction out of anything anymore.
   3 I am dissatisfied or bored with everything.

5. 0 I don't feel particularly guilty.
   1 I feel guilty a good part of the time.
   2 I feel guilty most of the time.
   3 I feel guilty all of the time.

6. 0 I don't feel I am being punished.
   1 I feel I may be punished.
   2 I expect to be punished.
   3 I feel I am being punished.

7. 0 I don't feel disappointed in myself.
   1 I am disappointed in myself.
   2 I am disgusted with myself.
   3 I hate myself.

8. 0 I don't feel I am any worse than anybody else.
   1 I am critical of myself for my weaknesses or mistakes.
   2 I blame myself all the time for my faults.
   3 I blame myself for everything bad that happens.

9. 0 I don't have any thoughts of killing myself.
1 I have thoughts of killing myself, but I would not carry them out.
2 I would like to kill myself.
3 I would kill myself if I had the chance.

10. 0 I don't cry any more than usual.
1 I cry more now than I used to.
2 I cry all the time now.
3 I used to be able to cry, but now I can't cry even though I want to.

11. 0 I am no more irritated now than I ever am.
1 I get annoyed or irritated more easily than I used to.
2 I feel irritated all the time now.
3 I don't get irritated at all by the things that used to irritate me.

12. 0 I have not lost interest in other people.
1 I am less interested in other people than I used to be.
2 I have lost most of my interest in other people.
3 I have lost all of my interest in other people.

13. 0 I make decisions about as well as I ever could.
1 I put off making decisions more than I used to.
2 I have greater difficulty in making decisions than before.
3 I can't make decisions at all any more.

14. 0 I don't feel I look any worse than I used to.
1 I am worried that I am looking old or unattractive.
2 I feel that there are permanent changes in my appearance that make me look unattractive.
3 I believe that I look ugly.

15. 0 I can work about as well as before.
1 It takes an extra effort to get started at doing something.
2 I have to push myself very hard to do anything.
3 I can't do any work at all.

16. 0 I can sleep as well as usual.
1 I don't sleep as well as I used to.
2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.
3 I wake up several hours earlier than I used to and cannot get back to sleep.

17. 0 I don't get more tired than usual.
1 I get tired more easily than I used to.
2 I get tired from doing almost anything.
3 I am too tired to do anything.

18. 0 My appetite is no worse than usual.
1 My appetite is not as good as it used to be.
2 My appetite is much worse now.
3 I have no appetite at all anymore.

19. 0 I haven't lost much weight, if any, lately.
1 I have lost more than 5 pounds.
2 I have lost more than 10 pounds.
3 I have lost more than 15 pounds.
4

I am purposely trying to lose weight by eating less. Yes _____ No_____

20. 0 I am no more worried about my health than usual.
1 I am worried about physical problems such as aches and pains; or upset stomach; or constipation.
2 I am very worried about physical problems and it's hard to think of much else.
3 I am so worried about my physical problems that I cannot think about anything else.

21. 0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

## Appendix 12: Hospital Anxiety & Depression Questionnaire (Anxiety only)

This part of the questionnaire is about your emotions and how you are feeling. Read each item and circle the reply which comes closest to how you have been feeling **in the past week**.

1 I feel tense or 'wound up':

| | | | |
|---|---|---|---|
| Most of the time | A lot of the time | From time to time, occasionally | Not at all |

2 I get a sort of frightened feeling as if something awful is about to happen:

| | | | |
|---|---|---|---|
| Very definitely and quite badly | Yes, but not too badly | A little, but it doesn't worry me | Not at all |

3 Worrying thoughts go through my mind:

| | | | |
|---|---|---|---|
| A great deal of the time | A lot of the time | From time to time | Only occasionally but not too often |

4 I can sit at ease and feel relaxed:

| | | | |
|---|---|---|---|
| Definitely | Usually | Not often | Not at all |

5 I get a sort of frightened feeling like 'butterflies ' in the stomach:

| | | | |
|---|---|---|---|
| Not at all | Occasionally | Quite often | Very often |

6 I feel restless as if I have to be on the move:

| | | | |
|---|---|---|---|
| Very much indeed | Quite a lot | Not very much | Not at all |

7 I get sudden feelings of panic:

| | | | |
|---|---|---|---|
| Very often indeed | Quite often | Not very often | Not at all |